The Plain Truth About Tobacco

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Introduction: Interrupting the Mantras

The effects of habitual tobacco use over decades on general health, and with regard to specific illnesses, vary greatly between individuals, and related statistical findings from around the world vary greatly as well. The single clearest statistical link between smoking and disease is the link with lung cancer.

Cigarette smoking particularly, far more than cigar or pipe smoking, clearly influences the risk of lung cancer statistically. The particular influence of cigarettes stands to reason, since cigarettes are designed using mild tobaccos which produce a mild smoke that smokers typically find pleasant to inhale into the lungs, while directly or deliberately inhaling the smoke of strong nicotine-rich cigar and pipe tobaccos is quite aversive to most smokers.

The link between cigarette smoking and lung cancer is often cited as the strongest case in the vast research on factors influencing cancer risk amongst the population at large and this is a fair assessment. However, what has typically been made of that link by medical and public health practitioners and institutions, is absolutely and terribly flawed.

The purpose of this paper is to point out clearly, with reference to statements and research of the medical and public health communities, what sense can be made of the smoking and lung cancer link, in contrast to the plain nonsense that most often has been made of it.

A kind of groupthink exists amongst health professionals on the subject of tobacco, based on statistical over-interpretations amounting to perfect misinterpretations, and leading to a long chain of fallacious reasoning, producing ever more fallacious conclusions. This mindless groupthinking is positively rampant in the professions. Critics – there have always been critics within and without the professions – are long and well acquainted with this sorry state of affairs. It has been called, and is, truly scandalous.

However, most among the public at large probably take fallacious statements from generally obtuse health workers on the subject of tobacco at face value. As these commonly expressed but certainly false statements are analyzed here the average reader will likely be surprised and appalled at the patent illogic displayed by health professional groupthinkers. Though this paper will delve considerably into the medical/statistical literature, it takes a perspective of common sense (something rarely evident in the literature itself), and is written for a general readership. Any intelligent person can understand the basics that will be here discussed. The perplexing tragedy is that most health "experts" manage so awfully to *misunderstand* them.

Particular notice will be given in this essay to so-called environmental tobacco smoke (ETS) or passive smoking, but an understanding of common myths about active smoking is necessary in advance of ETS discussion, so we may begin with a typical message conveyed to the public about active smoking, and crucial analysis of that subject, before proceeding at length to the topic of ETS.

Messages given on the cable network CNN on 7 March 2006, following on the lung cancer death of never-smoker Dana Reeve, wife of actor Christopher Reeve, are typical. The hosts were correspondents Heidi Collins and John Roberts. The well-known CNN medical spokesman Doctor Sanjay Gupta was among the guests. Portions of transcript follow.

> ROBERTS: OK, tonight, Dr. Gupta is going to help us sort through fact and fiction when it comes to cancer.
>
> Even though lung cancer is the leading cancer killer, there are clearly huge gaps in our understanding of it. So, let's do a little true and false with Dr. Gupta.
>
> And let's start with this one: If you smoke now, you might as well continue, because you're destined to get lung cancer.
>
> Dr. Gupta, true or false?
>
> GUPTA: That is absolutely false, John, a really important point that it's always a good time to quit smoking.
>
> Let me give you a couple of quick facts. If you're – if you're 50 years old and you have been smoking your entire life, quitting today will cut your risk in half in a few years. If you're 30 years old, and you quit smoking today, you can reduce your risk back down to zero within a few years. So, it's always a good day to quit.
>
> ROBERTS: All right. Question number two: Smoking is by far the number-one cause of lung cancer, but radon gas is the leading cause among non-smokers; true or false?
>
> GUPTA: That is true. And this is actually surprising to a lot of people. Smoking is far and away the number-one cause. You know, eight – eight or

> nine times out of 10, it's going to be smoking. But radon, which is this naturally occurring uranium byproduct found in the soil, can actually infiltrate into your basement, and has been associated with lung cancer as well. So, it's actually the second most common cause of lung cancer.
>
> ROBERTS: And I think I know the answer to this question, number three: Asbestos causes lung cancer; true or false?
>
> GUPTA: That is true as well – a lot made of asbestos over the years. You won't find much asbestos anymore, because of all the regulations with regards to building, John. But asbestos specifically causes a type of cancer known as mesothelioma. And that is a type of lung cancer that is – is somewhat treatable, but can also be very deadly, if not caught early.
>
> ... ROBERTS: We're also answering your e-mails tonight.
>
> Allison from Missouri sent in an e-mail, and she asked, "Do lungs ever fully recover after quitting smoking?"
>
> And Sanjay Gupta, why don't you handle that one. Is it dependent on how long a person has smoked or is it not dependent?
>
> GUPTA: Yes, it is dependent, to some degree, on how long the person's been smoking. Let me say a couple of things. One is that it's always a good time to quit smoking. So regardless of whether your lungs can fully recover or not, it's always good to quit smoking.
>
> A couple of quick stats, though. If you're 50 years old and you quit smoking today, you can cut your risk in half. That's really important. If you're 30 years old and you quit smoking today, you can actually bring your risk back down to zero. And if you consider that your lungs are fully recovering, taking your cancer risk back down to zero, then it certainly does.

In these excerpts, Sanjay Gupta mouths more than one of the dogmatic nonsensicalities which we shall here call the "mantras" of the tobacco control movement. We will discuss several of these in this paper. One that may have stood out to you is Gupta's repeated citation of the "zero risk" of lung cancer which can be attained by those who quit smoking before middle age. Note particularly that the doctor chanted this belief on a program

prompted by the then-recent death of Dana Reeve, who *never* smoked. Note also, that despite the patent absurdity of anybody's (smoker, former smoker, or never smoker) having a "zero risk" of lung cancer, both of the program's hosts accepted the doctor's statement, over and again, without ever once blinking an eye.

This is very typical. The statements made by doctors and health officials about tobacco are very often dogmatic, plain foolish, and infinitely repeated, yet rarely questioned. The mantras get chanted with considerable uniformity by one doctor or official, or another, in the media, and in supposedly scholarly papers.

Now, of course, nobody can be said to have a *zero* risk of lung cancer. Anybody *could* get it. Review of research *does* suggest that a person who quits a typical cigarette habit before middle age (i.e. within about twenty years of establishing about a pack-a-day habit: since smoking inception is typically on either side of age twenty, this usually would equate to quitting in one's thirties or forties) will eliminate all *excess* risk of lung cancer, but that's an altogether different thing from establishing a zero risk, which never existed for anybody.

We here call the common tobacco control dogmas "mantras" because of the monotonous uniformity of their widely repeated chantings. A further similar example lies in a *PBS Newshour* program of 10 August 2005. In reaction to the then-recent lung cancer death of television news man Peter Jennings, host Jeffrey Brown invited Doctor Mark Clanton, Deputy Director of Cancer Care at the US National Cancer Institute, and Doctor Joan Schiller, a practicing oncologist from Wisconsin, to comment.

The obdurately obtuse interchange which followed requires a bit of introduction in order to be fully appreciated. In transcript excerpt which follows below, note the near-identity of Clanton's comments to Gupta's shown above, particularly regarding "zero risk".

In this case, Jeffrey Brown is astute enough (very rare in the media) to try to correct the doctor, by suggesting Doctor Clanton might really mean that quitting smoking would result in a smoker's reducing risk to the more moderate level a never-smoker enjoys (which is the truth of the matter), rather than establishing a "zero" risk.

Note the reaction: the doctor acknowledges the comment but returns *immediately* to the "zero" mantra, revealing his apparent belief that ex-smokers with less than twenty years' habitual smoking, and never-smokers, *cannot* get lung cancer, despite all perfectly contrary evidence, and certainly despite any pesky interruption from a logical news reporter.

Many readers may not understand Doctor Clanton's reference, within the PBS interview, to a "twenty pack year history". This is a technical phrasing used amongst biostatisticians which describes a patient's history of smoking a pack of cigarettes a day for twenty years. The twenty pack-year history is often cited as the latest point at which a smoker can achieve a "zero" (foolishly over-interpreted, of course) lung cancer risk by quitting.

The twenty pack-year point was also formerly an international standard for acceptability of donor lungs for transplantation; smokers with a higher pack-year history were considered unsuitable donors. The proscription against longer-term and heavier smokers as lung donors was dropped several years ago, as discussed in a 2003 publication by the American College of Chest Physicians, excerpt below.

> In a related study from the University of Texas Health Science Center, researchers evaluated the clinical outcomes of lung transplants in patients receiving either extended or standard donor lungs. Donors were considered "extended" if they met any of the following criteria: donor age of 55 or older, smoking history of more than 20 pack-years, having a history of pulmonary disease, chest radiographic changes, purulent sputum on bronchoscopy, or a decrease in oxygenation on 100 percent oxygen. Donors were matched with recipients, resulting in 20 patients receiving extended donor lungs and 11 patients receiving standard lungs. Recipients in both donor groups had similar outcomes in all posttransplant evaluation categories, including hospital and intensive care unit length of stay, length of intubation, readmission to the hospital, 6- and 12-month lung function tests, and 30-day mortality.
>
> "Most of the donors whose lungs we are now transplanting have met at least one of the criteria for extended donors, which would have made them an ineligible donor in the past. By using the physician-directed protocol, working closely with the Texas Organ Sharing Alliance, and extending the criteria for lung donation, we have been able to significantly decrease a recipient's waiting time on the transplant list, without compromising recipient outcome," said Dr. [Deborah] Levine.

Discussion throughout the PBS interview of never-smoker Dana Reeve, who had not yet died but was known to have lung cancer at the time, likewise did not deter Clanton, the National Cancer Institute representative, any more than it did Sanjay Gupta in his CNN appearance, from stubbornly reflexive dogmatism.

In reaction to this, Jeffrey Brown, having in preface stated his aim of clarifying what seemed confusing, and then faced with the extra-confusingly incantatory responses of Clanton, gives up on him. He turns instead to Doctor Schiller.

This finally elicits an admission of reality from her which, in the face of Brown's direct question, is practically unavoidable. Yet, given the ubiquity of mindless dogmatism in the health professions, one doubts that reality and logic can ever really take root in the impossibly sullied minds of most health "professionals".

The specific double-thinking point illustrated here, is this, and it does apply to most health "authorities" generally: they *know* that ex-smokers and never-smokers get lung cancer yet they do not *believe* it, and as we shall see, with wide application, *they base their analyses on their beliefs rather than on reality*. Let us now look at Doctor Clanton's confusion as he expresses it himself before analyzing further.

> JEFFREY BROWN: Dr. Clanton, I would like to clear up some things that are still confusing to people. For example, in the case of Peter Jennings, as for so many people, they smoked at one time, and then they quit.
>
> Now, when a person quits smoking, to what extent does his or her risk of developing lung cancer go down?
>
> DR. MARK CLANTON: It doesn't matter what stage you stop smoking. Your lung cancer or your risk of getting lung cancer does begin to go down. And the longer you spend in terms of time between the time you smoked and the time you stopped smoking, your risk continues to go down.
>
> The problem is in those people who have smoked a great deal – a 20-pack-year history, it's clear that the risk never returns to zero –
>
> JEFFREY BROWN: Never goes down to the case of someone who never smoked?
>
> DR. MARK CLANTON: That's exactly correct. So the more you smoked, the less likely it is it will go back to zero. The issue is your risk does go down and continues to go down for as long as you stop smoking.

> JEFFREY BROWN: Dr. Schiller, another thing that I think a lot of people wondered about this week was, in the case of Dana Reeve, you mentioned earlier people who develop lung cancer who never smoked. Now, how unusual is that?
>
> DR. JOAN SCHILLER: Well, actually, about 10 to 15 percent of all lung cancers occur in people who have never smoked.

Poor Jeffrey Brown. He tried to clear confusion. He tried at least better than the CNN crew did, or most news men ever do. He made but little headway. We shall look further and try here to do better still.

1. Population Statistics

There is presently an anti-tobacco fanaticism abroad across much of the world. If you smoke your abolitionist doctor may blame nearly any medical condition you suffer on the smoking, whether there is any basis for this, or not. Smoking can be more or less plausibly linked with a number of afflictions, but as we have said, the single clearest statistical link is with lung cancer. It is therefore of lung cancer, a large enough topic in itself, that we shall treat in this paper.

We will look at the realities of the smoking / lung cancer link and contrast them with the fallacies that have taken root in the minds of far too many in the medical establishment. Similar fallacies exist regarding other supposed links between smoking and health problems. The situation with lung cancer provides the best example for illustrating widespread misunderstanding of smoking relative to health generally. Proper understanding of the topic requires proper grounding in the essential elements surrounding smoking in relation to lung cancer. So let us look at these, individually, and then collectively.

This section will present a comprehensive and easily comprehensible overview of lung cancer prevalence and provenance, in the West as specifically illustrated via United States national statistics, as well as discussion of lung cancer in the East, particularly regarding the case of Asian women, with national statistics from Taiwan employed as illustration. In order to present this comprehensive view we shall, before presenting the US population overview via clear tables, first review at some length each crucial part of the lung cancer puzzle, and then combine those pieces, so as to complete the big picture.

Let's begin with the plain question: what, if you never smoked, is your risk of lung cancer? What, in other words, is the "base risk" of lung cancer, amongst the population at large, independent of any excess risk imposed by a smoking habit? Very little specific research on this question has been done but the figure can be ascertained for regional populations.

Regarding region, it's necessary to note that statistical research on lung cancer varies considerably between the West and the East. Studies in the West (mostly done in North America and in Western Europe), while suggesting some inter-regional or national distinctions, have a general conformity, while Eastern studies (mostly from the Orient), though they likewise can be said to have a general conformity with each other, present a picture different in some crucial respects to that suggested by the Western studies.

Both Eastern and Western studies suggest a statistical link between smoking and lung cancer but they tend to show a different configuration between "base risk" and "relative risk" (terms to be discussed further along in this section).

This East/West divide is a result of an apparently greater degree of diagnostic bias amongst Western practitioners than amongst those in the East, as will be explored and explained in due course, but let us begin with the West, which shall be the primary focus of this essay overall.

In necessary preface to the question of a never-smoker's risk, let us ask: who is a smoker, and who is not? Lung cancer statistical research has burgeoned since the nineteen fifties. Biostatisticians in the West early learned that from amongst typical Western populations, from that period and even today, it was virtually impossible to collect substantial study groups of mature persons (lung cancer is a disease of old age as we shall illustrate) who had truly *never* sampled tobacco.

Mere sampling or very small experience of tobacco had to be eliminated from any definition of a "smoker" as a purely practical matter. Thus, although individual studies vary enormously in their methods, including how populations are categorized, a fairly consistent definition of "never smokers" has emerged.

A "never smoker" is a person who has never smoked more than 100 (or, in some studies, up to several hundred) cigarettes in a lifetime.

An "ever smoker" is a person who has smoked more than 100 (or a few hundred) cigarettes in a lifetime.

The most typical sub-categories of smokers are "former smokers / ex-smokers" (smokers who quit – usually at least a year or a few years before lung cancer diagnosis – this varies from study to study) and "current smokers" (who smoked until, or nearly until, the point of lung cancer diagnosis).

Various other subdivisions, based on length or intensity of smoking habit, or other factors, appear variously from one study to another, but "never", "former" and "current" are the fairly consistent "usuals", and the "never" category specifically is fairly consistently used in terms of its definition.

In our population computations here we shall use lung cancer death statistics. These are

similar to lung cancer incidence statistics (about 85% of persons diagnosed with primary lung cancer die of it within five years) and are far more reliable, being based on actual counts of death certificates, than are incidence statistics, which are purely estimates based on incomplete reports from various regional centers. These incidence estimates can be vastly unreliable. In the US, for instance, the estimation formula used by the American Cancer Society and the National Cancer Institute was recently altered, producing tens of thousands of additional estimated lung cancer cases per year by the new computation as would be produced by the former formula, which had been in use for many years.

For the West the base risk of lung cancer, the risk one may be said to face if one never smoked, is approximately two per cent. As we have seen in the Gupta and Clanton interviews, which typify the distorted view often expressed by health professionals, there is a strange (frankly eerie) reluctance to admit that persons who never smoked could have *any* risk of lung cancer. At times the "groupthinkers" force themselves to admit that such a "base risk" does exist. They may point to the study known as the "British Doctors Study", by Richard Doll and Austin Bradford Hill. This estimated an approximate one per cent risk amongst never-smoking doctors.

The best source, however, is the wider population study produced in 1998 by Eva Prescott et alia of The Copenhagen Center for Prospective Population Studies. Data shown on table below (following page) is excerpted from original publication.

Table 3 Age-adjusted mortality rates and relative risks (RR) by smoking status.[a] Mortality rates were indirectly standardized in 5-year age groups to the total study population

	Annual mortality per 100 000									
	Women					Men				
Cause of death (no. women/men)	Never smokers	Ex-smokers	<15/day	≥15/day	Test for trend[a]	Never smokers	Ex-smokers	<15/day	≥15/day	Test for trend[a]
Respiratory disease (168/308)	34	76	107	179		51	113	157	168	
RR[b]	1	2 1	2 9	4.7	<0.001	1	2.2	3.0	3.6	<0.001
Vascular disease (1023/2214)	400	459	631	664		662	821	1099	1130	
RR[b]	1	1 2	1.8	2.0	<0 001	1	1.2	1.6	1.7	<0.001
ischaemic heart (524/1419)	209	259	323	324		387	528	718	694	
RR[b]	1	1.3	1.8	2.0	<0.001	1	1.4	1.8	1.8	<0.001
cerebrovascular (216/332)	76	87	127	169		140	122	162	173	
RR[b]	1	1.2	1.8	2.5[c]	<0.001	1	0.9	1.1	1 2[c]	0.43
Cancers (937/1771)	374	388	592	734		396	540	706	1.010	
RR[b]	1	1.0	1.6	1.9[c]	<0.001	1	1.3	1.7	2.6[c]	<0.001
lung cancer (188/619)	23	65	140	225		25	145	215	424	
RR[b]	1	2.7	6.2	9.6	<0.001	1	5 5	8.1	17.3	<0.001
other tobacco related cancers (118/346)	42	21	75	130		72	85	130	212	
RR[b]	1	0.5	1.8	3.2	<0.001	1	1 2	1.7	3.0	<0.001
Other causes (493/994)	192	241	309	337		248	339	433	528	
RR[b]	1	1.3	1 6	1.8	<0.001	1	1.4	1.7	2.2	<0.001
All causes (2621/5287)	995	1162	1638	1915		1351	1813	2394	2840	
RR[b]	1	1.2	1.7	2.1	<0.001	1	1.3	1.7	2.1	<0.001

[a] Test for linear trend between never smokers, light and heavy smokers.

[b] The RR were adjusted for age, study cohort, and calendar period.

[c] Wald test for the null hypothesis of no difference between male and female RR. $P < 0.05$.

Based on long-term study of 30,809 subjects followed between the 'seventies to the 'nineties, the table shows computations of expected annual mortality per 100,000 of the population at large, rounded to whole numbers of expected deaths in each of four major cause of death classifications: respiratory disease, vascular disease, cancers, and other causes, with subset figures for certain specific conditions, including lung cancer. Numbers are categorized specifically by smoking status.

Of 1,351 annual deaths per 100,000 of population from all causes, amongst male never-smokers, lung cancer is shown to take 25. Of 995 annual deaths per 100,000 of population from all causes, amongst female never-smokers, lung cancer is shown to take 23.

Thus lung cancer is shown to account for two per cent of all deaths amongst never-smokers, equally for men, and for women (men, 25/1,351, women 23/995, all 48/2,246). This figure of two per cent has the advantage not only of being the best ever researched, but also has the virtue of meeting the test of concordance with overall analysis of US population statistics, to be displayed later in this section.

A base risk of 2% is a "piece of the puzzle" with which everything else about lung cancer fits, makes sense, and adds up, and without which, nothing does. If the base risk of lung cancer was taken to be as little as one per cent or as great as three per cent no sense whatsoever could be made of US population statistics in relation to what is known overall about lung cancer and about the smoking population. With knowledge of the two per cent base risk every factor coincides to produce a clear and comprehensive perspective view of lung cancer amongst the population at large, as we shall show.

Rather than confusion and misconstruction, we have here what Jeffrey Brown of PBS, and many others everywhere, have been seeking: a plain answer to a plain question. Statistically, what risk of lung cancer does one have, if one never smoked? If you live in the West the answer is two per cent, taking Western figures at face value; we shall deal separately with the question of diagnostic bias, juxtaposing the West with the East, after analyzing Western statistics *as they exist*.

That Western statistics suggest a base risk of 2% is a plain fact you can use. It is not based on the Prescott study alone: we will here, as mentioned, put the two per cent figure to the test in relation to what else is known about lung cancer and population statistics: it does add up and it does makes sense. If the Prescott study did not exist the 2% figure would be naturally obvious from all else we will consider in producing an overall population perspective on lung cancer.

We have said that most health professionals give this "base risk" for lung cancer little notice. Few of them are probably aware of it. They are always more interested in "relative risk". "Relative risk" excites them because it gets headlines.

Relative risk is what is being reported in typical headlines we see along the lines of "Meat Eaters Face Double the Risk of Disease X". In such a case, some study, usually poorly performed, found that some category of meat eaters, this category likely being poorly conceived, were taken to be twice as likely to develop Disease X than were some other category of persons who probably simply ate somewhat less meat.

It means these researchers came up with a "relative risk" of 2. This was likely expressed as 2.00, or perhaps more likely as something like 2.1 or 2.12, in the study itself, as it should not be in any such study of a common lifestyle factor: the result for what is often a very small study group, frequently just dozens or even fewer actual disease patients, might at best have rough applicability to the public at large, but never decimal precision, indeed not even with comparatively large studies except by luck.

The use of decimals in such individual studies, for other than computational purposes, is perfectly haughty and pretentious. Decimals should not be used in presenting individual studies' final relative risk results above 1.00. That is quite as pretentious as claiming stop-watch accuracy for a sundial.

Decimals should only be displayed for results at or below relative risk 0.5: i.e half, or less, equating to results of 2, double, or more. Simply because small decimal differences *can* be computed does not mean that they *should* be. They can *only* be misleading.

As we shall describe, biostatistical studies of general lifestyle factors, even when well-performed, are a very rough tool of intellectual inquiry: general indicators at best. Even on the basis of whole numbers – half or less, twice or more – one must review numerous studies on a given factor to gain a general indication of which whole number risk might have approximate applicability to the population at large. These studies may, in a clear case, be fairly consistent in suggesting a whole number risk. They will *never* be consistent as to which whole number that is.

In nearly all cases such as our Disease X example, the study, and the newspaper report, will ballyhoo relative risk but make no mention of the base risk of Disease X. Let's say, out of the wind, that the base lifetime risk amongst the overall population for Disease X is one in a thousand. If the study's result was applicable not just for the dozens studied (probably quite doubtful) then heavier "meat eaters" (whoever they are or aren't) would face a risk of two in a thousand.

Everybody dies of something, sometime, so an extra one in a thousand risk of a particular obscure disease, might not seem like much to the average person. This is another reason health workers tend to ignore base risks.

"Twice the risk" may alarm a patient and get him to obey lifestyle prescriptions and proscriptions demanded by health workers. A low absolute risk, such as a total two in a thousand chance, may induce the patient to wonder what the health worker is all uptight about. The patient might ignore the health worker and even wonder gravely at his or her capacity for sound judgement. Health workers don't want that. They want to command and be obeyed.

Relative risk really means nothing without knowing base risk. Multiplied together, base risk and relative risk show absolute risk for a given population. Absolute risk is the risk a given population segment of a given description faces in the all in all. With their sole

fixation on relative risk, health workers typically ignore the simple formula for absolute risk, which of course is this:

Base risk x Relative risk = Absolute risk

For never smokers as a group, relative to ever smokers as a group, the absolute risk is the same as the base risk.

Base risk (2%) x Relative risk (1) = Absolute risk (2%)

Relative risk, for an unaffected population, is stated as 1 (or 1.00 as many studies will show it): it simply does not change base risk. Of course it is not "zero", as fools might tell you, and do.

Amongst ever-smokers, some will have quit before "20 pack-years", others who might never quit altogether will never have smoked to a "20 pack-year" level (for example: a fellow who smokes a pack a week – and this is not uncommon – would take 140 years to smoke – in small doses – the number of cigarettes that a pack a day man does in 20 years, and objective review of research does suggest what common sense would also allow: that, just as a true cigarette habit cut short will not affect lifetime lung cancer risk, minimal smoking, even over several decades, will not affect lifetime lung cancer risk.) An overall relative risk estimate for ever-smokers must take all of this into account.

Nevertheless, it is clear that ever-smokers, as a whole group, would reasonably be expected to have the same base risk, but a higher relative risk, and therefore a higher absolute risk for lung cancer, than do never-smokers as a group. What are the relative risk and absolute risk for ever-smokers?

To begin, on this question, let us ask: what really is "relative risk" and how is it derived?

The term "relative risk" is used in two senses: a common usage, and a technical usage. Technically, "relative risk" is an exact synonym for a specific computation otherwise known as "risk ratio". Risk ratios are often, though not always, computed for what are called "cohort" or "prospective" studies of disease patients. Such prospective studies comprise a minority of studies in the literature. The specific "risk ratio" computation is appropriate *only* to cohort studies.

The great majority of studies are of the so-called "case-control" or "retrospective" type and

the great majority of these employ an "odds ratio" computation, very similar to "risk ratio", but which has wider applicability: odds ratios are appropriate for both case-control and cohort studies.

There is another similar computation known as "hazard ratio". In the sorts of studies we will most be looking at the hazard ratio computation is not commonly used. As a general statement, in like situations and if used as appropriate to study type, whether risk ratio, odds ratio, or hazard ratio computations are used, the intent of conveying risk relationship is the same and the result most often comes out similarly.

One will also come across the "rate ratio". This has a similar purpose to the others discussed but is only appropriately applied for analyses of disease incidence or death *rates* across a designated population size over a designated time period.

These are technical points, of no importance to the general reader, except that he will benefit to know of the distinctions: this avoids confusion when one is presented with similarly-based study results which may be described by one or another of the distinct but similar terms: risk, odds, or hazard ratio; rate ratio is likewise a similar method of conveying risk relationship but is singular in its application only to analyses of rates across populations.

In *common* usage, both within and without the health professions, "relative risk" is used as a catch-all term, referring to results derived by risk, odds, hazard, or rate ratio computation. It is in this common sense that we have and will use the "relative risk" term in this essay. Where relative risks are herein computed we shall use the most common and versatile calculation, which is, specifically, the odds ratio.

Rather than simply describe how relative risk is computed we shall put this in context by describing how a small case/control study might be done on a "disease" we'll make up for the occasion; this will give the reader a clearer idea of "relative risk" within context, and forms a useful digression as well, for understanding of actual study reports to be discussed further on in this essay. Caveat: lots of these sorts of studies deserve (really demand) derision so we'll let that show when necessary in illustrating with this make-believe example.

Meet Doctor Who, a medical doctor. He has recently completed his residency at Suburban Hospital which lies in a middle-class town ten miles outside of a major US city. He remains a visiting physician at Suburban Hospital and also recently received certification as a

specialist in nephrology (kidney disease). Supplementarily to his medical training, Doctor Who took additional courses in statistics, with the intention of publishing studies in the realm of what is called "lifestyle epidemiology" (which attempts to relate lifestyle factors to disease incidence) from time to time, as an adjunct to his general clinical practice.

During his residency at Suburban Hospital, Doctor Who occasionally treated patients with mild kidney inflammation, of no clear derivation, along with gastric distress. This combination of conditions, he found, was reported from time to time across the country with great consistency in terms of symptoms, and severity, which ranged from quite mild to briefly though remediably acute in terms of gastric distress.

Who discovered that the malady he thought he saw had a name, Funk's Syndrome, after the Doctor Funk who first described it in a fairly obscure medical article of decades ago. Funk described his syndrome in a spirit of great discovery, specifying the symptoms, reporting successful simple treatments, noting that the condition seemed probably more common in males than in females, that patients were typically generally healthy, and in the age range of 40-70, but offering little else.

Funk's Syndrome seemed to be a fairly common complaint, in Who's experience, and possibly across the country as well from what he could tell, though specific incidence figures were lacking. Its aetiology (origin) was mysterious. It was thought almost certainly not pathogenic (caused by a virus or "bug"), but apart from that, nothing was really clear as to what brought on what Who saw as the particular, consistent, and definite set of symptoms Funk described.

No formal lifestyle epidemiology studies had been done on the subject, in part because, while Funk's Syndrome was temporarily very unpleasant for about ten per cent of patients, who had to be hospitalized through several days of considerable abdominal pain, it was easily remediable in all reported cases, responding well and promptly to a simple treatment plan of pain control as needed, mild anti-nausea medication, and, especially, strict bed rest.

There was a further reason for the paucity of research on Funk's Syndrome. As a matter of fact, some critics had noted that Funk's Syndrome was really unworthy of specific classification, that it's simply common bum digestion dressed up with a pretentious name, its accompanying mild kidney inflammation being something likewise fairly common in itself, and trifling, whether or however it might be related to the digestive upset, or whether it might be purely and simply coincidental with digestive trouble in some cases.

The critics noted that people come down with digestive upset, or mild kidney irritation, through multifarious mechanisms, and that there would be no virtue in researching Funk's Syndrome as a distinct disorder. There might be real value in trashing it as a diagnosis altogether, the critics suggested.

Doctor Who, however, is unaware of these criticisms, and as an unimaginative technically-minded man, he would never think of them on his own. He is proud of himself for having researched Funk's Syndrome from the literature, and for recognizing it in some patients, as many clinicians might not. He has found it mentioned in a few medical journals published over the years, so believes in Funk's as a distinct diagnosis. He is surprised to find very little formal research on it in the medical literature, and thinks this gap in research should be filled.

Doctor Who notes what he can glean from the sparse literature, and follows his patients closely, with an eye to increasing knowledge amongst the medical community, and also to boosting his reputation and professional prospects, by publishing on the subject.

The prognosis with Funk's is consistently good. Who notes from his research that patients in the greatest distress typically could be returned to full health with a few days in a hospital, kept constantly in bed, followed by another week or two remaining mostly in bed at home, and with medication as needed. Milder cases received similar but less intensive treatment with no need of a hospital stay, and if patients followed advice to remain in bed as much as possible, they generally recovered within a week.

The benefit of lying still for days or weeks particularly struck Doctor Who, and he had noticed, anecdotally, during his residency, that several of his Funk's patients worked in professions requiring a lot of driving: a couple of freight truck drivers, a travelling salesman, a regional real estate appraiser, and a long-distance medical courier came to Who's mind. The doctor came up with a theory that the repeated jostling of long-distance driving might be a common factor that brought on the organic disturbance of Funk's.

Doctor Who got in touch with Doctor Watt, a fellow practicing nephrologist in his state whom he had met, and the editor of a small professional journal dedicated to nephrology. Who told Watt of his research, of his thinking, and of his plan: he had decided to do a simple case/control study of all Funk's patients at Suburban over the next couple of years specifically regarding driving.

Watt, also rigidly technically-minded, and as lacking in intellect as is Who, told Who his

thinking was brilliant. Watt would look forward eagerly to the results from Who's study and would certainly publish the study if it suggested, as Watt put it, "meaningful results."

Who's method would be to assess Funk's patients, via questionnaire, by two parameters: employment (which he would stratify by high or low driving intensity) and by a more comprehensive question as to whether, over the previous twenty years, the patients had driven more than or less than 10,000 miles per year, on average, overall, both in personal and professional driving.

Otherwise the questionnaire would collect only some very basic information: sex, race, age, town of residence, annual household income, and any serious underlying conditions. He hoped he could match his "controls" (his comparison subjects) closely with his studied Funk's patients. Since no previous epidemiological studies had been done on Funk's patients he would have little specific basis for adjusting his relative risk results regarding such background factors. That could be a nuisance, but with closely matched cases and controls, such adjustment should not be needed.

Within twenty-four months Who had collected information on twenty hospitalized Funk's patients. These comprised, as it turned out, ten males and ten females. The age range was between 45 and 66. Ten came from Wallyton, location of the hospital, and there were five from each of two surrounding towns: Beaverton, and Juneton. Annual household income (ranked in $10,000 increments) ranged from $40,000 to $100,000. (He had asked about income because, although income in itself is never a direct disease factor, it has been linked statistically with many medical conditions, and is therefore a common factor of statistical adjustment.) All twenty were in good overall health, apart from their Funk's, with no serious underlying conditions.

In the meantime, and toward the end of his data collection on cases, Who had an assistant make a general review of basic hospital admission data. The assistant collected basic information on forty potential "control" (or comparison) patients. Who had decided to use non-critical flu and pneumonia patients as his controls. Flu and pneumonia are common, occur commonly amongst otherwise healthy persons as is the case with Funk's, but clearly have an origin (pathogens) unrelated to Funk's, and are not evidently related to jostling of internal organs, as Who suspected was the case regarding Funk's, so flu and pneumonia patients should provide a good, fair, and ample comparison group.

The assistant had been asked to cull hospital admission records of potential control or comparison patients *only* on the bases of non-critical flu or pneumonia diagnosis without

any serious underlying condition, and the partial background criteria of sex, age, and town of residence. Who first chose thirty-five from the forty as best candidates. He then asked his assistant to request the permission of these thirty-five for participation in the study, to ask them if they had a history of a Funk's Syndrome diagnosis in the past (none did: any who had would have been excluded), and to ask about their household income level, which was not included in basic admission data.

Of the thirty-five, four simply refused to participate, and another three, though willing on general terms to participate, refused to discuss their income. From the remaining twenty-eight Who found he could make twenty reasonably close one-to-one case-to-control matches in regard to his several backgound criteria of sex (since Who's case group was ten men and ten women, he chose the same 50-50 split for his twenty controls), race (the region was predominantly Caucasian, as were all cases and controls), age (same within five years either way one-to-one), town (he chose to consider Wallyton, Beaverton, and Juneton, all proximate middle-class residential towns, to be exactly equivalent, and ultimately rejected controls from outside these three towns), and household income (same within $10,000 either way one-to-one).

Had he not been able to match cases with controls with any specificity on a one-to-one basis, he might have settled for matching the two groups on a general or average basis only, but by allowing some leeway in his definitions, he considered he had achieved fair one-to-one matching, and on average the two groups as two wholes also sized up similarly on all counts as well. Thus Doctor Who felt he could present his results without adjustment for background factors.

With his controls now chosen, Who visited or telephoned these twenty, reaching them according to mutual convenience, and questioned them on two crucial topics:

1.) Occupation title and job description including amount of job-related driving required (in miles per year, excluding commuting).

2.) He asked: over the past twenty years, including both personal and job-related driving, and including commuting to work, have you, on average, driven more than or less than 10,000 miles per year?

When interviewing on these points, Who made notes, with both cases and controls, of their specific driving histories and habits as they described them, in case certain factors should stand out as being worthy of detailed analysis or comment in his published paper.

Who then set about trying to standardize job types into comparable designations for presentation and discussion in his paper, and he decided more generally to classify any job requiring on average more than 5,000 miles per year on the road, exclusive of commuting, as a "high driving intensity occupation", and any requiring less than 5,000 miles per year along the same lines as a "low driving intensity occupation."

For his overall driving stratification, he defined those who in working, commuting, and all other personal driving, exceeded 10,000 miles per year on average over the past twenty years as having a "high driving intensity lifestyle", and those who drove less as having a "low driving intensity lifestyle".

For simplicity and brevity, on tables, he showed responses as "high intensity/work" with yes/no counts and "high intensity/overall" with yes/no counts. Responses from each of the sexes, within each group, corresponded closely, so Who presented results for the sexes combined and not individually.

Amongst his twenty cases, at work specifically, exclusive of commuting, 8 drove heavily, and 12 did not. Amongst the controls, 2 drove heavily at work, and 18 did not. Let us show this on a table. A table of this type is called a "two by two" or "cell count" table.

High Intensity / Work	Cases	Controls
Yes	8	2
No	12	18

To compute the odds ratio ("relative risk") multiply from left to right, first from the top to the bottom (8 x 18 = 144) to create a numerator, then left to right from the bottom to the top for a denominator (12 x 2 = 24). 144 / 24 = 6 (represented in study as 6.00). That is the relative risk result for the "High Intensity/Work" category.

Now to the overall driving question. In terms of total driving (work-related, commuting, and all personal), for cases, 9 drove heavily, and 11 did not. For controls, it was evenly split, 10 and 10. Cell count chart below.

High Intensity / Overall	Cases	Controls
Yes	9	10
No	11	10

Numerator (left to right, top to bottom) is 9 x 10 = 90. Denominator (left to right, bottom to top) is 11 x 10 = 110. Result, 90 / 110 = relative risk 0.82.

A relative risk of 1 (or 1.00) indicates no change from a norm. The 6.00 relative risk for specific on-the-job driving suggests six times the norm. The 0.82 relative risk for overall driving is below the norm.

Amongst Who's cases, one drove a large tractor trailer truck professionally, and one of his controls also drove such a vehicle professionally. The other heavy at-work drivers included three executives who made regular and lengthy business trips to regional corporate facilities (two cases and one control), and, amongst the cases, two travelling salesmen, a taxi driver, an architect often called to far-flung construction sites, and a courier. Apart from the two trailer-truck drivers all of the specific job-related drivers drove conventional passenger vehicles. Doctor Who decides that vehicle type does not have a demonstrable effect vis a vis his total study group of cases and controls.

While there were more job-related heavy drivers amongst cases, more of the controls had longer daily commutes to workplaces, and typically did more personal driving, much of it purely recreational. A general speculation one might make, though Who does not, is that persons obliged to drive a great deal at work might get enough of it in that way, and so avoid it on their time off more avidly than would others.

One might also say that Who's contention that miles driven equates to risk of Funk's Syndrome is not confirmed by his study. In so saying, one might point to the tiny case sample of twenty persons (studies on case groups as small or smaller than this are not uncommon), and offer the opinion that studies as small as this are bound to produce fluke results of no applicability to the general population, that such studies are very likely to produce nonsense and confuse rather than illuminate. Would you put faith in a public opinion poll on an important social concern which was compiled via twenty random respondents?

One might consider that the more inclusive "overall" category result outweighs the more contrived "job-related" result if one considered that either result had any value. Really

neither does though. The sample size is ridiculous and the relevant questions asked require a level of recall and precision guaranteed to produce unreliable responses from most respondents.

Doctor Who, if he is a typical biostatistician of this boggled era, and he is, will not consider any of these things. He will focus with delight on his larger relative risk (hereafter to be referred to as RR) of 6.00 and he will also point to something called "statistical significance".

So what is that?

Statistical significance tests are tests of statistical data, based on various theoretical models, designed to suggest whether, on the basis of accounting for random error, a given RR computation should be considered self-negating. A self-negating RR computation is one which can – whether high or low in itself – be considered, on the basis of accounting for such random error, to support the so-called "null hypothesis" (RR 1.00: no difference from the norm).

These significance tests, which include among others the popular "Pearson's chi square" and "Fisher's exact test", produce results which may in some cases be referred to as a "P value" (or "probability value", which, in most common usage, is considered to suggest "statistical significance" if it comes out as *less than 5%* (0.05) and "statistical insignificance" if it comes out as *5% or more*.)

In recent decades, while P values are still used, one more often sees "confidence intervals" applied to individual RR results, and these are most usually computed using what is known as the "Mantel-Haenszel method with 95% confidence level"; this is another test modelled on and similar to Fisher's and Pearson's.

A confidence interval (hereafter CI) is displayed as a range which is theoretically supposed to suggest what the RR might actually be in reflection of the random play of chance; if the "confidence level" is stated as 95%, as conventionally (but not always) is the case, the idea is that the actual RR, even in reflection of random chance, still *probably would not* lie outside the range shown, in 95 of 100 such computations, although, again in reflection of chance, it *would probably* lie outside that range in about 5 cases out of a hundred such computations.

The CI range displayed is supposed to suggest "statistical significance" with (typically but not always) 95% confidence if it *does not* include "the null" (1.00) or "statistical

insignificance" if it *does* include or encompass 1.00.

A CI of, for instance, 1.01-2.01, since it does not include 1.00, would be taken to suggest "statistical significance". A CI of 0.99-2.01, or of 1.00-2.01, would suggest "statistical insignificance" since those do include or encompass 1.00.

The term "*statistical* significance" creates confusion amongst the public (and, pathetically, amongst most of today's "authorities" as well) because unknowledgeable or bovinely thoughtless persons tend to equate it with *actual* or *practical* significance. Statistical significance implies no such thing.

Statistical significance is the most base of standards, theorized along the lines of accounting *only* as to whether a given RR is *self-negating* (*not* whether it has any real value or applicability apart from reflecting the responses of study subjects themselves), and at this *only* considering what statisticians call "random error" (i.e. pure chance).

The statistical significance test assumes that there is no "systemic error" (i.e. that the statistical study is soundly rational in its premise, perfectly designed, and that data included – in our Doctor Who example, the questionnaire responses – is perfectly reliable and correct).

In many studies, systemic error is positively rampant, and when, as in our tiny Who study example, this results in a large whole number RR such as the 6.00 result (or also with a very low decimal RR result such as 0.20), the test of random error is positively *overwhelmed* by the effects of gross systemic error, such that the *most flukey* results are actually *likely* to come out as "statistically significant" in many cases.

The debased and pitifully frail standard of "statistical significance" has been criticized for decades and continually up to the present. For instance, in a 2004 paper ("The Missed Lessons of Sir Austin Bradford Hill") researchers Carl V. Phillips and Karen J. Goodman reviewed numerous warnings that Doctor Hill voiced decades ago regarding the necessity of sound judgement on various crucial levels when practicing biostatistics, and what he saw even then as a debasing of epidemiologic standards.

Phillips and Goodman lament: "Overlooked are Hill's important lessons about how to make decisions based on epidemiologic evidence. He advised epidemiologists to avoid over-emphasizing statistical significance testing, given the observation that systemic error is often greater than random error."

Hill, in his own words, addressed this repeatedly, most famously in a celebrated speech on medical statistics at the University of London in 1965, later published and widely circulated: "No formal tests of significance can answer these questions [of logical inference from statistical association]. Such tests can, and should, remind us of the effects that the play of chance can create, and they will instruct us in the likely magnitude of those effects. Beyond that they contribute nothing to the 'proof' of our hypothesis."

Overlooked, indeed, have been such cautions to avoidance of over-interpretation, to prudence in evidentiary review, and specifically against reliance on the base and flimsy standard of "statistical significance", for a very long and wearying period of time. But our Doctor Who, like the esteemed doctors Gupta and Clanton, and innumerable other modern health "experts"worldwide, surely would overlook all of this.

Doctor Who would, most likely, produce his confidence intervals via the Mantel-Haenszel method with 95% confidence level, probably using the popular EpiCalc computer program, as we shall do here (and elsewhere in other circumstances, by the same method, from time to time henceforward in this paper.)

Who's result for specific work-related driving, with CI, would be: RR 6.00 within CI 1.08-33.28.

Note the *vast* CI range, typical of the more comical RRs. Is a professional driver's risk really almost exactly 1, just a .08 hair above the norm? Or is it more like *33 times the norm*? Don't miss the humor in that. Or in the galling pretension of the decimals which are almost invariably presented. You'll see similar, and even far worse, in numerous results reported verbatim from real studies published in real "professional" journals, presented here later on.

Who's more encompassing result, for overall driving, would be presented as: 0.82 within CI 0.24-2.84.

What to make of that one? More driving cuts your risk of Funk's Syndrome to about a quarter of the usual? We don't really know what "the usual" (the base risk) is either; neither does Doctor Who, and he doesn't care, never mentions or even much thinks about it. Or maybe, gosh, it's that more driving multiplies your Funk's risk by something like three frightening times? That's less than 33 but maybe still kinda scary. Oh, dear. What to make of it all? How about making nothing of it? That's all it's worth.

What to make of Who's whole study? There must be plenty of waste baskets at Suburban Hospital. That's where Who should have put his idea for a study of a measly handful of patients in the first place. But he didn't reject his trash idea and he's actually now in love with his trash result.

So the end of the Who and Watt story is this. The two statistical dunderheads, perfectly biased toward interpreting results according to their pet theories and preconceptions, decide to trumpet in publication that the 6.00 work-related result is pregnant with vast meaning, necessarily, they will say, because it is statistically *significant*.

They underscore the statistical *insignificance* of the 0.82 result for Who's overall category. In other circumstances Who and Watt often believe devoutly in statistically insignificant results, but since this one displeases them, they denigrate it.

Who, in his study's conclusion, pleads the urgent need of further study and Watt, in an accompanying editorial, does the same. Newspapers pick up the buzz from the medical community and announce in headlines: "Professional Drivers at Grave Risk of Funk's Syndrome".

Nephrologists who subscribe to Watt's journal, and newspaper readers across the state and the world, are all soberly impressed. So is the director of a state health agency funded by tobacco tariffs, who awards Who a grant of $150,000 for further research on Funk's. Doctor Who's second career is assured and, with successive grants into the future, he carries on producing epidemiologic drivel on many topics for decades to come.

So much for Who and Watt, and let us return, enlightened by their professionally poor but technically revelatory example, to the question at hand: what is the relative risk, and absolute risk of lung cancer, for "ever-smokers".

Though not to say that studies as poor in method as Who's have never been conducted on this question, here we do have a question worth asking, and many studies on it have been conducted admirably. These include numerous cohort (or prospective) studies amongst a majority of case/control (or retrospective) studies.

Let us describe the difference between the two study types here. We have illustrated a case/control study with our Who example. Such studies are called "retrospective" because they inquire of cases after diagnosis rather than before.

Cohort studies are produced by gathering data on a wide group of persons (sometimes tens of thousands, though in other cases a vastly smaller number) via questionnaire, and *years later* reviewing particular disease incidence amongst respondents in light of lifestyle factors already accounted for. Periodic updates to original questionnaire responses are usually made. The Prescott 1998 study, referred to earlier in our discussion of base risk, is a cohort study. Cohort studies are called "prospective" because the lifestyle data was generally received before, rather than after, diagnosis.

The method of comparing afflicted with unafflicted remains essentially the same with both study types. With cohort studies, if considering lung cancer incidence, for example, the portion of the cohort without lung cancer serves, in effect, as the "control" or comparison group, relative to the "cases" who do have lung cancer.

"Cell counts" for cohort studies are assembled similarly as we have shown in the Who example. As previously explained, "relative risk" results for typical cohort studies may commonly be produced either by "risk ratio", a slight variation on the odds ratio computation shown in the Who example, or also commonly by the very same odds ratio computation.

It is often said that cohort studies are superior to the case/control type in having the specific attribute of minimizing "recall bias" (for example, with all the publicity over years about the smoking link, if asked about smoking history *after* lung cancer diagnosis, a smoker may emotionally tend either to exaggerate smoking history, out of an impulse of self-condemnation, or to minimize it, out of an impulse to self-absolution, but if asked his smoking history *before* diagnosis, when likely feeling well, he might be expected to have answered more objectively, and therefore more reliably.)

Many, though not all, cohort studies will also report on a considerable number of lung cancer cases. A larger case count increases the possibility of rough applicability to the population at large (whereas, in a case such as our fictional Who study – not an unreasonable example as studies on mere handfuls of patients disgracefully proliferate in the literature – the sample of patients is so pitifully low that results could not have anything close to general pertinence except through pure luck.)

This points to an important caveat: if you see a headline such as "Study of 50,000 Shows Driving Causes Funk's Syndrome", the headline will generally be stating the size of the whole cohort. If the number of Funk's Syndrome cases amongst that cohort is 20, a ridiculously small sample, comparison with the other 49,980 in the cohort will still produce

what is probably a meaningless result (not to mention that the headline is defining a statistical link as denoting "cause": a cardinal sin in itself.)

Cohort studies, on the other hand, suffer from having, in effect, a widely disparate "control" group. The lung cancer patients, for example, may be decidedly older on average than are the members of the cohort as a whole. They may differ decidedly and crucially in other ways as well. This means that cohort RR results may often need considerable adjustment for background factors. Each adjustment is really an approximation – derived via subjective choice between various methods of reckoning – of the supposed effect of a given background factor.

There are, however, case/control studies of considerable size, comparing in this respect to cohort studies, and studies of both types, when well-performed, are worthy of close review. So let us now get to that.

Specific reference to the risk of lung cancer amongst smokers, in the medical literature, dates back to 1911. The American Doctor Isaac Adler, in his monograph *Primary Malignant Growths of the Lung and Bronchi,* tentatively suggested his suspicion that lung cancer could be related to tobacco use.

In 1929, German physician Fritz Lickint, in his treatise *Tabak und Tabakrauch als ätiologischer Factor des Carcinoms* (*Tobacco and Smoking as Aetiological Factors of Cancer*), cited research suggesting four to five times as much lung cancer in Germany's men as in her women, ascribing this in his opinion to high male versus low female smoking rates of the time, and also indicting the increasing popularity of cigarettes particularly. Lickint did no specific study to investigate his theory.

Some other published research addressed the tobacco / lung cancer question tentatively in the nineteen-thirties and 'forties. Notice was given to crude but specific research by the German Franz Hermann Müller (whose findings were published both in Germany and in the *Journal of the American Medical Association* in 1939.) Similarly crude research was conducted by the Germans Eberhard Schairer and Erich Schöniger, acting as a team, in 1943; in the midst of the war, Schairer and Schöniger's research received scant worldwide attention, but after the war it received some notice. These German researchers variously suggested a heightened lung cancer risk for smokers which can be computed by conventional modern methods to suggest an RR for ever-smokers in the area of 3 to 6.

Beginning in about 1950 in the West, and later in the same decade in the East, research on

smoking and lung cancer burgeoned, and at its best became more sophisticated. Today this research comprises hundreds of better and worse biostatistical studies on the subject.

For ever-smokers, overall, studies in the early decades of research are fairly consistent in suggesting a low single-digit risk, both in the West and in the East. From about 1970, and accelerating from 1980 onward, however, the RR reports grow, in the West rather more than in the East. An overview of research from its beginnings to the present, eliminating the far extremes, would suggest an RR for ever-smokers as a whole number single digit risk, i.e. something between about RR 2 and RR 9.

Now, all of these studies approached the very same question, of an ever-smoker's risk. The 2 to 9 RR range suggested (and this eliminates extremes) reveals the *great imprecision* of lifestyle epidemiology. Nevertheless, that there is a link, seems clear.

Why do we say this?

Generally, in terms of common sense:

1.) To anyone who smokes cigarettes considerably, irritation of the lungs over time is self-evident. If a person smokes heavily he will come to recognize his "smoker's cough". The cough is due primarily to irritation of the breathing passages of the lungs, and it is particularly cancers of these breathing passages, to which smokers are at heightened risk.

Though not really surprising in itself, that irritated tissues are more vulnerable to cancer, likewise appears evident based on wider observation. For example, excessive tanning enthusiasts may be subject to skin cancer. Heavy drinkers may be subject to digestive system and liver cancers. Gynecologists know that women who have had HPV (human papillomavirus) infection (genital warts) carry a heightened risk of subsequent cervical cancer.

That a long-term and considerable smoking habit, continued beyond middle age, should bring about a heightened lifetime lung cancer risk, eminently meets the test of common sense.

More particularly, upon review of the lifestyle epidemiology:

2.) RR reports, for lung cancer generically, amongst ever smokers, only uncommonly fall below 1.00 and there has not evidently been a "statistically significant" result below 1.00

published regarding lung cancer generically.

There is a case, still famous in that it inspired a kind of feud between some well-known British researchers, whereby Ronald Aylmer Fisher, an originator of lifestyle epidemiology methodology, pointed out that data in a 1950 study by Richard Doll and Austin Bradford Hill suggested with statistical significance that inhaling tobacco smoke, as opposed to not inhaling, was actually beneficial toward avoiding lung cancer. That was true (the result for risk from inhalation in the 1950 study computes in today's standard method of presentation to RR 0.78 within CI 0.63-0.98) but such exoneratory results are by no means typical.

3.) There is what lifestyle epidemiologists call a "dose response".

Those who have smoked little show no increased lifetime risk. For the limit of this, the often cited "20-pack-year" level provides a fair yardstick (of course and as we have discussed, meaning no *heightening* of risk – or increase in relative risk from the normative level of 1 – up to this level of smoking, rather than the common but foolhardy assessment of "zero risk".)

Beyond this, those who have smoked appreciably show appreciably increasing risk according to intensity and duration of the smoking habit: the RR for a class of smokers within a study who smoked, for instance, multiple packs per day for fifty years, will, with fair consistency, be considerably higher than the RR for another class who smoked one pack per day for thirty years.

As with RR figures between studies, and as for most everything in lifestyle epidemiology, the evidence of increasing risk with increased smoking is not by any means perfectly consistent or numerically uniform from study to study, and one will find individual studies where RR figures *decline* with heavier smoking levels, *but this is not common*. It does appear, as follows logically if cigarette smoking is an irritant inviting lung cancer, that more smoking over a longer time means higher risk.

Thus, primarily and simply based on these three crucial points, we can indict long-term and substantial smoking (in combination with advanced age as we shall illustrate) assuredly as the most common factors influencing lung cancer amongst the population at large.

Now, we have said that RR indications for the ever-smoker class typically range between about 2 and 9. The higher numbers show up mostly in the West and, by far, mostly over the past thirty years or so. Previous to that, one might have reconciled the figure, for both

West and East, at the low end, or at between about 2 to 4. Our initial focus, however, is in building an overview of the present era in the West through the perspective of recent US lung cancer mortality statistics and other crucial data.

For this purpose one can reconcile that RR for the ever smokers group, in the West, would lie in the mid-range of 2-9, or at about 5 or 6. This is the approximate level we shall include for the overall perspective tables to be presented for the present era, on which we will divide the ever smokers group into two categories, one of these representing those who smoked beyond the approximate "20 pack-year" limit, and the other category representing those who quit before reaching this limit, or else never effectively reached it because they smoked very moderately.

At various times over the decades, typically following release of one or another particular study's results, articles have been written suggesting either that men are at a higher lung cancer risk from smoking, or that it is the women who suffer more from smoking.

Decades ago the more common suggestion was that men were more vulnerable. After about 1990 it became more the fashion to allege that women have the greater vulnerability. Looking at the body of research as a whole it seems that the risk is about the same for smokers of both sexes. Formal wide-ranging studies of this specific question in both Europe and the US bear this out.

Excerpt follows from a 1998 article in the journal *Epidemiology* entitled "Gender and Smoking-related Risk of Lung Cancer" by Eva Prescott, et alia, who studied populations in Denmark:

> Our aim was to compare risk of lung cancer associated with smoking by gender and histologic type. A total of 30,874 subjects, 44% women, from three prospective population-based studies with initial examinations between 1964 and 1992 were followed until 1994 through the National Cancer Registry. There were 867 cases of lung cancer, 203 among women and 664 among men. Rates among female and male never-smokers were similar, although confidence intervals around rates were wide. Rate ratios (RRs) increased with number of pack-years for both men and women to a maximum of approximately 20 in inhaling smokers with more than 60 pack-years of tobacco exposure. RRs did not differ much between men and women: adjusted for pack-years, age, and study population, the ratio between female and male smokers' RRs of developing lung cancer was 0.8 (95% confidence

interval = 0.3-2.1). All histologic types were associated with smoking, with the largest RR seen for squamous cell carcinoma and anaplastic carcinoma. This prospective population-based study does not confirm previous reports from case-control studies of a higher relative risk in women than in men for lung cancer associated with smoking.

Similar large-scale research in the US has produced the same conclusion:

Males, Females Have Same Lung Cancer Risk

Study challenges belief that women are more susceptible to smoking dangers

Harvard University Gazette, June 3, 2004

Researchers at Brigham and Women's Hospital (BWH) have found new evidence that suggests that women and men with similar smoking histories have the same risk of developing lung cancer. The large-scale analysis of more than 85,000 men and women shows that the nation's top cancer killer strikes male and female smokers at similar rates – a finding that contrasts with the popular belief that women are more susceptible to the disease. The research appears in the June 2 issue of *The Journal of the National Cancer Institute*.

"Our data indicate that women are not at an excess risk of lung cancer compared to men, given similar smoking levels and smoking histories," said Assistant Professor of Medicine Diane Feskanich.

An estimated 46.2 million adults in the United States smoke cigarettes. According to the National Cancer Institute, cigarette smoking is responsible for 87 percent of lung cancer deaths, and lung cancer is now the leading cause of cancer deaths among U.S. women and men.

The BWH and Harvard Medical School research team analyzed rates of lung cancer and compared them within several categories, including the number of cigarettes smoked per day, years of smoking, and age at start of smoking. Their report found that the overall risk of lung cancer did not differ by gender, contrary to earlier research.

> "Arguing that female smokers are at an increased risk of lung cancer is losing validity and thus studies aimed to examine biological differences may be less warranted," noted Feskanich. "Instead, resources should be heavily invested in programs and projects that aim to reduce smoking across the board, especially in young people where tobacco use is on the rise."

The confusion over the years about whether one or the other sex might be more vulnerable to adverse effects of long-term smoking is a reflection of the very rough accuracy of epidemiological studies, individually, and even taken as a whole. Looking at one study after another a reader will receive one after another alternating impression regarding sex or other factors. The studies are simply imprecise: rough indicators. To a balanced perspective, however, the idea that smoking considerably over a long time increases statistical lung cancer risk, about equally for men as for women, is apparent.

If an ever smoker RR of about 5 or 6 is representative of the West today, for the East, one could still reconcile closer to between about 2 to 4. Following our tables illustrating the broad view of Western lung cancer epidemiology we shall discuss Eastern figures for comparison; some areas of the East have a particularly illuminating aspect: a nearly entirely never-smoking female population; it is on this female population that our East/West discussion will take focus.

The East/West RR divide is accompanied by a base risk divide: Eastern RR figures tend to be *lower* than those seen for the West, but Eastern base risk figures tend to be *higher* than those seen in the West. This points strongly to diagnostic bias (also called detection bias.)

What is diagnostic bias? Simply this:

Cancer can grow insidiously nearly anywhere in the body over months or even years without producing serious symptoms. One of the great problems with cancer is that it "metastasizes", or spreads, from one part of the body to another.

Cancer is diagnosed according to its "primary site" within the body, i.e., where it first began. If it began in the brain the patient is diagnosed with primary brain cancer, if in the breast with primary breast cancer, and so on.

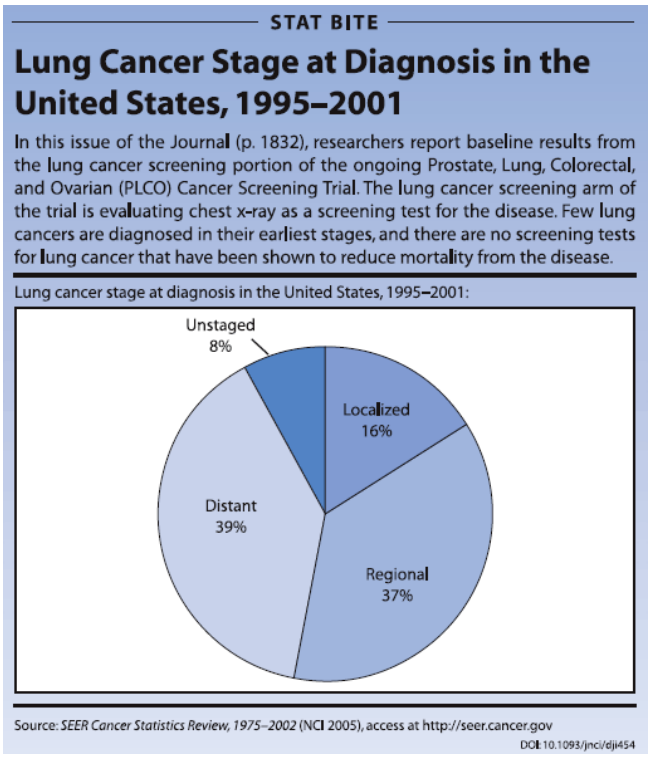
The primary diagnosis remains regardless of future developments. A patient with breast cancer, by example, might have the breast tumor seemingly successfully removed, but ultimately die not long after from a metastatic tumor which came to grow in the brain. Still,

the primary diagnosis remains breast cancer, and that is properly shown on the death certificate (as with all things related to cancer statistics, there is some play of error in this, but recent research has estimated that US physicians and coroners designate the primary cancer site as it was originally diagnosed on approximately 85% of cancer-related death certificates generally, and also about 85% of the time, specifically regarding lung cancer.)

It is very common that patients will not show symptoms, or be diagnosed with cancer, until their cancer has already grown and spread to more than one area of the body. As with the brain, and even more so, the lungs frequently become the sites of metastatic, or secondary, tumors.

Now, as we have seen, and shall keep seeing as our analyses proceed, there is a deep-seated and actually eerie level of bias amongst health professionals regarding smoking and lung cancer. Despite all evidence, despite the obvious fact that never-smokers and those who quit smoking before "20 pack-years" can still get lung cancer, they remain stubbornly resistant, virtually defiant, toward that idea.

At the very same time, although it is commonly known that around or about 9 out of 10 ever-smokers will never get lung cancer, and while the "authorities" obdurately insist on the "zero risk" of those who quit smoking before middle age, they also, simultaneously and with astonishing foolishness, will *insist* that *all* lung cancer in *all* ever smokers is caused, in an absolute sense, by smoking. This we shall illustrate further on.



Biased clinicians clearly *expect* to see lung cancer in any or all persons with any smoking history, and therefore *do* see what they want to see with a hopelessly biased perspective: as Doctor Michael McFarlane noted in a 1986 study of bias in lung cancer diagnosis: "[I]f a lesion was present, chest films were more likely to be radiologically interpreted as a cancer in smokers."

Furthermore – note illustration at left from the *Journal of the National Cancer Institute* (2005) – which shows that only 16% of persons, when diagnosed with lung cancer, have localized lung cancer: i.e cancer

located only in the lung. In the great majority of cases when lung cancer is diagnosed the patient already has tumors in two or more parts of the body, either in near or distant regions from the lung. Added to this: the NCI also reports: "The most common sites of cancer metastasis are the lungs, bones, and liver."

When clinicians treat smokers they have an unrealistic expectation that the smokers *will* get lung cancer. When cancer actually comes to be suspected they tend to *see* lung cancer in X-rays whether it is there or not. If cancer does occur, with a primary site other than the lungs, it may very well spread before symptoms appear, and *very commonly* it will spread or metastasize to the lungs. Sometimes, relative growth of tumors in various locations within the body, or the nature of cancer cells, may give strong clues to where the cancer originated. In many cases, however, strong evidence will be lacking to suggest the primary site.

When the question of the primary site is not clearly evident clinicians may opt to diagnose as "carcinoma of unknown primary origin" or "occult primary malignancy", but this option is only exercised in an estimated 2% to 6% of all cancer cases. Further description from the American Cancer Society:

> What is a cancer of unknown primary?
>
> Cancers often spread from their primary site (the part of the body where the cancer started) to one or more metastatic sites (other parts of the body). Cancers are named based on their primary site, regardless of where in the body they spread. For example, a lung cancer that spreads to the liver is still classified as lung cancer and not as liver cancer.
>
> Sometimes it's not clear where a cancer may have started. When cancer is found in one or more metastatic sites but the primary site is not known, it is called a cancer of unknown primary (CUP) or an occult primary cancer. This happens in a small portion of cancers.
>
> Further tests may eventually find the primary site of some of these cancers. When this happens, they are no longer considered a cancer of unknown primary and are renamed and treated according to where they started.
>
> As an example, a person may have an enlarged lymph node on the side of the neck. When it is removed, it is found to contain cancer. But under the

> microscope it does not look like a cancer that normally starts in lymph nodes. At this point it might be called a cancer of unknown primary. The way it looks under the microscope might suggest that the cancer started in the mouth, throat, or voice box (larynx). During a thorough exam of this area, a small cancer of the larynx might be found. From then on, the patient is said to have laryngeal cancer rather than a cancer of unknown primary and will get treated for that type of cancer.
>
> In many cases, the source of the cancer is never determined. Even the most thorough search may not find the primary site. Even when doctors do autopsies on people who have died of cancer of unknown primary, they are often still unable to find the site where the cancer started.
>
> When cancer is found, it is only natural to want to know where it came from. But the main reason to look for the primary site for a CUP is to guide treatment. Since a cancer that starts in one place needs the same treatments when it spreads, knowing where a cancer started tells the doctor what types of treatments to use. This [is] especially important for certain cancers that respond well to specific chemotherapy or hormone drugs. Knowing where the cancer started may tell the doctor which drugs to use to give the patient the best chance. When the types of cancer with the best hope for responding to treatment have been ruled out by certain tests, it usually becomes less important to find the exact origin or cancer type.
>
> But even if the primary site is not known, treatment can still be successful. How the cancer looks under the microscope, the results of lab tests, and information about which organs it has already affected can help doctors predict what kinds of treatment might be helpful.

Although a considerable proportion of cancer patients generally will present with cancer in more than one body region, and although the primary site is often subject to question, clinicians avoid the "unknown primary" diagnosis an estimated 94% to 98% of the time. They do so because patients find it disturbing when they cannot be told where their cancer began – as research has suggested, the patients tend to suspect ineptitude on the part of their physician in such cases – and also because knowledge of the primary site is an important factor for determining a suitable treatment plan. Thus physicians nearly always choose to diagnose a particular primary cancer based on what they consider their best judgement.

With all of this the opportunity for diagnostic bias is positively enormous and the result is inevitable: when faced with the typical situation of a patient who, upon cancer diagnosis, already has tumors in more than one area of the body, and where one of those sites is the lung, the biased perspective tends strongly to perceive that, if the patient is a never smoker, the cancer *cannot* have begun in the lung, but if the patient is an ever smoker, the cancer *surely did* begin in the lung.

Bias is a phantom. Specific statistical adjustment for it would require speculation but that strong diagnostic bias exists regarding lung cancer is clear indeed, simply from encountering clinicians' common statements revealing colossal over-interpretation of the smoking / lung cancer link, and this has long been apparent.

The medical community is not quick to investigate or report on its own biases – most clinicians today would deny bias and insist that their judgements were soundly scientific as surely as the eugenicists of a previous generation would insist on the same regarding their grotesque racial views – but researchers such as Doctor McFarlane, mentioned earlier, and Doctor Alvan Feinstein of Yale University, have produced research on such bias and have concluded that it is widespread.

As Doctor Feinstein, an early critic on general terms of epidemiological and clinical over-interpretation, wrote on the overall subject of diagnostic bias in 1974: "It seems important to recall that in epidemiologic surveys of causes of disease, the investigators get data about the occurrence of diagnoses not the occurrence of diseases, and that the rates of diagnosis may be affected by bias ..."

Doctor Feinstein summed up the particularly egregious bias surrounding smoking and lung cancer then in famously saying, "Cigarette smoking may contribute more to the diagnosis of lung cancer than it does in producing the disease itself." Things have only grown worse over time.

Naturally, there is a direct and unfortunate clinical result of biased diagnosis, both for ever smokers incorrectly diagnosed with primary lung cancer, and for never smokers who should have been diagnosed with primary lung cancer, but were not. Since cancer therapies are tailored according to the primary site of the cancer, when diagnosis is faulty through bias the patient may suffer from inappropriate treatment.

In terms of statistics, of course, the plain result of this diagnostic bias is that clinicians under-diagnose "primary lung cancer" amongst never smokers (thereby statistically

understating the base risk) and over-diagnose "primary lung cancer" amongst ever smokers (thereby statistically *overstating the relative risk from smoking*).

This is exactly the situation we see reflected in the East/West divide. Of necessity, given the virtually smokerless female population in portions of the East, the *opportunity* for diagnostic bias vis a vis that population dries up considerably, while at the same time Eastern clinicians have had to recognize throughout the decades that women they treat do indeed get lung cancer, even though very few of them smoke.

Eastern clinicians know, because they have been *forced* to see it all the time, that a nearly smokerless population certainly *can* get lung cancer. That practical situation, likewise, at least tempers the *inclination* to diagnostic bias.

But for now let us return to the specific question of the West, taking Western statistics at face value for the time being, and returning to the East/West divide upon completion of Western analysis on that "face value" basis.

To proceed from this point we need to acquaint ourselves better with the lung cancer population. To begin with, what is their vintage: how old are they?

For the answer to this let us refer to the US National Center for Health Statistics (NCHS) death counts, by cause of death, and by age at death, employing figures reflecting the most recent year for which final reports were available (2008) at time of composition. It may be noted that figures have been very similar over recent years. It may also be noted, for reference to NCHS tables, that what clinicians generally refer to as primary lung cancer is today specifically described in the International Classification of Diseases (ICD) as "malignant neoplasms of the trachea [windpipe], bronchus [main breathing passages] and lung [as a whole, excluding the pleura, or lining of the lung]".

Pertinent table from NCHS 2008 appears on following page. Age at death computations appear on succeeding page.

Table 10. Number of deaths from 113 selected causes, Enterocolitis due to *Clostridium difficile*, drug-induced causes, alcohol-induced causes, and injury by firearms, by age: United States, 2008

[The asterisks (*) preceding the cause-of-death codes indicate that they are not part of the *International Classification of Diseases, Tenth Revision* (ICD-10), Second Edition; see "Technical Notes"]

Cause of death (based on ICD-10, 2004)	All ages	Under 1 year	1-4 years	5-14 years	15-24 years	25-34 years	35-44 years	45-54 years	55-64 years	65-74 years	75-84 years	85 years and over	Not stated
All causes	2,471,984	28,059	4,730	5,651	32,198	42,275	76,370	186,542	296,182	401,579	653,560	744,691	147
Salmonella infections (A01-A02)	44	4	2	1	2	1	1	3	3	8	8	11	-
Shigellosis and amebiasis (A03,A06)	6	-	-	-	-	-	1	-	3	1	1	-	-
Certain other intestinal infections (A04,A07-A09)	7,876	8	6	-	4	15	39	142	435	1,003	2,791	3,432	1
Tuberculosis (A16-A19)	585	-	2	3	6	14	26	80	90	114	145	105	-
Respiratory tuberculosis (A16)	449	-	2	1	5	8	15	58	69	85	121	85	-
Other tuberculosis (A17-A19)	136	-	-	2	1	6	11	22	21	29	24	20	-
Whooping cough (A37)	20	18	-	-	-	-	1	-	-	-	-	1	-
Scarlet fever and erysipelas (A38,A46)	3	-	-	-	-	1	-	-	-	1	-	1	-
Meningococcal infection (A39)	102	9	11	8	19	11	11	8	6	8	6	5	-
Septicemia (A40-A41)	35,927	289	93	61	139	359	892	2,514	4,552	6,448	10,717	9,863	-
Syphilis (A50-A53)	34	-	-	-	1	-	2	3	5	3	7	13	-
Acute poliomyelitis (A80)	-	-	-	-	-	-	-	-	-	-	-	-	-
Arthropod-borne viral encephalitis (A83-A84,A85.2)	2	-	-	-	1	-	-	-	-	-	1	-	-
Measles (B05)	-	-	-	-	-	-	-	-	-	-	-	-	-
Viral hepatitis (B15-B19)	7,629	2	-	2	9	51	447	2,732	2,751	882	600	153	-
Human immunodeficiency virus (HIV) disease (B20-B24)	10,285	-	2	1	168	975	2,838	3,730	1,908	516	116	31	-
Malaria (B50-B54)	5	-	-	-	-	1	-	2	1	-	1	-	-
Other and unspecified infectious and parasitic diseases and their sequelae (A00,A05,A20-A36,A42-A44,A48-A49,A54-A79,A81-A82,A85.0-A85.1,A85.8,A86-B04,B06-B09,B25-B49,B55-B99)	5,914	148	66	46	78	107	219	507	912	1,129	1,557	1,145	-
Malignant neoplasms (C00-C97)	565,469	70	394	890	1,663	3,521	12,699	50,403	104,091	141,159	160,960	89,610	9
Malignant neoplasms of lip, oral cavity and pharynx (C00-C14)	8,019	-	-	2	17	53	236	1,144	1,995	1,941	1,633	998	-
Malignant neoplasm of esophagus (C15)	13,714	-	-	3	3	29	236	1,441	3,304	3,838	3,443	1,417	-
Malignant neoplasm of stomach (C16)	11,352	-	-	1	20	129	407	1,182	1,978	2,558	3,059	2,018	-
Malignant neoplasms of colon, rectum and anus (C18-C21)	53,321	1	-	1	52	314	1,419	4,802	9,076	11,770	14,936	10,948	2
Malignant neoplasms of liver and intrahepatic bile ducts (C22)	18,213	1	20	17	38	81	335	2,480	4,737	4,105	4,465	1,934	-
Malignant neoplasm of pancreas (C25)	35,236	1	-	1	7	53	506	2,840	6,681	9,115	10,414	5,616	2
Malignant neoplasm of larynx (C32)	3,760	-	-	-	-	3	52	411	973	1,136	853	332	-
Malignant neoplasms of trachea, bronchus and lung (C33-C34)	158,656	4	3	5	29	145	1,604	12,532	30,796	48,293	47,948	17,297	-
Malignant melanoma of skin (C43)	8,623	2	1	-	40	188	412	1,104	1,742	1,897	2,123	1,114	-
Malignant neoplasm of breast (C50)	41,026	-	-	-	13	331	2,148	5,962	8,797	8,441	8,820	6,514	-
Malignant neoplasm of cervix uteri (C53)	4,008	-	-	-	13	152	609	977	880	653	473	251	-
Malignant neoplasms of corpus uteri and uterus, part unspecified (C54-C55)	7,675	-	-	-	6	30	147	612	1,702	2,032	1,896	1,250	-
Malignant neoplasm of ovary (C56)	14,362	-	-	3	12	94	379	1,539	2,921	3,609	3,783	2,022	-
Malignant neoplasm of prostate (C61)	28,472	-	1	-	-	1	23	458	2,385	5,645	10,721	9,237	1
Malignant neoplasms of kidney and renal pelvis (C64-C65)	12,895	1	12	33	24	49	225	1,268	2,679	3,211	3,436	1,957	-
Malignant neoplasm of bladder (C67)	14,036	1	-	-	3	14	95	592	1,600	2,952	4,932	3,847	-
Malignant neoplasms of meninges, brain and other parts of central nervous system (C70-C72)	13,724	11	119	299	220	362	844	2,016	3,161	3,139	2,587	966	-
Malignant neoplasms of lymphoid, hematopoietic and related tissue (C81-C96)	54,954	28	127	287	594	813	1,320	3,555	7,859	12,329	17,591	10,449	2
Hodgkin's disease (C81)	1,171	-	-	1	56	94	117	146	177	210	256	114	-
Non-Hodgkin's lymphoma (C82-C85)	20,369	1	4	34	127	221	469	1,290	2,910	4,413	6,791	4,108	1
Leukemia (C91-C95)	22,335	27	123	252	410	490	621	1,436	2,956	4,798	6,840	4,381	1
Multiple myeloma and immunoproliferative neoplasms (C88,C90)	11,020	-	-	-	1	8	112	679	1,811	2,898	3,676	1,835	-
Other and unspecified malignant neoplasms of lymphoid, hematopoietic and related tissue (C96)	59	-	-	-	-	-	1	4	5	10	28	11	-
All other and unspecified malignant neoplasms (C17,C23-C24,C26-C31,C37-C41,C44-C49,C51-C52,C57-C60,C62-C63,C66,C68-C69,C73-C80,C97)	63,423	20	111	238	572	680	1,702	5,488	10,825	14,495	17,847	11,443	2

Age at death computation table appears below. On left, for general reference, age at death from all causes. On right, age at death from lung cancer specifically. Average age at death is computed. The construction of "life expectancy" which one often comes across is a statistical abstraction estimating remaining life probability – from birth or from a stated age – based on theoretical assumptions. What is computed here is a plain average of ages at death with average age for "over 85" group cited as 91 based on average life expectancy probability at age 85 as per NCHS 2008.

Average Age at Death, USA 2008, All Causes (Total of deaths excludes 147 certificates lacking stated age)				Average Age at Death, USA 2008, Lung Cancer (No exclusions)			
Age group [multiplier]	# Deaths in age group	Proportional multiplicand	Product / Addend	Age group [multiplier]	# Deaths in age group	Proportional multiplicand	Product / Addend
All ages	2,471,984	N/A	N/A	All ages	158,656	N/A	N/A
Under 1 [0.5]	28,059	.011351	.005676	Under 1 [0.5]	4	.000025	.000013
1-4 [2.5]	4,730	.001913	.004783	1-4 [2.5]	3	.000019	.000048
5-14 [9.5]	5,651	.002286	.021717	5-14 [9.5]	5	.000032	.000304
15-24 [19.5]	32,198	.013025	.253988	15-24 [19.5]	29	.000183	.003569
25-34 [29.5]	42,275	.017102	.504509	25-34 [29.5]	145	.000914	.026963
35-44 [39.5]	76,730	.03104	1.22608	35-44 [39.5]	1,604	.010110	.399345
45-54 [49.5]	186,542	.075462	3.735369	45-54 [49.5]	12,532	.078989	3.909956
55-64 [59.5]	296,182	.119816	7.129052	55-64 [59.5]	30,796	.194105	11.549247
65-74 [69.5]	401,579	.162452	11.290414	65-74 [69.5]	48,293	.304388	21.54966
75-84 [79.5]	653,560	.264387	21.018766	75-84 [79.5]	47,948	.302214	24.026013
Over 85 [91]	744,691	.301252	27.413932	Over 85 [91]	17,297	.109022	9.921002
Total: average age at death from all causes			73 years old	Total: average age at death from lung cancer			71 years old

As is the case with the majority of cancer types, age is a clear factor – as clear a factor as is considerable smoking over a long period – with lung cancer, which is primarily a disease of old age. The average age at lung cancer death, at 71, is similar to the average age at death from all causes, at 73. About three quarters of lung cancer deaths (72%: 113,538 / 158,656) occur in persons over age 65. Nearly all (99%: 156,866 / 158,656) lung cancer deaths occur in persons over age 45.

Let us say, then, for purposes of our forthcoming overview tables, that the present era's typical lung cancer patient is in the vicinity of 70 years old – suggesting the "vintage

population" which is equivalent to the "mortality population" – therefore that he or she would typically have been born in or around the Second World War era, and then let us move from there to the crucial question: how much, of what forms of tobacco products, and for how long are persons of this general vintage likely to have smoked (ever smokers) or not (never smokers).

Over the decades, a goodly amount of research has been done on US tobacco consumption, some by health community researchers, some by government tax bureaus, and quite a lot by the tobacco industry.

Records on the subject are widely available. Some are quite general. Others stratify considerably. It's ironic, given today's circumstances, that on tables stratified by profession, male physicians sixty years ago smoked at a rate rivalled amongst few professions (these including male journalists and military men) of about 75%.

Comparing records more generally, it is apparent that cigarettes (practically unknown until the mid-nineteenth century) began to catch on as milder tobaccos and mechanized production appeared in the late nineteenth century, and that cigarette consumption grew exponentially from about the First World War era. Today's lung cancer vintage population much preferred cigarettes to cigars and pipes.

Less than ten per cent of this vintage smoking population (nearly all men) were primarily cigar or pipe smokers. Consumption level research suggests that many of the cigar smokers, in particular, would have smoked only occasionally. The lung cancer risk with cigars and pipes is also much lower than is the case with cigarettes. We shall consider the elements of cigar and pipe smoking, together with low level cigarette smoking, as moderating factors in the overall picture of this predominantly cigarette smoking population.

The typical smoker began the habit in his or her late teens or early twenties. That would equate, typically, to about the early nineteen sixties. This is the period in which smoking prevalence reached its very height (circa 1960 for men and circa 1965 for women.) At its height, smoking prevalence amongst the adult population was right about 50%, greater for men (about 60%) than for women (about 40%). The proportion of the overall US population has always been, throughout the period in question, very close to 50/50 male/female.

The most common level of cigarette smoking (what statisticians call the mode) was right around a pack (20 cigarettes) per day, but average (or mean) consumption was lower at

about 15 cigarettes per day: persons who smoked under the pack per day level outnumbered those who smoked more than a pack per day.

Beginning in the mid-'sixties (the 1964 Surgeon General's report had much to do with this) the sharpest drop-off in smoking yet seen ensued. Over the next twenty years, from the mid-'sixties to the mid-'eighties, smoking prevalence dropped consistently, by about 40%, from its peak of 50% down to about 30%.

After that, despite increasingly harsh anti-smoking measures, the rate of decline slowed. Today something like 20% of the US adult population smokes, but recent figures are less reliable, since with ever-increasing tobacco taxes and tariffs, the "grey" and "black" markets for tobacco have been growing indecipherably.

For our overview let us speak in round numbers. About half of the vintage population would be ever smokers. About half would be never smokers. Regarding ever smokers, roughly 40% would have quit within twenty years of establishing a smoking habit. About 10% more would have smoked for a longer time, but very moderately, such that they would not reach a "20-pack-year" level in a lifetime.

Regarding moderation in smoking: as it was with the US alcohol prohibitionists of a century ago, who ridiculed the idea that any level of drinking was moderate and spoke of the "fatal glass of beer", amidst today's tobacco abolitionists the idea of moderation in tobacco use is likewise ridiculed. Ridicule of moderation is a hallmark of fanaticism. Moderation, however, is a virtue at all times, even during crazy times.

Moderation is likewise a virtue in all things but in nothing more than in thinking. We can do well, for instance, to recall the verified longest-lived human in history. The gracious French lady Jeanne Calment (21 February 1875 – 4 August 1997: pictured at left) reported that she drank wine and smoked cigarettes, every day from a young age, for a period of about one hundred years.

By all accounts Jeanne Calment was moderate in her habits. She died in her sleep when her heart stopped. She was one hundred twenty-two years old. We can also do well to note

that, since deaths from heart failure are included in the tabulations our governments ballyhoo as "deaths caused by smoking", Madame Calment qualified for inclusion. Crazy times indeed.

As we have seen, the 20-pack-year milestone is commonly cited. It is not, however, accounted for in typical purblind analyses. We shall account for this crucial consideration by considering for our overview that amongst ever smokers (50% of the vintage population: about 40% for women and 60% for men) about half of these (25% of the total vintage population) smoked more than, and half (another 25% of the total vintage population) smoked less than at a 20-pack-year level in a lifetime.

Those who smoked less, though they certainly would not as a group have a "zero risk", would not have an elevated risk. We shall call our class of heavier long duration smokers "major smokers" and the other smokers "minor smokers".

This creates a clearer picture. We keep to round numbers because we are trying to present a broad overview. The value in such an overview is that it corrects for baffled over-interpreted and overly-precise cherry-picking of wild numbers relating to isolated aspects of lung cancer epidemiology. In other words, a broad view is an antidote to the purblind nonsense babbled by the likes of Gupta, Clanton, and all of their like-minded double-thinking colleagues.

Now, as we've noted, amongst groups of longer term and higher intensity smokers, RR reports increase with increased smoking; this is one of the reasons the smoking / lung cancer link is compelling. Beyond a 20-pack-year level, RR figures will for higher or lower level smoking strata typically range from about 2 to 20 depending on both smoking level and duration. Our major smokers group encompasses these consequentially smoking strata.

For our class of smokers who have smoked beyond 20-pack-years a round figure of RR 10 is appropriate. For ever smokers as a whole, with RR 10 for major smokers, and RR 1 for minor smokers, this suggests – (10 + 1)/2 – an RR of 5.5. This well reflects what one can best deduce for ever smokers from review of studies with pertinence to Western smokers and present conditions of diagnosis.

Next we can consider what proportion of lung cancer is contributed by ever smokers and what proportion is contributed by never smokers.

In the PBS interview previously quoted Doctor Schiller said: “Well, actually, about 10 to 15 percent of all lung cancers occur in people who have never smoked.”

In the CNN interview Doctor Gupta alluded to the percentage contributed by ever smokers. But note how he alluded to it in response to the moderator’s question:

> ROBERTS: All right. Question number two: Smoking is by far the number-one cause of lung cancer, but radon gas is the leading cause among non-smokers; true or false?
>
> GUPTA: That is true. And this is actually surprising to a lot of people. Smoking is far and away the number-one cause. You know, eight – eight or nine times out of 10, it's going to be smoking ...

It is true that between 8 and 9 out of 10 lung cancer cases will be contributed by ever smokers, but recall what Doctor Gupta had already told us, and would again in the same interview. He told us that smokers who quit before middle age had a zero risk. Here he is perfectly defying himself by telling us that *any* lung cancer in *anyone* who ever smoked is “caused by smoking.”

The “all caused by smoking” formulation is the daffy flip side of the “zero risk” mantra. Both mutually exclusive bafflements are chanted widely and incessantly by the ideologues. They may be cavalier in pulling specific numbers out of their hats but the message of the brainless chants is always the same. Just a few examples of the “all caused” mantra here:

> Florida Adventist Hospital: "80-90% of all lung cancers are directly attributable to cigarette smoking."
>
> Cancer Research Foundation of America: “Smoking is responsible for more than 80 percent of all lung cancer deaths.”
>
> Doctor Desmond Carney, Secretary General, International Association for the Study of Lung Cancer: "Ninety-five percent of lung cancer deaths are due directly to cigarette smoking."
>
> Doctor Leah DiPlacido of the Livestrong / Lance Armstrong Foundation: “A staggering 87 percent of lung cancers would disappear if every person in the U.S. stopped smoking.”

> American Cancer Society's *Cancer Journal for Clinicians*: "Cigarette smoking causes about 82% of lung cancer deaths."
>
> American Lung Association: "Smoking is directly responsible for 90% of lung cancer deaths."
>
> Palo Alto Medical Foundation: "About 85% of all lung cancers are attributable to cigarette smoking."

The best numerical reconciliation, substantiated by the close research of Doctor Heather Wakelee, is that about 15% of lung cancers are contributed by never smokers and 85% by ever smokers, whether reference is to incidence, or to mortality.

Now, a bit of logic that the distinguished experts cannot digest, never have, and possibly never will digest. You can, though, and easily. Ever smokers constitute half of the vintage population. Half of the population, all things being equal, should be expected to contribute half of the lung cancer. Instead the ever smokers contribute about 85% of the lung cancer. Ergo: smoking is an *influence* which increases lung cancer risk. Hypothetically, absent smoking, there would be *less* lung cancer amongst the half of the vintage population that has smoked, not "*zero*" lung cancer.

In other words, if smoking did not exist, the half of the population constituted by ever smokers would be expected to contribute about the same level of lung cancer as do the never smokers currently. Because there is a *base risk*. If there weren't a base risk the never smokers would have no lung cancer today. But they *do* have lung cancer today. So there *is* a base risk. A child could understand this. The "experts" do not. They are blinded by daft ideology.

Ideologues honestly believe that, absent smoking, about 85% of lung cancer would disappear. Because they attribute all lung cancer in all who ever smoked singly and strictly to the smoking. They daffily assume in this that anybody who smoked *could not*, as a cosmic certainty, have gotten lung cancer had he not smoked. At the same time they say many smokers, who quit, have a zero risk, except if they do get lung cancer, which cancer is then 100% "caused" by smoking.

Ignore plain nonsense. Taking figures at face value, and knowing that nobody has a zero risk, since never smokers now account for 15% of lung cancer, if smoking did not exist, very simply, we would still have 30% as much lung cancer as now exists: the ever smokers,

absent smoking, would get about as much lung cancer as the never smokers do now. Thus, again taking figures at face value, 70% of US lung cancer, not 85%, could logically be placed at the feet of smoking.

This is a-b-c elementary logic. It escapes the "experts" *absolutely and at all times.* They are totally lacking in intellectual scope. They only see one factor at a time. They only see "alls" and "zeroes" in these factors and are capable of seeing the same ones interchangeably as either "all" or "zero". They are crazy.

A fundament of this craziness is the construction that smoking is the "cause" of lung cancer. It is not. Even critics of wrong-headed lung cancer research tend to apply the word "cause" casually to the statistical link between smoking and lung cancer. They should not. Indeed, it is a *cardinal rule* of statistics generally, that *statistical association is not causation*.

Now, one can as easily say that smoking causes lung cancer as to say that the sun rises and sets, and even a professional astronomer might well make reference to sunrise and sunset in a scholarly essay, but he and his expert readers, and for that matter any reader of any intelligence, would appreciate that he was speaking of the sun's rising and setting in a casual and colloquial manner, certainly not in a literal or absolute sense.

The astronomer knows that the sun does not literally rise and fall in the sky. He knows it is our planet which moves relative to the sun. He bases his scientific analyses on the reality of the situation. He does not base them on perfectly wrong ideas inherent in casual phrasing. He bases his reasoning on fact rather than on fallacy. If he did not most of his conclusions would be positively daft: ridiculous, and comic, to any person possessed of ordinary intelligence and common sense.

Scandalously, medical "professionals" do, constantly, take smoking to be the "cause" of lung cancer, in the very most literal and absolute sense. This is at the root of their idea that about 85% of lung cancer would disappear if smoking did: remove the "cause" and you remove the effect.

It's why they drivel that persons who never smoke have a zero risk: no cause= no effect. It's why they say persons who quit before 20 pack-years have a zero risk: cause quickly removed = effect altogether banished. It's why they say at the very same time that all lung cancer in all persons who ever smoked (even in their zero-risk quitters) is entirely "caused" by smoking: when they see the classification of "ever-smokers" their zero-risk quitters become smokers again in their dull-witted eyes: cause returned = effect banishment

nullified.

The self-deluded doublethinkers believe in the most literal and profound way that smoking is the cause of lung cancer. Only when *forced* under circumstances to recognize that there is lung cancer occurrence absent smoking, will they admit to the reality. They admit to the reality under duress but believe in the fallacy at all times. *They base their analyses on the fallacy virtually at all times*. Their conclusions become crazier and crazier over time. This has been going on for a very long time.

So we see: correction of the "cause" issue is by no means a mere semantic matter. Nor does saying that smoking very certainly is not properly referred to as "the cause" or "a cause" of lung cancer by any means exonerate smoking. It allows for proper and crucial consideration of smoking (when it is practiced considerably over a long time) as an *influence* on lung cancer occurrence.

As sure as the sun does not spin around the Earth, smoking is not "the cause" or "a cause" of lung cancer. Folks may say that at the dinner table. Analysts must think rationally and apply factors of their analysis *based on reality*. *Sanity* really does assist in coherent analysis, as we have seen, and will see more.

What *is* the cause of cancer? There isn't one, not in any similar sense to the way one can consider a particular pathogen as the cause of a particular communicable disease. Cancer is cell replication gone wrong. Our cells, within our bodies, are replicating all the time. As with nearly all physical processes, cell replication is sometimes imperfect, sometimes grossly so.

Our immune systems, just as they can fight pathogens that enter our systems, also fight against malformed or mutant cells produced by our own bodies. Our immune systems usually win, against most pathogens we typically encounter, and against most mutant cell replication. Sometimes, in either case, our immune systems lose.

Mostly, with regard to cell replication, the immune system simply monitors and checks irregularity, effectively nipping it in the bud. In some cases, there is a real battle, where small tumors begin to grow, but get beaten back before becoming very problematic, or symptomatic: you may have had a small bout of proto-cancer that "went away" (the immune system rallied and caught up) before you ever felt it: something similar to having a bit of sniffles that never turns into a full-blown cold.

When the mutant cell war goes particularly badly for your immune system you'll eventually feel it. If you are lucky your tumor may be "benign" (not prone to metastasizing or spreading: typically benign lung cancers, called carcinoid tumors, represent a single digit percentage of lung cancer cases, and are not evidently related to smoking. These slow-growing carcinoid lung tumors can become "malignant", or prone to spreading, but usually do not spread.)

Even if not benign, your cancer may be localized to one area, also lucky for you: easiest treatment, relatively good prognosis. Or your cancer may have metastasized. Individuals, even with the same primary cancer diagnosis, may have cancers that are either more or less aggressive in terms of spreading.

When enough signs appear, and your doctor comes to find cancer, you will be diagnosed with one or another primary cancer (correctly, you'll hope.) That's the point from which you've got a serious battle ahead. But it's part of an overall war that's been going on inside you all your life.

So cancer is reasonably best understood as an inherent vulnerability. We're all born with a potential for cancer, because we are an organized collection of cells which replicate, and replication can be faulty. Virtually all organized animals are subject to cancer. Evidence of cancer has been found in dinosaur bones. Some plants are vulnerable to metastasizing disease analogous to cancer. If there is a cause of cancer it is Nature.

Irritation of tissues influences the risk of mutant cell replication in the affected area. As we've discussed, some things we may commonly do (e.g. over-enthusiastic sun-tanning, or risking venereal disease, or smoking or drinking considerably) may have consequences inviting particular cancers. Lifestyle factors may *influence* cancer risk, but cancer risk in itself, is an inheritance we receive at birth. It's part of being a mortal creature. It is your base risk.

Now we have taken good perspective on cancer, and on lung cancer in particular, and we have carefully reviewed each crucial factor necessary to forming an enlightened overview of lung cancer prevalence and provenance amongst the US population. This US perspective we will consider as roughly representative for the West generally, but not for the East, of which we shall speak distinctly and more generally, following tables and discussion of Western dimensions. Let's review the individual points discussed.

The absolute risk for never smokers (Base risk x Relative risk = Absolute risk):

2% x 1 = 2%

The absolute risk for minor smokers (moderate lifetime smoking):

2% x 1 = 2%

The absolute risk for major smokers (considerable lifetime smoking):

2% x 10 = 20%

The absolute risk for ever smokers (minor and major smokers average):

2% x 5.5 = 11%

The typical lung cancer patient today is in the vicinity of 70 years old. About half of the vintage population ever smoked. The ever smoking population is divided about evenly between major and minor smokers. Half of the vintage population never smoked.

Ever smokers contribute about 85% of lung cancer and never smokers contribute the remaining 15%.

Let us now put the pieces together on a table illustrating how all pertinent elements dovetail to produce a complete and congruent overview when factors and influences are properly considered. Following this table (on next page) we will discuss concordance with recent NCHS death statistics.

Overview of Lung Cancer Mortality in the United States, Both Sexes, by Smoking Status					
A. Smoking status by % of mortality vintage population	**B.** Base risk	**C.** Relative risk	% contribution to all-cause mortality: (A x B x C)	% contribution to all-cause mortality (by ever / never smoker status).	% of lung cancer mortality (by ever / never smoker status).
25% Major smokers	2%	10	5%	5.5%	5.5 / 6.5 = 85%
25% Minor smokers	2%	1	0.5%		
50% Never smokers	2%	1	1%	1%	1 / 6.5 = 15%
Total lung cancer mortality as proportion of total all-cause mortality ➜				6.5%	

Pertinent reference table from NCHS 2008 is presented on following page.

Table 12. Number of deaths from 113 selected causes, Enterocolitis due to *Clostridium difficile*, drug-induced causes, alcohol-induced causes, and injury by firearms, by race and sex: United States, 2008

[Data for specified races other than white and black should be interpreted with caution because of inconsistencies between reporting race on death certificates and on censuses and surveys; see "Technical Notes." The asterisks (*) preceding the cause-of-death codes indicate that they are not part of the *International Classification of Diseases, Tenth Revision* (ICD-10), Second Edition; see "Technical Notes"]

Cause of death (based on ICD-10, 2004)	All races			White[1]			Black[1]		
	Both sexes	Male	Female	Both sexes	Male	Female	Both sexes	Male	Female
All causes	2,471,984	1,226,197	1,245,787	2,120,233	1,046,183	1,074,050	289,072	147,143	141,929
Salmonella infections (A01-A02)	44	24	20	32	20	12	9	2	7
Shigellosis and amebiasis (A03,A06)	6	4	2	5	3	2	-	-	-
Certain other intestinal infections (A04,A07-A09)	7,876	2,996	4,880	7,268	2,749	4,519	467	178	289
Tuberculosis (A16-A19)	585	375	210	332	210	122	136	87	49
Respiratory tuberculosis (A16)	449	294	155	246	154	92	106	73	33
Other tuberculosis (A17-A19)	136	81	55	86	56	30	30	14	16
Whooping cough (A37)	20	9	11	18	7	11	-	-	-
Scarlet fever and erysipelas (A38,A46)	3	2	1	2	1	1	-	-	-
Meningococcal infection (A39)	102	56	46	75	42	33	25	13	12
Septicemia (A40-A41)	35,927	16,328	19,599	28,697	13,066	15,631	6,426	2,877	3,549
Syphilis (A50-A53)	34	23	11	13	11	2	19	10	9
Acute poliomyelitis (A80)	-	-	-	-	-	-	-	-	-
Arthropod-borne viral encephalitis (A83-A84,A85.2)	2	1	1	2	1	1	-	-	-
Measles (B05)	-	-	-	-	-	-	-	-	-
Viral hepatitis (B15-B19)	7,629	5,019	2,610	6,111	4,064	2,047	1,115	728	387
Human immunodeficiency virus (HIV) disease (B20-B24)	10,285	7,406	2,879	4,339	3,489	850	5,780	3,790	1,990
Malaria (B50-B54)	5	4	1	2	2	-	3	2	1
Other and unspecified infectious and parasitic diseases and their sequelae (A00,A05,A20-A36,A42-A44,A48-A49,A54-A79,A81-A82,A85.0-A85.1,A85.8,A86-B04,B06-B09,B25-B49,B55-B99)	5,914	2,979	2,935	4,952	2,458	2,494	738	408	330
Malignant neoplasms (C00-C97)	565,469	295,259	270,210	485,893	254,124	231,769	63,954	33,019	30,935
Malignant neoplasms of lip, oral cavity and pharynx (C00-C14)	8,019	5,488	2,531	6,712	4,551	2,161	1,002	730	272
Malignant neoplasm of esophagus (C15)	13,714	10,847	2,867	12,019	9,629	2,390	1,432	1,016	416
Malignant neoplasm of stomach (C16)	11,352	6,735	4,617	8,469	5,064	3,405	2,057	1,196	861
Malignant neoplasms of colon, rectum and anus (C18-C21)	53,321	27,094	26,227	44,751	22,741	22,010	6,908	3,508	3,400
Malignant neoplasms of liver and intrahepatic bile ducts (C22)	18,213	12,302	5,911	14,377	9,631	4,746	2,466	1,748	718
Malignant neoplasm of pancreas (C25)	35,236	17,515	17,721	30,124	15,125	14,999	4,109	1,894	2,215
Malignant neoplasm of larynx (C32)	3,760	2,949	811	3,059	2,399	660	645	503	142
Malignant neoplasms of trachea, bronchus and lung (C33-C34)	158,656	88,586	70,070	138,715	76,761	61,954	16,250	9,638	6,612
Malignant melanoma of skin (C43)	8,623	5,672	2,951	8,450	5,586	2,864	120	57	63
Malignant neoplasm of breast (C50)	41,026	437	40,589	34,055	350	33,705	5,928	77	5,851
Malignant neoplasm of cervix uteri (C53)	4,008	...	4,008	3,018	...	3,018	799	...	799
Malignant neoplasms of corpus uteri and uterus, part unspecified (C54-C55)	7,675	...	7,675	6,176	...	6,176	1,286	...	1,286
Malignant neoplasm of ovary (C56)	14,362	...	14,362	12,725	...	12,725	1,200	...	1,200
Malignant neoplasm of prostate (C61)	28,472	28,472	...	23,362	23,362	...	4,588	4,588	...
Malignant neoplasms of kidney and renal pelvis (C64-C65)	12,895	8,206	4,689	11,352	7,241	4,111	1,203	743	460
Malignant neoplasm of bladder (C67)	14,036	9,791	4,245	12,853	9,107	3,746	950	516	434
Malignant neoplasms of meninges, brain and other parts of central nervous system (C70-C72)	13,724	7,686	6,038	12,568	7,049	5,519	840	455	385
Malignant neoplasms of lymphoid, hematopoietic and related tissue (C81-C96)	54,954	30,449	24,505	48,348	26,927	21,421	5,269	2,804	2,465
Hodgkin's disease (C81)	1,171	639	532	1,026	559	467	119	64	55
Non-Hodgkin's lymphoma (C82-C85)	20,369	11,004	9,365	18,476	9,984	8,492	1,360	736	624
Leukemia (C91-C95)	22,335	12,711	9,624	19,894	11,374	8,520	1,903	1,057	846
Multiple myeloma and immunoproliferative neoplasms (C88,C90)	11,020	6,057	4,963	8,900	4,978	3,922	1,882	943	939
Other and unspecified malignant neoplasms of lymphoid, hematopoietic and related tissue (C96)	59	38	21	52	32	20	5	4	1
All other and unspecified malignant neoplasms (C17,C23-C24,C26-C31,C37-C41,C44-C49,C51-C52,C57-C60,C62-C63,C66,C68-C69,C73-C80,C97)	63,423	33,030	30,393	54,760	28,601	26,159	6,902	3,546	3,356

In 2008 there were 2,471,984 all-cause deaths of which 50% occurred amongst males (1,226,197 / 2,471,984) and 50% occurred amongst females (1,245,787 / 2,471,984). The US population at large is also presently close to 50-50 male/female (estimated 49% male and 51% female, total 313,232,044 in 2012, per *CIA World Factbook* online, January 2012) as has long been the case.

Death statistics have been very similar, with regard to total all cause deaths, and total lung cancer deaths, in recent years. The specific numbers vary just marginally and the lung cancer / all cause proportion varies only decimally from year to year.

Our overview suggests that lung cancer should be expected to produce about 6.5% of all-cause mortality. In 2008 there were 158,656 lung cancer deaths. This is 6.4% of the 2,471,984 all-cause deaths.

To illustrate the general applicability of the 6.5% suggestion for recent times let us note: for 2007 the calculation is 158,760 / 2,423,712 = 6.6%, for 2006: 158,664 / 2,426,264 = 6.5%, for 2005: 159,292 / 2,448, 017 = 6.5%. Similar should likewise be expected for several years to come. The overall lung cancer picture changes only gradually over time.

And now we have that picture as it is today, taking the state of diagnosis and resultantly biased statistics as they are today, but we will have more to say about that shortly. Drawing the picture requires nothing more than relating the very obvious crucial factors and influences properly as they should be considered, and the mathematical ability of a grade school graduate.

Consider this the "landscape" of lung cancer. Consider that it's drawn from the vantage of a lake's bank affording clear view of the hills and greenery on the other side of that lake. Why do our confounded "experts" fail to see the lung cancer landscape? They have never taken the perspective view. They've never once visited the bank of the lake that offers it. They're lost in the hills and greenery on the other side. They wander aimlessly through the woods, bumping repeatedly into variegated trees, which they take to be all of one species, or else of just one other: the "all" trees and the "zero" trees. They can't help mixing up even these two confabulated varieties.

It does seem about as simple as that. The "experts", to the extent they may have brains, have shut them down with biased ideology. They have made *themselves* simple through doublethink and disrespect for logic. The old adage applies. They can't see the forest for the trees.

For anyone who cares to think clearly, however, the perspective view is necessary, and it *must* be essentially and approximately as shown on the overview table, for the simple reason that *in no other way* can the pertinent factors (lung cancer proportion of all mortality, smoking prevalence amongst the vintage population, contribution of ever and never smokers to lung cancer mortality in conformance with base and relative risks) be shown to balance one and all with the others. It adds up and makes sense. *Nothing else does or can*.

Nevertheless conventionally boggled contemporary health professionals – although all data we've covered is widely available, and the logic and math are simply elementary – do not and would not take this balanced perspective. Drag them to where the view is offered and still they will fail to see the landscape. They will tell you there are some "all" and some "zero" trees out there. They will see them, from any point of vantage, as unconnected pieces, and proceed to misunderstand them perfectly, one by one, and all in all. They will do so because they always have. They've been trained to see that way. They are as sure that things are that way as were their medical forbears in believing that mental illness should be treated by drilling holes in patients' skulls and that virtually all patients required leech bleeding.

Thus they analyze and pompously expound based on their *consensus of belief* rather than on reality. Reason as you might, the believers would insist – for instance, and as they have committed themselves in public statements – on such ideas as that base risk for lung cancer amongst never smokers, when not strictly zero, might be one per cent or less, or that the relative risk for ever smokers as a whole is about 20 (which might apply to a very heavy-smoking sub-group of ever smokers), or that twenty-somethings who smoke only occasionally have a high risk of lung cancer within a decade; for this last they distort lifelong risk amongst "ever smokers" as applying in the short term and even to minimal smoking, incidentally and crowningly ignoring that lung cancer before late middle age is abundantly unlikely for non-smokers and smokers alike.

How can they think this way? How can they with such consistency disdain reason in preference for *astounding* vacuity? An essential dissertation on this conundrum has been provided by the Australian philosopher and psychologist, Doctor Vincent-Riccardo Di Pierri, who likens today's medical ideologues – entranced by lifestyle epidemiology – to their equivalents of a previous generation who – similarly in thrall to eugenics – advocated logically baseless and sociologically calamitous racial bias. Excerpts follow from Doctor Di Pierri's *Rampant Antismoking Signifies Grave Danger: Materialism Out of Control*.

Lifestyle epidemiology cannot be considered as a pseudo or even poor/bad

science. Its belief structure (materialist/externalist bias) and gross incompetence on a systemic basis make the resulting conduct *antithetical* to scientific enquiry, i.e., *antiscientific*. It demonstrates a poor grasp of the assumptions and considerable limitations of statistical inference, it violates *every* principle of causal argument, and is daft with regard to psychological, social and moral health. In the hands of epidemiology, the term "cause," which is the strongest in scientific parlance, has been reduced to the fostering of superstitious belief (mental dysfunction) and is flung about the medical literature and the media with reckless abandon. The medico-materialist bias and the misguided attempt to coerce societal change on the basis of what is a "statistics madness" can well be characterized as a contemporary form of witchdoctoring. One needs to be reminded regularly that this conduct is being produced by a supposed scientific discipline and, even more absurdly, a supposed health authority. Furthermore, all detrimental repercussions of this misconduct are iatrogenic [i.e. harmful in result of medical error].

Another critical problem is that the capacity for self-correction is non-existent in the "discipline" of epidemiology. In well-functioning scientific disciplines, there is a coherent grasp by at least a majority of the practitioners as to the central principles that define scientific enquiry. If there is errant research conduct by any members, e.g., violation of principles of causal argumentation, the peer group itself, through critiques, reviews, etc., will bring the problem into correction. It has already been briefly considered that epidemiology has never come to terms with the principles of scientific enquiry and particularly causal argument. In epidemiology there is no coherent, collective grasp of principles such as consistency/specificity, strength of an association, etc.. Since the problem is systemic (institutionalized), *most* demonstrate the errant thinking, and therefore, self-correction is impossible. Explanation in this context is reduced to consensus effects devoid of coherent argument.

... Those offering critiques of the orthodox view have assumed that the conduct of the orthodoxy reflects isolated instances of over-interpretation coupled with a cavalier attitude. However, Berridge (1999) notes a very critical defining time for a particular idea of health and its promotion. In the mid-1970's there were enough numbers within the "health" bureaucracy and the medical establishment that shared a materialist worldview that allowed one of the numerous "consensus" effects that medico-materialism, in

> particular, is notorious for. The capacity to quantify risk, as through epidemiological investigation, is a centrality in the materialist idea of health. Numerics and quantification appeal to the mentality in that this is about as much as it can comprehend. Being superficial, devoid of any spiritual, moral, psychosocial, and psychological dimensions, it jumps to the most simple-minded, ill-considered interpretations of data possible. Worse still is that, comprehending no higher standard of inference, it is utterly convinced of the "rightness" of its surmising.

By researching and reasoning we have done better than simply to surmise. Let us now do better still by applying the perspective we have developed to what have often been stated as the most prominent "mysteries" about lung cancer epidemiology in recent years.

First, while apparent for decades, it was long held as a "mystery" that the proportion of never-smokers with lung cancer is higher amongst women than is the case for men. What on Earth could the reason be? Why had cruel fate imposed such a tragic and positively indecipherable disparity against women?

Let's illustrate the perplexity of orthodox analysts, to begin with, by returning to the PBS interview, previously quoted, from where we left it off:

> JEFFREY BROWN: Dr. Schiller, another thing that I think a lot of people wondered about this week was, in the case of Dana Reeve, you mentioned earlier people who develop lung cancer who never smoked. Now, how unusual is that?
>
> DR. JOAN SCHILLER: Well, actually, about 10 to 15 percent of all lung cancers occur in people who have never smoked. And interestingly enough, in the majority of those patients, it tends to occur more commonly in women. So of all the never-smokers who have gotten lung cancers, the majority of those are women, for reasons that we don't understand yet.

The media trumpeted this purportedly inscrutable situation, particularly around the time of Dana Reeve's death, in 2006. For example, an article excerpt of 8 March 2006 from *Daily News Central* (its motto: "News You Can Trust"), entitled "Dana Reeve's Death Highlights Mystery of Lung Cancer Among Nonsmoking Women":

> About one in five women who get lung cancer have never smoked.

> Researchers know that smoking causes cancer, but they don't know why people who never smoke get it. And they have no idea why more women who have never touched a cigarette get lung cancer than men who have never lit up.
>
> Lung cancer killed Dana Reeve on Monday. She was 44 and had never smoked. People know her as the constant caregiver and support for her husband, actor Christopher Reeve, whose tragic fall from a horse in 1995 paralyzed him. He died in 2004.
>
> Now, her own early death is bringing attention to another tragedy: lung cancer among people who have never smoked, especially its disproportionate impact on women.

The closest research on the question of never smokers' proportional contribution to lung cancer statistics has been conducted by Doctor Heather Wakelee of Stanford University, who studied both US and European populations. Doctor Wakelee found somewhat mixed results from the various populations but concluded overall, in a 2010 presentation, that:

"We see about 20% of women in the US with lung cancer have never smoked.

For men, that was about 10%, and that fits with what people have been guesstimating for a while, around 15% overall, but we were showing the sex difference, and that's been shown by some other studies also."

Now, as we have previously discussed, the health community has gone back and forth over the years, suspecting either that men are more vulnerable, or that women are, to the effects of smoking. The evident truth is that the two sexes are about equally vulnerable.

Here, we are talking about never smokers exclusively, and the experts foundered about interminably trying to understand why there are more female than male never-smokers with lung cancer. With their characteristic focus on relative risk they suspected that never smoking women must have a higher lung cancer death rate in result of sensitivity to some one or other external factor such as diet or radon in the atmosphere, but ultimately were faced with the fact that the death rate per standard unit of population amongst never smokers of both sexes is about the same, in fact somewhat higher for males rather than for females.

The closest research on the question of related death rates appeared in the *Journal of the National Cancer Institute* in May, 2006. It suggested that rates were similar for both male and female never smokers. Rates were presented as expected deaths per "100,000 person-years": a statistical basis of comparison. In fact, males were shown to be at somewhat higher risk: about 17 to 19 male versus about 12 to 15 female deaths per 100,000 person-years in the whole population group studied (abstract below refers only to the more recently studied portion of the total study group – "CPS II" – showing figures of 17.1 per 100,000 person-years for men and 14.7 for women.) In plain terms, the overall findings suggest never smoking females are inherently about one quarter less, rather than any more, prone to lung cancer as compared to never smoking males, but this is of negligible consequence to our overall perspective, which we have based on whole number proportional estimates. Of course the marginal disparity is in the exact opposite direction to what our confused experts expected to see and had been trying to see. Abstract of the *JNCI* article follows.

Lung Cancer Death Rates in Lifelong Nonsmokers

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Abstract

Background: Few studies have directly measured the age-, sex-, and race-specific risks of lung cancer incidence and mortality among never tobacco smokers. Such data are needed to quantify the risks associated with smoking and to understand racial and sex disparities and temporal trends

that are due to factors other than active smoking. Methods: We measured age-, sex-, and race-specific rates (per 100 000 person-years at risk) of death from lung cancer among more than 940 000 adults who reported no history of smoking at enrollment in either of two large American Cancer Society Cancer Prevention Study cohorts during 1959–1972 (CPS-I) and 1982–2000 (CPS-II). We compared lung cancer death rates between men and women and between African Americans and whites and analyzed temporal trends in lung cancer death rates among never smokers across the two studies by using directly age-standardized rates as well as Poisson and Cox proportional hazards regression analyses. All statistical tests were two-sided.

Results: The age-standardized lung cancer death rates among never-smoking men and women in CPS-II were 17.1 and 14.7 per 100 000 person-years, respectively. Men who had never smoked had higher age-standardized lung cancer death rates than women in both studies (CPS-I: hazard ratio [HR] = 1.52, 95% confidence interval [CI] = 1.28 to 1.79; CPS-II: HR = 1.21, 95% CI = 1.09 to 1.36). The rate was higher among African American women than white women in CPS-II (HR = 1.43, CI = 1.11 to 1.85). A small temporal increase (CPS-II versus CPS-I) in lung cancer mortality was seen for white women (HR = 1.25, CI = 1.12 to 1.41) and African American women (HR = 1.22, CI = 0.64 to 2.33), but not for white men (HR = 0.89, CI = 0.74 to 1.08). Among white and African American women combined, the temporal increase was statistically significant only among those aged 70–84 years (P<.001).

Conclusions: Contrary to clinical perception, the lung cancer death rate is not higher in female than in male never smokers and shows little evidence of having increased over time in the absence of smoking. Factors that affect the interpretation of lung cancer trends are discussed. Our novel finding that lung cancer mortality is higher among African American than white women never smokers should be confirmed in other studies.

Michael Thun's research finally brought the obvious to mind – or at least to his mind – and he then announced it to his colleagues. The answer is of course as plain as it could possibly be. It *always had been* plain and obvious. It simply wasn't seen by any "experts" until Michael Thun was *forced* by his data to see it.

While men and women are about evenly split proportionately in both the mortality

population and the overall US population, amongst the mortality vintage population, *women were less likely to smoke than were men*.

With the sexes combined ever smokers comprise about half of the mortality population, but for men it would be about 60%-40% ever to never smokers and for women that would be reversed: about 40%-60% ever to never smokers.

It is therefore elementary that, if lung cancer base risk, as is the case with relative risk, is approximately the same for both sexes, as appears to be the case based on review of all crucially related evidence, then one would *of course* expect a higher proportion of never smoker lung cancer cases amongst women, than is the case amongst men, *because the vintage population of never smoking women is a numerically larger population than is the vintage population of never smoking men.*

It is perhaps insufficient to say this is logical. From a rational viewpoint, a greater proportion of never smoker lung cancer amongst women as compared with men is positively and transparently *inevitable*.

Why, then, for so long, was this inevitability *never* mentioned in the ideologues' discussions on this topic? Why did they need to see close research on death rates, and receive instruction from those who researched the death rates, in order to see what had always been glaring before their faces? In especially obtuse or plain ignorant "expert"circles the babbling *still* goes on; some "esteemed authorities" have continued rehashing the "mystery" even after the death rate data, and the elementary conclusion about smoking rates, were published and widely discussed in the press.

The ideologues were so long *blind* to the obvious because their *belief* is that smoking is the *cause* of lung cancer. This supremely unyielding belief *precludes consideration* of smoking for what it is in reality: an *influence* on lung cancer: a factor which can only be properly appreciated through understanding of its interplay with other equally crucial factors.

But such scope of thought is beyond the capacity of the typical groupthinking ideologue. Lung cancer, in the minds of our "experts", is *supposed* to happen to smokers, while if it happens to never smokers at any level or proportion, this must *always* be altogether baffling and mysterious.

In particular: *the childishly blockheaded ideologues simply cannot understand – they virtually block their minds to the very conception of – the fundamental concept of a base risk.* If anything *can*

be considered the cause of cancer it is just that and only that: the inherently imperfect nature of cell replication represented by base risk. But they madly and firmly shut their eyes to this. They didn't recognize it in this case until forced to do so. They do not recognize it generally in any other circumstances. Probably they never will.

That supposed scientists can be so blind would be mysterious, indeed, if the supposed scientists had not been making this clear for decades. That around or about 10% of male lung cancer victims are never smokers versus about 20% for females *is by no means mysterious*. Let's simply look at the *altogether unsurprising* result of stratifying by sex on our overview table.

For males:

Overview of Lung Cancer Mortality in the United States, Male Sex, by Smoking Status					
A. Smoking status by % of mortality vintage population	**B.** Base risk	**C.** Relative risk	% contribution to all-cause mortality: (A x B x C)	% contribution to all-cause mortality (by ever / never smoker status).	% of lung cancer mortality (by ever / never smoker status).
30% Major smokers	2%	10	6%	6.6%	6.6 / 7.4 = 89%
30% Minor smokers	2%	1	0.6%		
40% Never smokers	2%	1	0.8%	0.8%	0.8 / 7.4 = 11%
Total male lung cancer mortality as proportion of total male all-cause mortality ➜				7.4%	

Let us check our table's suggestion of about a 7.4% lung cancer mortality contribution to all-cause mortality in males with reference to NCHS 2008 death statistics. Lung cancer deaths amongst males were 88,586 while all-cause male deaths were 1,226,197. Thus 88,586 / 1,226,197 = 7.2%. Calculations for: 2007: 88,372 / 1,203,968 = 7.3%, 2006: 88,279 / 1,201,942 = 7.4%, 2005: 90,187 / 1,207,675 = 7.5%.

Naturally and inevitably, in congruence with all crucial factors, never smoker contribution to lung cancer amongst men is in the area of 11%. Eleven per cent of about 90,000 annual male lung cancer deaths equates to about 10,000 male never smoker lung cancer victims per year in the present era.

For females:

Overview of Lung Cancer Mortality in the United States, Female Sex, by Smoking Status					
A. Smoking status by % of mortality vintage population	**B.** Base risk	**C.** Relative risk	% contribution to all-cause mortality: (A x B x C)	% contribution to all-cause mortality (by ever / never smoker status).	% of lung cancer mortality (by ever / never smoker status).
20% Major smokers	2%	10	4%	4.4%	4.4 / 5.6 = 79%
20% Minor smokers	2%	1	0.4%		
60% Never smokers	2%	1	1.2%	1.2%	1.2 / 5.6 = 21%
Total female lung cancer mortality as proportion of total female all-cause mortality ➜				5.6%	

Let us check our table's suggestion of about a 5.6% lung cancer mortality contribution to all-cause mortality in females with reference to NCHS 2008 death statistics. Lung cancer deaths amongst females were 70,070 while all-cause female deaths were 1,245,787. Thus 70,070 / 1,245,787 = 5.6%. Calculations for: 2007: 70,388 / 1,219,744 = 5.7%, 2006: 69,385 / 1,224,322 = 5.7%, 2005: 69,105 / 1,240,342 = 5.6%.

Naturally and inevitably, in congruence with all crucial factors, never smoker contribution to lung cancer amongst women is in the area of 21%. Twenty-one per cent of about 70,000 annual female lung cancer deaths equates to about 15,000 female never smoker lung cancer victims per year in the present era. The figure for men is about 10,000. There is about half again as much lung cancer amongst female never smokers as amongst male never smokers.

The statisticians usually parse according to proportions so let's look at it that way. The split, in proportional terms, is around or about 60% female never smokers with lung cancer (15,000 / 25,000) to 40% males (10,000 / 25,000). The female never smoker population, about 60% of vintage females, is half again the size of the male never smoker population, about 40% of vintage males. Hence, again and of course, female never smokers produce about half again as much lung cancer as do male never smokers. Are you mystified by this?

Why were they? Because they have never developed balanced perspective. They are, simply, unbalanced. Taking the rational vantage, whatever differences in terms of lung cancer susceptibility there might be between males and females, they are not of statistical

consequence in the task of forming basic perspective. The basic difference between women and men in this respect is exactly what one *must* expect given that female never smokers in the vintage population numerically outnumber male never smokers in that population; no further explanation for the general situation is required. The much and long debated "mystery" on this subject *never* existed except in baffled minds.

The other most-discussed "lung cancer mystery" of recent years – this one still baffling "experts" worldwide – is the situation regarding Asian women. In many areas of the Orient smoking rates are very low amongst women. The ideological outlook of most health professionals leads them to believe that lung cancer should be nearly non-existent amongst these female populations. But that is not the case. Although lung cancer rates are lower amongst such nearly smokerless female populations as compared with heavier smoking Western female populations they are not dramatically lower. This also has been known for decades. Excerpts follow from a report on worldwide statistics – which reflect the lower smoking rates in many countries compared to US figures – from *Nature Reviews Cancer*, October 2007:

> Global statistics indicate that 1.18 million lung cancer deaths occurred in 2002. An estimated 15% of lung cancer in men and 53% of lung cancer in women (25% of all cases) are not attributable to tobacco use, making lung cancer in never smokers the seventh leading cause of cancer death for both sexes worldwide. Lung cancers in never smokers show geographic and gender variations. ... The proportion of female lung cancer cases in never smokers is particularly high in East and South Asia.

Of course, the proportion of female never smokers, amongst the mortality vintage population, is particularly high in East and South Asia. Taiwan provides a good example of a female population with a very low proportion of smokers. This has been noted in medical journals over many years, for example in a study by Chun-Yuh Yang et alia in the *Archives of Environmental Health*, May 1999. Excerpt:

> ... Approximately 80% of female lung cancer cases in the United States in 1991 were attributable to cigarette smoking [a typical "all-caused" over-interpretation of course]. However, smoking habits cannot fully explain the epidemiological characteristics of female lung cancer in Taiwan, where female lung cancer rates are relatively high and female smoking prevalence is relatively low. In Taiwan, the percentage of female lung cancer patients who smoked was only about 10%. This figure is far lower than what has been

> reported from Europe or the United States (i.e., 70-90%). Furthermore, the prevalence rate of cigarette smoking was 3-4% in females aged 16 y[ears] or more in a nationwide annual investigation. The prevalence has remained constant over the past 30 y[ears], but there has been a small decrease in recent years – possibly related to health education. Therefore, given the relatively low prevalence of cigarette smoking for women in Taiwan and the relatively high death rates, we may conclude that cigarette smoking accounts for only a small fraction of female lung cancer in Taiwan today.

The article's mention of an approximate 3% - 4% female smoking rate is about right, as per comparison with best sources, which more generally suggest that a very small, single-digit rate, of about 2%-5%, has applied from back beyond the Second World War era up to the present in Taiwan.

As regards Taiwanese males, over the past seventy years or so, overall smoking rates up to about 75% are reported. Today, smoking for the Taiwanese population as a whole is about 30%, comparing to about 20% today in the USA, but in Taiwan around or about 98% of all of today's smokers are men. Taiwanese men have been rather more likely to smoke, and rather less likely to quit, than is the case for American men over the same general period.

In reports generally the proportion of smokers amongst Taiwanese females with lung cancer has been shown for years and up to the present as about 10% or less. That around or about 4% of the female vintage population should be considered ever smokers, and that they produce around or about 10% of total lung cancer amongst Taiwanese women, points to what we have previously mentioned in this essay: relative risk for ever smokers amongst many Asian populations would be closer to about 3 rather than the RR closer to 6 indicated for the West in recent decades, which reflects greater diagnostic bias in the West.

As we have discussed, the paucity of smokers amongst women in countries such as Taiwan limits the opportunity for diagnostic bias, and also limits incentive toward such bias, since Oriental clinicians have long been faced with the circumstance that lung cancer certainly exists in their female populations, despite the dearth of smoking. This enforced level of objectivity, by general indication, seems to carry over somewhat to the male population, although there remains great opportunity for bias in this case, since Taiwanese males, alike with males of many Asian nations, have been prone to smoking as much or more than are American men.

Let us take a closer look at the smoking / lung cancer situation in the Orient using Taiwan as our example. Statistical data relating to tobacco and lung cancer is not so prevalent for most countries as for the US. However, Taiwan's Department of Health mortality statistics are on a par with the NCHS statistics of the US, and we may refer to these, from the most recent available compilation at time of composition, the final 2008 figures. Just as with the US mortality statistics, Taiwan's 2008 figures are very similar as for several years back, they well represent the present era, and the general shape of Taiwan's mortality is not likely to change appreciably for years to come.

Taiwan has prospered, household income has grown, and life expectancy has considerably lengthened, since the vast influx of mainland Chinese came to the island, with Chiang Kai-Shek, around the time of the Communist take-over of the mainland in 1949. The considerable extension of life expectancy has created increasing lung cancer mortality over the intervening period since lung cancer is a disease of old age. Per government records, in the mid-nineteen fifties, life expectancy at birth was 60 years for males and 64 years for females. As of 2010 the figures were 76 for males and 83 for females. The highest risk age group for lung cancer is 75-79 per 2008 government mortality tables. Lung cancer became the biggest cancer killer of Taiwanese women in 1986 and remains so to the present era.

Although the *CIA World Factbook* online shows Taiwan's 23,071,79 population, as of 2011, to be 50%-50% male/female, the mortality vintage population is heavily weighted toward males: officials of Chiang Kai-Shek's government who fled with him more than sixty years ago, and of course his military forces which also followed him, were predominantly male by a vast margin. The disparity created still exists in the elderly portion of the current population.

All-cause mortality in Taiwan for 2008 was 142,283: 62% males at 87,682, and 38% females at 54,601. The average age at death from all causes was 69 and the average age at death from lung cancer was the same; in the US the 2008 average age for all-cause mortality was 73, and for lung cancer, 71. In Taiwan there were 7,777 total lung cancer deaths in 2008. That is 5.5% of all deaths comparing to the lung cancer proportion of 6.4% in the US for the same year.

Lung cancer deaths for males were 5,306 out of the all-cause 87,682, or 6.1%, comparing to the 7.2% for US males for the same year. This despite that Taiwanese males of the mortality vintage smoked more, rather than less, than did their American counterparts. This would reflect differences in base risk and relative risk between the two populations but data is insufficient to parse on this. Although lung cancer is the biggest cancer killer amongst

Taiwanese women it is not the biggest cancer killer amongst Taiwanese men. That is liver cancer. Taiwanese men both smoke and drink heavily.

The case of the Taiwanese females requires little parsing to be understood. Lung cancer deaths for females were 2,471 out of the all-cause 54,601, or 4.5%, comparing to the 5.6% for US females for the same year. This despite that American women (40% ever smokers) were ten times as likely as Taiwanese women (4% ever smokers) to smoke. The Taiwanese women, proportionately, got less lung cancer than did American women. They surely did not get one tenth as much, as our distinguished experts would childishly expect. In fact, proportionately, the Taiwanese women got 80% (4.5 / 5.6) as much lung cancer as did the American women. More precisely: Taiwanese women, proportionately, were diagnosed with 80% as much primary lung cancer as was the case for American women.

The base risk for Taiwanese women is self-evident. One can even eliminate all ever smoker contribution (10%) toward the figure of 4.5% (90% of 4.5% = 4.05%) in keeping with the positively mad idea that smoking is the only factor in ever smokers' lung cancer; the contribution of ever smokers to total lung cancer, in this circumstance, is statistically trivial; it's numerically insignificant. As a whole number percentage the base risk of lung cancer, absent smoking, for these Asian women is 4%, or twice, what biased statistics of the West suggest at 2%.

The distinguished baffled experts have for many years now been offering theories as to what might explain the undeniable 4% risk, absent smoking, of Asian women such as those in Taiwan. With their ingrained blindness to the fundamental concept of base risk and their adoration of relative risk they have speculated, for instance, that Asian women may have a bad diet, or that they have a genetic flaw lacking in all men and in Western women, or that Oriental cooking produces more fumes than do Western cooking methods. You will see reference to some of their research in the following section of this paper. This research is confused, wildly inconsistent, and altogether unilluminating.

Of course, the addled authorities likewise know that smoking-related relative risks for Asia are lower than those reported for the West. Does it ever occur to them that they are seeing in the Asian mirror a sharp reflection of their own diagnostic bias? Is this possibility ever mentioned in their journal articles?

By no means! The dogmatists are perfectly content in thinking that never smoking Asian women have a much higher inherent *vulnerability* to lung cancer *and* – at the very same time, as shown by their small smoking-related relative risk – a greatly superior *resistance*

to that which most famously can promote lung cancer. Doublethink is a genuinely mysterious and a very sad thing.

There very clearly is bias in Western statistics. Absent this bias the base risk for the West would certainly be shown to be more than 2%. Taiwanese women, and like Asian female populations, provide a near-perfect example of a population unaffected by smoking or, largely, by the diagnostic bias surrounding smoking.

The real base risk for the West, absent diagnostic bias, could well be 4% rather than 2%. The present statistical divide between West and East is certainly reflective of greater Western diagnostic bias and there may well be nothing more to the divide than that.

We have noted that, taking Western statistics *as they are*, if smoking did not exist, instead of there being about 160,000 lung cancer cases per year in the US, there would be about 30% as much, or something around or about 48,000 cases. If the 4% base risk is the unbiased truth we would see about twice as much as that.

The late Doctor Alvan Feinstein was particularly renowned for admonishing against and ultimately reforming errant diagnostic and clinical practices relative to rheumatic fever, to the great benefit of patients. Recall his admonition of 1974, regarding lung cancer, “Cigarette smoking may contribute more to the diagnosis of lung cancer than it does in producing the disease itself." He likely was perfectly correct, but in this case, his warning has gone vastly unheeded. The anti-tobacco ideologues are deaf as well as blind.

Before concluding this section let us consider one more issue. The reader will well have noted very caustic description in this essay of professional damn foolishness on the subject of tobacco and health. This condemnatory tone is proper and necessary in discussing these issues. As noted by the philosopher and psychologist Doctor Vincent-Riccardo Di Pierri in comments previously quoted, correction of this problem from within the ranks of lifestyle epidemiologists, or the public health bureaucracy, or from the medical profession, is not to be expected. These supposed experts have for so long “dumbed themselves down” with what Di Pierri calls “statistics madness” that they have lost all sight of reason. The preference is strong among nearly all of them for the idiocy of fanaticism. A return of reason could now only be forced upon them from outside. It is high time we all got angry about the miserable situation, and moved, to fix it.

Lifestyle epidemiology has always been sorely prone to intellectual folly and societally damaging misuse; this shall be further described in the course of this essay. The public

health bureaucracy, in common with bureaucracies generally, is likewise naturally prone to woefully thoughtless rigidity. That medical practitioners should become caught up in such a technocratic nightmare is sadly unsurprising when one considers the history of medical complicity in pseudo-sciences such as the old American "race science", which justified black slavery, or the worldwide influence of medically-endorsed and hateful eugenics mere decades ago, which reached its criminal apogee under the synonymous appellation of "race hygiene" in Germany.

Laudable advances in medicine and its practice, over the past century, are among the great boons of the period. But the descent of the medical profession into partnership with eugenics, and now with base practices of lifestyle epidemiology, has occurred within the same period. A good general review of how well, and how poorly medicine can be practiced, is provided in Doctor James Le Fanu's popular book *The Rise and Fall of Modern Medicine*. The medical community can perform well. It can also perform dismally. Dismal indeed has been its thinking, and its advocacy on the tobacco issue, since about the nineteen-sixties.

Discussion of one example of egregious advocacy, relating to active smoking, is appropriate at this point. There always have been, and always will be, fanatical elements wishing to abolish practices they consider impure, such as sexual license, or drinking, or smoking. Anti-tobacco fanaticism experienced a resurgence after the US Surgeon General's Report of 1964 and gained considerable ground within a decade thereafter.

In the late nineteen-sixties the US National Cancer Institute had commissioned its Director of Tobacco Research, the toxicologist and epidemiologist Doctor Gio Batta Gori, to assemble a blue ribbon panel of worldwide experts in various disciplines, to the purpose of developing, with the coöperation of the US tobacco industry, a modified design of the cigarette.

Cigarette smoking is the riskiest form of tobacco smoking because cigarette smoke is typically inhaled. The higher risk with cigarettes is particularly noticeable in regard to lung cancer. It was believed that smoking, widely enjoyed worldwide for centuries, and for millennia in the Americas, was no more likely to go away than was the drinking of alcoholic beverages. The disastrous experience of alcohol Prohibition in the US (1919 - 1933) was still fairly fresh in American minds.

Alcoholic beverages cannot be made much safer than they are since the element in them that is potentially dangerous is the alcohol itself. The case with cigarettes is altogether

different. Nicotine is a mild stimulant and not harmful in itself. The 1964 Surgeon General's report stressed that the use of nicotine via smoking, even amongst regular users, though habituating for some, did not meet the definition of addiction. Of course the fanatics now insist that it does.

That matter is moot, as in practice, cigarette smokers regulate their smoking so as to derive the mild effects of nicotine, at once soothing and beneficial to concentration of the mind, according to their mood, and will temporarily desist from smoking when nicotine levels get too high in their systems.

The harmful element in cigarette smoke is what makes the smoke visible: the "tar" or microscopic matter within the smoke. Both tar and nicotine contribute considerably to the characteristic flavor of tobacco. There is also a quantity of water vapor in cigarette smoke. Gori's team decided that cigarettes could be made vastly less dangerous by increasing the level of water vapor, drastically decreasing the level of tar, and, crucially, maintaining the level of nicotine such that smokers would not tend to smoke more cigarettes or to smoke their cigarettes more intensely.

Research went on for several years. Prototype cigarettes were made and tested by various means. It was decided by the late 'seventies that modified cigarettes could be made in the short term which would reduce risk by about half, and it was believed that over time, further improvements could ultimately reduce risk to about a quarter of that produced by conventional cigarettes. The risk of a typical cigarette habit could be reduced to a level comparing to the estimated "risks" of moderate indulgence in such conventional products as snack foods and soda pop.

The revolutionized nature of the new product was such that this risk reduction could be effected with or without cigarette filters; the modified tobacco element itself reduced tar to a minimal level. The increase in water vapor and decrease in tar diminished flavor but it was thought that this could be overcome over time.

Plans were made to improve the new cigarettes in terms of flavor and to introduce them to the market advertised as reduced risk cigarettes. They would be more expensive to produce, but it was discussed that, if taxed at a lower rate than are conventional cigarettes, they could be sold at a similar or somewhat lower price at retail.

Conventional cigarettes, it was predicted, could be phased out of the market over a period of years. With risk ultimately reduced by 75%, lifetime risk to the average smoker would

be virtually eliminated, while the incidence of lung cancer and other related afflictions would be very drastically reduced, even amongst chain-smokers.

That was the state of things in 1979. By that time fanatical elements in the health professions, including at the National Cancer Institute (NCI) in particular, had gained great sway. They killed the project. The tobacco companies were forbidden against introducing any new product with any advertised indication of reduced risk.

The mantras of "there is no such thing as a safe cigarette" and "quit now" have been chanted by the fanatics ever since. The *de facto* message is "quit or die". Those messages have been hammered into the public for decades now. The development of safer cigarettes, back in the 'seventies, is *never* mentioned by the orthodox medical "authorities", and remains little known amongst the public.

Smoking rates were dropping steadily in the 'seventies. The ideologues convinced themselves that, by firmly denying the value or acceptability of moderation in smoking, and likewise denying the very possibility of less risky cigarettes, they could, with an iron fist, reduce cigarette consumption down to their ever-favored quantity of zero. Soon the militant Surgeon General C. Everett Koop was predicting "a smoke-free society by the year 2000." It didn't happen. Smokers have been denied enormously less risky cigarettes for decades now.

Doctor Gori, reflecting on the abrupt termination of the safer cigarette project, commented, "The new policy was: Smokers shouldn't be helped – smokers should be eliminated." He has condemned the morally blind abolitionist crusade, and the enormous harm it has done to smokers worldwide by blocking the introduction of safer products, consistently to this day.

In 2001, the Institute of Medicine of the US National Academy of Sciences, revisited the question of reduced risk cigarettes. They endorsed the very same design that was suggested by the NCI research of the 'seventies and suggested such cigarettes be licensed and released promptly. The tobacco control establishment has stonewalled. Nothing has been done.

The abolitionists continue to condemn the idea of reducing risks of smoking; to them the very idea of safer cigarettes is an anathema: an impediment to the ideal of smoking eradication which they pursue with all the quasi-religious zeal of yesteryear's bible-thumping temperance lecturers.

The anti-tobacco religionists also viciously condemn Doctor Gori for working with the tobacco industry (that was his assignment from the NCI: you do not change the recipe without consulting the cook.)

In continuing research, independently and for the tobacco industry, Gori and a number of his colleagues have calmly adhered for more than thirty years now, in the face of unceasing vituperative fanaticism, to urging government and the industry finally to reëstablish coöperation to a morally compelling end. As Doctor Gori wrote in 2004:

> An implicit but clear premise of the Institute of Medicine report [of 2001] is that smokers – loaded with exorbitant taxes – are entitled to compassionate public health assistance as others are, and should not be cast out, punished, and denied available help.
>
> Indeed, it is unimaginable that public health and legislative authorities should resort to curbing tobacco use by murdering smokers – as they seem inclined to do. The unavoidable implication is that a continuing official reluctance to endorse the development of LHCs [Less Hazardous Cigarettes] is not ethically tenable and amounts to a culpable dereliction of public duty.
>
> This is even more true currently, because an assortment of governments in the U.S. receive around $40 billion annually from tobacco, against less than $9 billion received by the entire industry. It is governments that benefit from and control the tobacco trade far more than the tobacco industry, which has been virtually nationalized by taxation and the Master Settlement Agreement with the states.
>
> At the same time, the cigarette industry should actively seek official approval for developing and promoting LHCs, as a defense against legal challenges if it fails to heed the Institute of Medicine instructions, and as a responsibility of due diligence and concern for smokers.
>
> Key to success would be a realistic FDA [US Food and Drug Administration] acting under an enabling statute, free of undue pressure from prohibitionist and industrial interests.
>
> Predicting the future dynamics of risk reduction remains problematic, but technological achievements already on the table could realize in about a

> decade the Institute of Medicine prediction that "regression of risk ... might eventually bring a smoker to a risk level equal to some lower level of life-time exposure to conventional products." ...
>
> Men of good will have no choice but to embrace the wisdom of less-hazardous cigarettes. Then public health justice will be restored; a majority of world adults who smoke will benefit immensely; and a most awkward and distressing controversy will be extinguished.

Gori referred in this 2004 piece to the potential for the Food and Drug Administration to take control of this situation and to release safer cigarettes urgently. The FDA did, by act of Congress, take control of tobacco products in July of 2009.

In the years since no move has been made on less hazardous cigarettes. The FDA has instead concentrated on trying to get gruesome photographs of corpses and dismembered body parts put onto US cigarette packages. They are fighting this issue gradually through the court system.

As Gori put it: "Indeed, it is unimaginable that public health and legislative authorities should resort to curbing tobacco use by murdering smokers – as they seem inclined to do." They remain so inclined. They even want to glory in color pictures of the corpses.

The fanaticism of the tobacco control movement took firm hold in the health professions and in government decades ago. Its hold in these precincts only increases over time. Correction of fanatically-driven and gross misdirection in the sciences and in government is not likely *ever* to come from within the related professions or agencies. Health professionals who question the fanatical orthodoxy face violent reaction, sure destruction of their careers, from the medical establishment.

Until the nineteen-nineties the tobacco industry defended itself vigorously. Since that time the government, and the law, have taken a firmly adversarial stance against the industry, for instance in coming to accept the statistical evidence of lifestyle epidemiology as "proof" of "causation." The industry has reacted by appeasement, most particularly in the United States, where all the tobacco giants agreed in 1998 to a so-called Master Settlement Agreement, which effectively gags them against any criticism of healthist dogma.

Alvan Feinstein, in a 1992 critique published in the journal *Toxicologic Pathology*, commented on the tyrannical domination of health professions and agencies by anti-

smoking zealots:

> [I]n the current fervor of anti-smoking evangelism, what young scientists would want to risk their careers and what older scientists would want to risk their reputations by doing anything that might be construed as support for the "bad guys" of the tobacco industry? What governmental agency would fund research in which the established "accepted" anti-smoking doctrines were threatened by a study proposed by someone – an obviously deranged skeptic – who wanted to do an unbiased, objective investigation?
>
> The governmental agencies that fund scientific research were once expected to be above the battle, uncommitted, and devoted to seeking truth. For diverse political, social, and fiscal reasons, however, those agecies have often in recent years become mechanisms of advocacy rather than scholarship, pursuing goals of policy rather than science.
>
> ... Besides, the "bad guys" sometimes turn out to be correct. Galileo was assailed by the Church when he doubted Earth's centrality in the solar system; Semmelweiss was denounced by obstetricians when he said their inadequately cleansed hands were transmitting disease; Florence Nightingale was detested by the British establishment when she campaigned for better sanitation of water and sewage; and Joseph Goldberger was deemed a fanatical nuisance when he questioned an esteemed epidemiologic commission's report that pellagra was an infectious disease.
>
> Just as "bad guys" are sometimes right, the "good guys" are sometimes wrong. The history of medicine and public health is replete with the errors (sometimes harmful blunders) committed by revered, respectable leaders in the field. The most recent memorable public events were the unnecessary, fallacious hysteria about Agent Orange, and the needless evacuation of homes (and harm to lives) by residents of an entire town in Missouri, responding to the mistaken zeal of a governmental agency.
>
> The "bad guys", of course, are not always right, but if they are denied a fair and proper scientific hearing, neither society nor science will benefit. Society is entitled to make political decisions based on advocacy. The scientific basis for those decisions, however, should depend not on political advocacy, but on scholarship – no matter how it is produced or by whom.

Vincent-Riccardo DiPierri describes contamination of government and the judiciary by the fanatical medical establishment (which he describes as "medico-materialist") thus:

> The redefining of law, then, is not the result of science or judicious consideration, but sets the severely flawed medico-materialist framework, particularly statisticalism, as the "standard" for legal evidence and argument: The law now represents the anti-smoking, medico-materialist view ... – by definition – as infallible. ... It also has the effect of removing all legal defense from the tobacco industry. Understandably, the tobacco industry, with essentially no hope of victory on these terms, has attempted to contain the "fallout" with a global settlement.
>
> Until this redefinition, the tobacco industry relied on the idea of assumed risk by smokers and the increased taxation/insurance imposed on smokers. While no more was made of the idea of risk, this minimalist approach sufficed. However, by not properly questioning the medico-materialist view over the last decades and relying on the bare minimum in approach, the tobacco industry has now been caught out by the improper enshrining of low-order statistical risk as a legal maxim. This should highlight that the tobacco industry has very little insight into the smoking habit or medico-materialism; in many instances it is its own worst enemy.

The industry has indeed become an enemy to itself. By appeasing fanatics it has become an enemy to its customers as well. It is the people who suffer the most from anti-tobacco fanaticism. A condemnatory tone is very proper and perfectly necessary in discussing these issues. A return of reason can now only be *forced* upon empowered fanatics from outside their corrupted precincts. It is indeed high time we all got angry about the miserable situation, and moved, to fix it.

2. Statistics Madness

Doctor Vincent-Riccardo Di Pierri, the Australian philosopher and psychologist previously quoted, sees in the rampant anti-smoking movement an example of distorted perspective leading to gross dysfunction, notably in the practice of lifestyle epidemiology, which the wider medical community never questions, and transmits to the public at large.

He refers to the purblind philosophical outlook as "materialist", i.e. lacking any genuine academic, intellectual, moral, ethical, or spiritual insight, and as "medico-materialist" specifically in reference to lifestyle epidemiology and its adherents.

Doctor DiPierri considers that the "anti-scientific" perspective of lifestyle epidemiology is in many respects limited to the point of total blindness, as we have seen in terms of its gallingly illogical, simple-minded statistical interpretations, but as DiPierri discusses, this simplistic and anti-intellectual perspective is blind not only to basic logic, but also, and perfectly so, to the terrible harm it does to the public and to society: e.g. in blocking less hazardous cigarettes for decades, and in much else, as we shall further describe in the course of this essay.

The medico-materialist outlook is, simply, ethically reckless and morally wretched. The medico-materialist attitude is, furthermore, arrogantly dictatorial. The former US Surgeon General C. Everett Koop (term of office 1982-1989: pictured at left) was the first among the Surgeons General to affect military garb; all his successors have "followed suit" in this. Koop saw it as his job to dictate personal behavior as a kind of Mussolini of Medicine. (Imagine the outcry if the US Attorney General dressed up as a Führer of the Law and began issuing unilateral diktats.) Doctor Koop expressed the morally vacuous medico-materialist philosophy succinctly in 1996: "From my point of view, anything that stops smoking is good."

DiPierri discusses the self-superior and "externalist" aspects of what he calls the anti-tobacco "cult". The cultists do not detect the patent illogic of many of their statements because they unthinkingly parrot each other without ever actually analyzing the bases of their own beliefs.

They never look inward, critically, at their individual thoughts. Their idea of critical insight is limited to corroborating an individual cultist's statements and beliefs with those of others within the cult. If their fellow believers are babbling the same things they babble, then by definition, there is a consensus of belief amongst "superior" thinkers, thus their fallacious surmises are right, infallibly so.

On the other hand, since the cultists believe their own consensus-thinking to be unimpeachable, any cult outsider who criticizes their mantras is considered, also by definition, as an inferior, unworthy of being listened to, and as a danger, which must be attacked.

This vigilant resistance to critical thought underlies the anti-scientific nature of the mindset: cultists, in their debased research and thinking, seek *only* to strengthen their biased preconceptions, so are reflexively antagonistic to facts, and to basic logic, and to persons, which refute these preconceptions.

Recall, for instance, Doctor Clanton in the PBS interview, brushing off the moderator's logical interjection, and reacting to it *instantly* with recapitulation of his lunatic belief that persons who do not smoke, or who quit within 20 pack years, have a "zero" risk of lung cancer.

Recall also the ideologues' personal attacks on Gio Gori and his colleagues, and their vehemence in insisting that safer cigarettes not be made, despite that such cigarettes, as National Cancer Institute and Institute of Medicine research have mutually and clearly verified, could have been produced for decades now, to the immeasurable benefit of smokers.

Safer smoking? A value in moderation? Heresy! Smokers benefitted? The ideologues would rather see them dead. That is the pretty picture they want and need to have emblazoned on all cigarette packs.

The mentality believes, profoundly, right into its bones, that adherence to absolute anti-smoking dogma is the only standard of correctness, or as DiPierri put it: "[C]omprehending no higher standard of inference, it is utterly convinced of the 'rightness' of its surmising."

Just as the "externalist" view leads the believers to unwavering, bullheaded insistence on their debased cult-held beliefs, it likewise leads these cultists to perceive that their bodies, and the bodies of all those who obey their health advice, are inherently infallible. The fault

is never within, always without.

Therefore they tend strongly to think that virtually all physical ailments are caused by external factors. This is why they never, except under duress, consider base risk with regard to cancer. Relative risk, in their fallacious outlook, is everything: "all" is "caused" by external factors.

DiPierri notes that this externalist bias is classically symptomatic of disordered or pathological thinking. In order to exalt the self, persons suffering perceptual disorders often "project" their inherent deficiencies onto other persons: e.g. "My co-workers are all boors" instead of "I have poor social skills": or onto outside factors: e.g. "My co-workers are too noisy" instead of "I can't concentrate at work because of my own disordered thinking."

As DiPierri has explained:

> In this materialist view psychological, psychosocial, moral and spiritual dimensions are obliterated; the human is no more than a "biological organism" with behavioral reactions to external events.
>
> It will be considered that since the 1970's materialism has been building in domination of key social institutions such as governments, the medical establishment, academia, and the media. Medico-materialism has figured highly in this circumstance. Medico-materialism has been given more and more say in health policy and now even attempts to prescribe the "ideal" lifestyle. Medico-materialist prescriptions/proscriptions are underlain by epidemiology which is the study of factors (e.g., diet, exercise, smoking) associated with what are termed "lifestyle" diseases (e.g, cancer, coronary heart disease).
>
> ... Statisticalist over-interpretation, which involves an improper straddling of both deterministic and probabilistic frameworks, promotes superstitious belief as a matter of course, i.e. anti-scientific. Unfortunately, it is also self-serving in fostering the misperception that medicine understands far more about disease aetiology than it actually does. Additionally, promoting false belief also subserves a greater medico-materialist production-line of screenings, testings, consultations, etc. (economic opportunism or raw capitalism). With the dawning of the new millennium, the public is under a

> constant barrage of questionable "health" promotion, e.g., diet, smoking, exercise.
>
> ... Troubled minds, unwilling to address problems where they occur (internally), project internal conflict outward. Externalities then appear "dangerous," the greater the internal conflict, the more "dangerous" seem the externalities. This underlies much contemporary obsession with the environment and particularly antismoking. Smoking and exposure to smoke have been manufactured into a conduit for projected inner conflict, i.e., a contemporary scapegoat, bigotry.

We have said that the statistical link between active tobacco smoking and lung cancer is very clear and have analyzed the nature of this influence in the previous section of this paper, accounting for it broadly and approximately in constructing a balanced overview, across wide populations. The influence of smoking is the only clear influence affecting a large percentage of the population with regard to lung cancer.

There are industrial exposures (most notably relating to asbestos workers and miners) which have affected particularly assigned workers in particular industries. Some of these risk factors rival long-term heavy smoking as influences on lung cancer. These industrially-related influences have affected both smoking and non-smoking workers in particular professions. Since the effect of these exposures is limited to a very small subset of the population their overall influence, on population-wide statistics, is quite marginal.

With its firm externalist bias, however, lifestyle epidemiology has over the years studied a positively enormous array of lifestyle factors, trying to find links with lung cancer, often in as or *more* idiotic a manner as did our fictitious Doctor Who, with his altogether fictitious Funk's Syndrome.

This section will report on such "externalist" research. Let us begin on this by revisiting the CNN interview with Doctor Sanjay Gupta.

> ROBERTS: All right. Question number two: Smoking is by far the number-one cause of lung cancer, but radon gas is the leading cause among non-smokers; true or false?
>
> GUPTA: That is true. And this is actually surprising to a lot of people. Smoking is far and away the number-one cause. You know, eight – eight or

> nine times out of 10, it's going to be smoking. But radon, which is this naturally occurring uranium byproduct found in the soil, can actually infiltrate into your basement, and has been associated with lung cancer as well. So, it's actually the second most common cause of lung cancer.
>
> ROBERTS: And I think I know the answer to this question, number three: Asbestos causes lung cancer; true or false?
>
> GUPTA: That is true as well – a lot made of asbestos over the years. You won't find much asbestos anymore, because of all the regulations with regards to building, John. But asbestos specifically causes a type of cancer known as mesothelioma. And that is a type of lung cancer that is – is somewhat treatable, but can also be very deadly, if not caught early.

Asbestos products have become increasingly regulated, many of them banned, variously in various nations, over the past few decades. Some asbestos products are still manufactured, under tightly controlled conditions, but the use of asbestos products is no longer very common. Decades ago asbestos was quite commonly used, in products including automobile brake shoes (still common but with a reduced quantity of asbestos), household flooring and exterior tiles, and especially as home insulation; the home uses are extinct with regard to new construction but still exist commonly in old homes. Asbestos insulation was almost universal in early twentieth century homes as this insulation was extremely efficient and also cheap.

Asbestos is a naturally occurring fibrous silicate mineral which is mined from the earth. When inhaled asbestos fibers can lodge into lung tissue. Breathing these fibers in high concentration over a long period of time can damage the lungs sufficiently to impair lung function. Impairment of lung function in result of asbestos exposure is called asbestosis. Asbestosis is not a cancerous condition but can in some cases be a harbinger of cancer.

Extreme asbestos exposure can also lead to a cancer called mesothelioma. This may occur long after the exposure occurred; the average age at diagnosis is similar as with lung cancer. Mesothelioma is not unique to persons with high asbestos exposure but is very strongly associated with such exposure. Mesothelioma, though it can appear in some other body locations, most commonly appears in the pleura, or outer lining, of the lung.

Decades ago, cancers of the lung pleura were included in the definition of lung cancer, but by modern definition pleural cancers are considered separately. However, pleural cancers

can spread to the inner lung, and persons with high exposure to asbestos are also prone to (primary) lung cancer generically; i.e. to the common forms of lung cancer. Smoking does not increase the risk of mesothelioma but it can work in synergy with asbestos exposure to increase the risk of primary lung cancer.

The risks of asbestos-related ailments are highest amongst asbestos miners, and amongst workers in asbestos product manufacturing plants (it is reported that in some plants, during the heyday of asbestos product manufacturing, the proliferance of asbestos fibers in the air resembled a snow storm), and amongst those in professions which may involve tearing of materials, and thus of releasing fibers into the air, such as asbestos insulation removal.

As long as asbestos home insulation is not disturbed or allowed to deteriorate it presents no practical danger. Exposed pipes with old asbestos insulation are often wrapped with tape to keep fibers from drifting into the air. Hard products such as linoleum or home exterior tiles which may contain asbestos are not a concern unless they are broken up or pulverized in the process of removal.

Reaction to high asbestos exposure has varied greatly between individuals. Some will develop asbestosis, or mesothelioma, or both, while others will not. So it is with primary lung cancer. Asbestos-related health risks are a serious concern for persons with high occupational exposure. Some others have experienced high exposure, for instance, in living proximately to where asbestos waste was carelessly disposed of in the past. These represent very small subsets of the population at large.

The other most common occupational risk for lung cancer is that of miners generally. Asbestos miners are subject to a specific risk of asbestosis since the silicate asbestos can lodge into lung tissue. In other types of mines, asbestos silicate may be absent or nearly so, but there will always be silicate matter (in effect, tiny slivers of rock dust) in the mine atmosphere, which is generally dank and dirty. Thus miners generically are at risk of lung impairment called silicosis, which has similar symptoms as described for asbestosis, and which likewise can be a harbinger of lung cancer. Coal miners particularly can also get what is called black lung disease and this may also increase their risk of future lung cancer.

The dank atmosphere of mines typically also includes high concentrations of such particular nasties as metallic dusts, including lead, mercury, tungsten, nickel, and silver, as well as arsenic, carbon monoxide, sulphur dioxide, diesel fumes, nitrous fumes, formaldehyde, and combustible dust. Another aspect of mine atmosphere is a

concentration of radon, a gas with radioactive properties, in vastly higher quantities than one encounters above ground.

Lifestyle epidemiologists have taken a focus on radon in relation to relatively high rates of lung cancer in both smoking and in non-smoking miners. Radon may certainly play a role in influencing lung cancer rates in miners. Uranium miners may be particularly prone to lung cancer as compared with miners generically although not by a very great margin. Radon is a by-product of uranium decay.

The relative influence of radon, in synergy with the other foul and suspect ingredients of deep mine atmospheres, on lung cancer risk, is and must always remain an open question; it's really impossible to separate risks out of the comingled factors.

Doctor Gupta's flat statement that radon is "the second most common cause of lung cancer" is an entirely baseless mantra. The potential contributory role of radon in miners' lung cancer risks is eminently debatable, and Gupta is not speaking of miners' risks anyway, as miners, like asbestos workers, represent a very small subset of the population. He is speaking of extrapolations that lifestyle epidemiologists have made, from what is likely an over-interpretation of the role of radon in miners' risks, to the population at large.

Radon can seep into basements, particularly dirt-floored basements of antique homes, and it can also rise, to a limited extent, into higher storeys of buildings, in higher concentrations than one would encounter outdoors, but in vastly lower concentrations to those found deep within mines. The lifestyle epidemiologists, similarly as they brainlessly attribute all lung cancer in smokers exclusively to smoking, go on brainlessly attributing lung cancer, sometimes the same person's lung cancer, to other things more or less pulled out of their hats. In one mood, they would attribute a smoking miner's lung cancer to smoking, in another mood to radon. Radon has simply become a favored "cause of lung cancer" from the hodge-podge batch they hold in their mad hats.

Radon, in small quantities, is *everywhere*. All of us breathe it in all day every day together with all the constituents of the air around us. As DiPierri notes: "Typical air contains all manner of material: viral, bacterial, fungal, dead skin particles, human and animal dander, other particulate matter and gases, i.e., a veritable debris-field ... Whether air is safe does not rely on whether it is 'clean' but whether it can be adequately processed within a normative range of functioning ..."

You will see a number of residential radon study results in the tables to follow. They

signify nothing. Attempts at meta-analysis (a dubious cult-favored method of pooling studies, all of which, regarding radon, are individually debased in method) have simply further muddled the matter with conflicting and converging inconsistencies.

There never was good reason to suspect and there is no reason whatsoever to think that the public at large is at any particular risk from radon, something all animals have been breathing, all the time, every day, since first there were any animals.

How much does a very high concentration of radon contribute to miners' lung cancer risk? Very possibly some, in synergy with other aspects of mine atmospheres, but it's impossible to say how much radon in itself contributes to miners' risks. How much sense does it make to apply a guess about the radon risk to miners onto everybody on the planet? None. But that's what the mad hatters have done.

The orthodox consensus about radon peril relies on ludicrous extrapolations from studies of miners extended to the public at large. For example, an excerpt from the June, 1995 *Journal of the National Cancer Institute* article "Lung Cancer in Radon-exposed Miners and Estimation of Risk From Indoor Exposure" reports on research conducted by Jay H. Lubin et alia, primarily on uranium miners, as well as some other metal miners.

> Radioactive radon is an inert gas that can migrate from soils and rocks and accumulate in enclosed areas, such as homes and underground mines. Studies of miners show that exposure to radon decay products causes lung cancer [always "causes", never "influences risk": most miners, of course, never get lung cancer]. Consequently, it is of public health interest to estimate accurately the consequences of daily, low-level exposure in homes to this known carcinogen. Epidemiologic studies of residential radon exposure are burdened by an inability to estimate exposure accurately, low total exposure, and subsequent small excess risks. As a result, the studies have been inconclusive to date. Estimates of the hazard posed by residential radon have been based on analyses of data on miners, with recent estimates based on a pooling of four occupational cohort studies of miners, including 360 lung cancer deaths.
>
> ... This risk model estimates that reducing radon in all homes exceeding the U. S. Environmental Protection Agency's recommended action level may reduce lung cancer deaths about 2%–4%. These estimates should be interpreted with caution, because concomitant exposures of miners to agents

> such as arsenic or diesel exhaust may modify the radon effect and, when considered together with other differences between homes and mines, might reduce the generalizability of findings in miners.

You mean, there's a difference, between your house and, say, a uranium mine? Really? So one might use caution in generalizing between miners and folks who simply live indoors, and a daft guess of a reduction of US lung cancer deaths from about 160,000 to about 155,000 annually from better basement ventilation, might be just a bit speculative? Don't folks, even those who live in basement apartments, open their windows or doors most days, anyway?

Should we then doubt the killing potential of radon for the average person? Of course, yes, but then again no, not for the simplistic Doctor Guptas and all the other mantra-chanting Doctor Whos of the world. Damn the doubt! Radon's killing folks all over the place all of the time. Never mind that all of them, including all the folks who never get lung cancer, are breathing it all the time. That actually makes radon a perfect Enemy Without. It can be applied to anybody anytime.

Radon, say it clear, is "the second most common cause of lung cancer." Why not? And why, then, doesn't everybody get lung cancer? Well, the vast majority of people are obviously freaks, immune to the positively ubiquitous cause of lung cancer. That's that, tell the world, and they'll believe it.

The cultists can say radon causes lung cancer. They could as easily say oxygen causes it. Everybody breathes that too. The trouble is, only a single digit percentage of deaths are lung cancer deaths, so we have to face the facts that while everybody breathes, not everybody gets lung cancer, and that lots of those who do get it have no history of markedly unusual exposures, and didn't smoke, didn't work with asbestos, and were not miners.

They also all lived indoors, at least most of them most of the time we hope, and how much radon there might have been in their houses varied drastically whenever they opened a door or window. They all breathed radon, since everybody does, and there is just no telling how much one or another of them did, over their lifetimes.

Going back to their houses with a radon monitor after they're hospitalized or dead tells nothing about the past. Ask any realty agent about radon tests and about cracking windows open: any house can register as relatively low in radon. On the other hand, if you

keep all doors and windows shut tight for days or longer specifically for the test, you're doing what the ailing or deceased homeowner did not do, or rarely did. But the "radon kills" tenet is foolish on any account. Blaming lung cancer on simply breathing just doesn't help.

We do all breathe, and there is no zero risk of lung cancer for anybody, and animals have always breathed, and they have always been susceptible to lung cancer, for time immemorial. There is inherent cancer risk. It is a base risk that has always existed. Cancer can occur, most anywhere in our bodies, with or *without* any particularly suspect outside influence.

The "why" of that lies inscrutably in Nature as much as does the "why" of mortality itself. If ever there may be a real cure for cancer it will come through better understanding of life processes themselves: particularly of cell replication and of its regulation by the immune system. Blaming breathing for cancer doesn't hit the mark. The true base of the cancer enemy lies within, not without.

You will never convince the "externalist" groupthinkers of that but an expansive and objective view allows of no other conclusion. An imbecilic view naturally demands something else. Thus, in their vigilant attack on Reason, our stalwart grant-seeking lifestyle epidemiologists have labored for decades now, creating an illimitable list of other "causes" of lung cancer.

Before proceeding to a representative sample of results from these researchers' "scientific studies", because the subject will come up within the reports on these studies and also further on in this paper, let us digress to a summary description and some pertinent discussion of the major types, or "histological" classifications, of lung cancers.

We have already mentioned the usually benign carcinoid lung tumors, which are not related to smoking, and which are uncommon. The more typical types of lung cancers, all with considerable potential for metastasizing, are squamous cell, small cell, large cell, and adenocarcinomas; there is a sub-type of adenocarcinoma called bronchiolo-alveolar lung cancer, or bronchio-alveolar, or sometimes and more simply, alveolar lung cancer, the term we will here prefer.

In some researchers' reports alveolar lung cancer is treated as a distinct histological type rather than as a sub-type of adenocarcinoma. We shall in this essay generally use the term adenocarcinoma, according to standard definition, as including the alveolar sub-type.

All of the four main lung cancer types, squamous cell, small cell, large cell, and adenocarcinoma, occur in both smokers and in never smokers, but the types appear in different proportions amongst the two groups.

A clinician's determination of type entails examination of tumors and their location within the lung as well as microscopic examination of cancer cells. Since the nature of cancer is to grow and spread the location of tumors becomes variable over time, and also with regard to the appearance of tumors and their cells, distinction of type is not always clear or easy.

Various clinicians, looking at the same person's lung cancer, will sometimes differ in their opinions regarding histological type. In some cases a patient's lung cancer may not fit well into just one of the diagnostic slots. Clinicians simply use their best judgement.

Squamous and small cell lung cancers are the types most associated with smoking risk and they appear usually in central portions of the lung, in or near the bronchi, or main breathing passages. Large cell lung cancer may appear more deeply in the lung and is less associated with smoking.

Adenocarcinoma accounts for the lion's share of never-smokers' lung cancer and is characteristically located deep within the lung. Its alveolar sub-type is found deepest of all within the lung, in the air sacs, near the lungs' perimeter.

While smoking has, since close research began in the nineteen-fifties, been considered to influence risk of squamous cell, small cell and, to a lesser extent, large cell lung cancers, for decades smoking was considered unrelated to adenocarcinoma. Studies over many years suggested that smokers were no more, indeed possibly less prone to adenocarcinoma, than were never smokers. It was taken as established that adenocarcinoma, which has always been the most common type amongst never smokers, entailed no heightened risk attributable to smoking.

Beginning in the nineteen-eighties researchers started suggesting that adenocarcinoma was related to smoking though to a lesser extent than is the case for the other main lung cancer types. This new suggestion came on the heels of an enigmatic rise in the diagnostic incidence rates of adenocarcinoma, relative to the other main types, in most of the world, this being particularly notable in the United States.

Researchers attempted to explain the relative and surprising advance of adenocarcinoma, which began to show itself in the nineteen-fifties, to decreases generally in tar and nicotine

levels of cigarettes, which also occurred in the 'fifties, and to smokers' growing preferences from that period onward for filtered and light cigarettes. Adenocarcinomas, characteristically, are located more deeply in the lung than are the other main lung cancer types, and it was suggested that smokers were breathing in milder cigarettes more deeply.

However, with lung cancer being a disease of old age, and with changes in cigarette design affecting the young more than the old in terms of lifetime smoking, the steady increase in incidence of the adenocarcinoma types, in the immediate time frame, did not reflect the time lag one would expect in reflection of the introduction of weaker strength cigarettes.

Incidence of squamous cell lung cancer, a type more typical of smokers than of never smokers, has declined over time, more notably amongst men than amongst women, since men smoked more than did women, and since men's smoking rates peaked somewhat earlier than is the case for women. This decline in incidence rates of squamous cell lung cancer reflects an expected delay of many years from the time when smoking rates began declining. The squamous decline makes sense in terms of comparing historic smoking behavior with subsequent incidence rates. The steady increase in adenocarcinoma did not make sense on such a basis.

The cigarette design explanation, therefore, was quite inadequate. This inadequacy became more starkly evident when, in 1999, the diagnostic incidence rates of adenocarcinoma began declining, rather more sharply than they had been increasing from the early nineteen-fifties through to the late 'nineties. This suddenly appearing decline continues to the present. Of course, there has been no mass return to old unfiltered cigarettes, in the meantime. Cigarette design conclusively does not explain the rise and fall of adenocarcinoma over the past sixty years.

A chart showing the US incidence of lung adenocarcinoma, and also of squamous lung cancer, between 1973-2003, from the journal *Chest* of May, 2007, appears on following page.

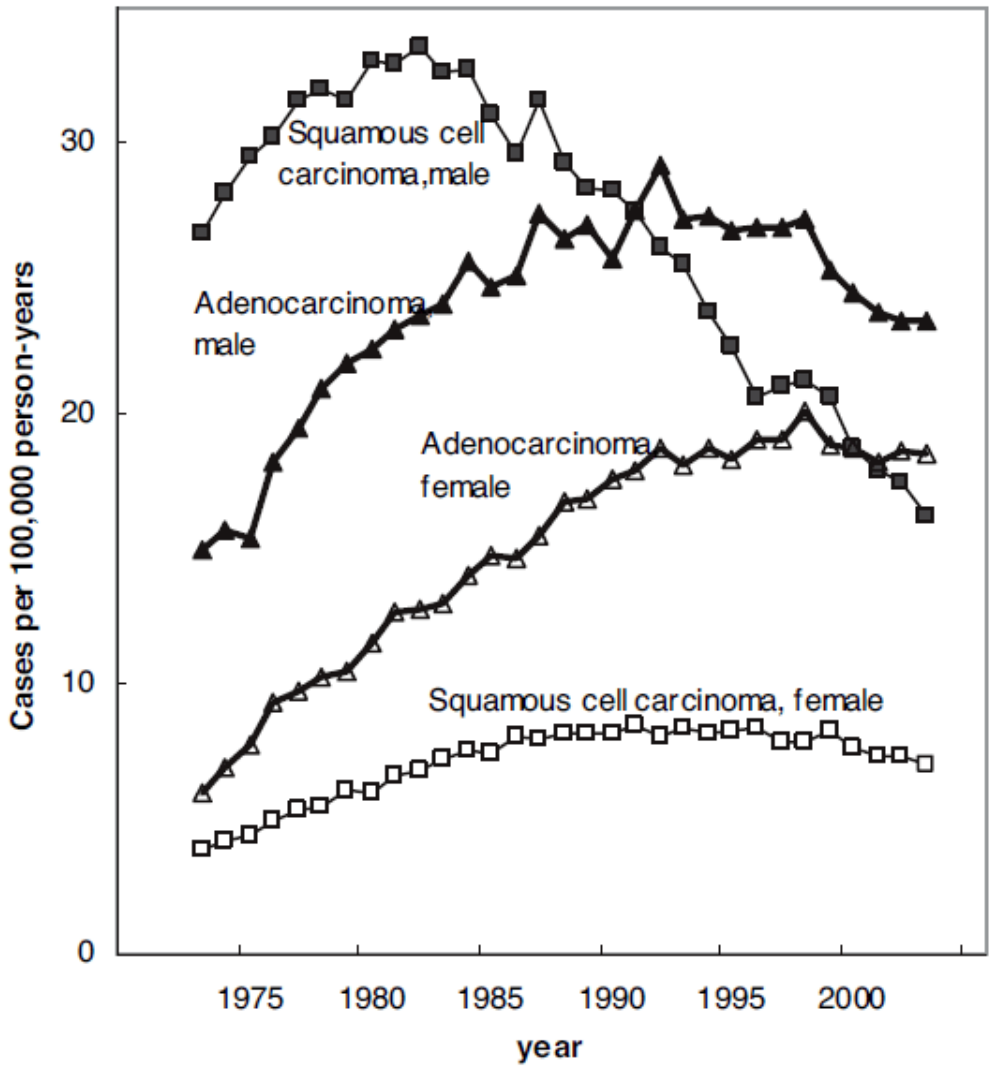

FIGURE 1. Incidence rates according to histologic type and gender. Rates are age adjusted to the 2000 standard population.

The incremental rise over decades, and now the sudden fall in adenocarcinoma, strongly resembles what one typically sees in classic epidemiology, which is, distinctly from the "lifestyle epidemiology" we have discussed, a field of research which traces the course of infectious agents (contagious viruses or "bugs") as they move through populations. The rise and fall of lung adenocarcinoma may well reflect the movement of a pathogen through the population, gaining stealthily over time, and then abruptly waning, which is the typical course of infectious agents.

Pathogens can be strong influences on cancer. The most famous example is that of HPV (genital wart) infection and subsequent heightened risk in females of cervical cancer. HPV

infection is likewise implicated in some other cancers of the pelvic areas, amongst both sexes, and there plausibly may also be some lesser relationship between HPV infection and lung cancer generically.

Study relating more specifically to adenocarcinoma has suggested that a retrovirus very strongly associated with adenocarcinoma in sheep, called JSRV or Jaagsiekte Sheep Retrovirus, may have mutated and migrated, or else may have retrovirus cousins that have found their way, into the human population.

Research on this subject is scanty, however, and certainly inconclusive. As of the present, it can only be stated that incidence of lung adenocarcinoma over the past sixty years has risen and fallen oddly, very possibly under the influence of an infectious agent, but if that is the case, there is no clear answer yet as to what infectious agent may be in play.

An alternate explanation which has been suggested by American researchers for the rise and fall of adenocarcinoma is that it may be a reaction to air pollution. This is implausible in that air pollution levels have been reduced, between about the 'seventies to the present, in slow gradation. Air pollution did not suddenly fall off, at any point, as the adenocarcinoma rates are now doing.

One can also note that the suggestion that adenocarcinoma is linked with smoking came at the same time that adenocarcinoma incidence was oddly rising, and also coincidentally, with the emergence of the fanatical anti-tobacco element in the health professions. This was the period when smoking-related RR reports for lung cancer generically were rising.

The oddities in adenocarcinoma incidence becloud the era, but growing diagnostic bias is certainly the primary element in the switch from the old contention that smoking was not related to adenocarcinoma, to the new, that smoking is least associated with adenocarcinoma, but is related to all types of lung cancer except rare carcinoid tumors.

There are some other fairly common alternative terms used to designate the basic lung cancer histological types we have mentioned. These include:

Oat cell lung cancer: The more common form of small cell lung cancer. The term "oat cell lung cancer" is often also used as a direct synonym for small cell lung cancer generically.

Combined small cell lung cancer: A less common variant form of small cell lung cancer.

NSCLC or non-small-cell lung cancer: Large cell, squamous cell, and lung adenocarcinoma (NSCLC is generally meant to exclude both small cell lung cancer and carcinoid tumors.)

Anaplastic / Undifferentiated: The term "anaplasia" refers to degenerative mutation of cells. Small and large cell lung cancers are typified by degenerate or primitively undifferentiated cellular characteristics and are sometimes called "small cell anaplastic" or "large cell anaplastic." When reference is made simply to "anaplastic lung cancer" or to "undifferentiated lung cancer" the speaker may be referring to small cell, or to large cell lung cancer, or to both.

Kreyberg group I: Originally defined by researcher Liev Kreyberg, in the nineteen-fifties, to include squamous and small cell lung cancers. Kreyberg's basis for his definition was that squamous and small cell lung cancers were typically centrally located in the lung, closely linked with smoking, and more common in men than in women. The term Kreyberg I is not always used by study authors according to its original definition. Some authors also include large cell lung cancer as a Kreyberg I cancer.

Kreyberg group II: Originally defined by Kreyberg to include large cell and adenocarcinoma lung cancer. The basis was that large cell lung cancer was not strongly associated with smoking, and that adenocarcinoma was not believed to be associated with lung cancer, with these types being characteristically located deep in the lung, and more common in women than in men. Some study authors revise the original definition of the Kreyberg II classification, using it to designate only the adenocarcinoma types of lung cancer.

Centrally located tumors: A general term of classification, not specifically defined, but equating to Kreyberg I, i.e. usually referring to squamous and small cell lung cancer.

Peripherally located tumors: Another general term of classification, not specifically defined, but equating to Kreyberg II, i.e. usually referring to lung adenocarcinoma and to large cell lung cancer.

Apart from lung cancers of the four major histological types, and carcinoid tumors, there are some rare and exotic lung cancers which are given distinct designations. These anomalies, representing a minuscule proportion of lung cancer as a whole, do not pertain to the lifestyle epidemiology we shall review here.

The study results shown on table below are culled from professional literature dating from

the ‘seventies to recent years. Statistical studies on characteristics of population segments, typically performed under the banner of eugenics, became increasingly common from the early twentieth century onward. Studies on lifestyle factors, particularly on smoking, and the relation of these factors with disease outcomes, proliferated from mid-century onward. These came to be known as studies in “lifestyle epidemiology”, a distinction from classic epidemiology, or the study of communicable disease transmission across populations.

The studies on smoking, in the ‘fifties and ‘sixties, attracted increasing attention. Particularly in regard to lung cancer these studies did suggest a link, albeit very roughly, and although the rough import of this research was over-interpreted to the point of gross misinterpretation, this was overlooked amidst a tremendous growth in grant funding for more research along the same lines. “Lifestyle epidemiology” became a big business.

The lifestyle epidemiologists began making careers out of studying just about everything under the sun. Like our fictitious Doctor Who, they plumbed for any “statistically significant” fluke link they could find between just about anything about public habits or the environment that they could think of, in relation to any disease they could think of.

The “experts” began to treat any result they came up with, even if “statistically insignificant”, as being of serious import, so long as it leaned in the direction of their biases. Results that conflict with their prejudices tend to receive scant and denigratory mention in their final reports.

Studies that conflict strongly with general preconceptions may be rejected by journal editors or, in anticipation of this, may never get submitted for publication: a phenomenon which the “discipline” of lifestyle epidemiology occasionally admits to, calling this “publication bias.” Like diagnostic bias amongst clinicians, publication bias in lifestyle epidemiology is a phantom, clearly present, but elusive of delineation.

As previously described, the statistics madness of lifestyle epidemiology has spawned a generally mad outlook on human behavior and health amongst the health professions, and the more fanatical health professionals came to hold great sway in the ‘seventies. It was at this time that lifestyle epidemiology reports themselves became a widespread epidemic which has not yet reached its peak. It spreads the madness. It seeks, with success amongst much of the public, to spread fear about everyone’s every move.

Lung cancer remains a favored topic in studies with wide-ranging focus. We will not here report on asbestos or miner studies as these risks, and the merits and some demerits of

research upon them, have already been discussed. There is risk in working long term as a miner. There was risk in working long-term and closely with asbestos although use of asbestos is now greatly curtailed and those who do work closely with it now benefit from comprehensive precautionary measures which did not exist decades ago.

Research on other and more common occupations is widespread. There has been suggestion that general construction workers, who come into contact with various suspect working conditions, including some contact with asbestos even today, may have risks. We include a number of results on that topic.

Suspicion of excess risk amongst professional cooks and food service or bar and restaurant staff, who are exposed to cooking fumes, has produced studies in the East and in the West. Cooking fume exposure in the home has likewise been studied. Numerous results from such studies are shown here.

Argument has occurred amongst lifestyle epidemiologists regarding the occupation of farming. There has been considerable study on this. Farmers have been supposed to be at low risk of lung cancer because they work in the open country air, or at high risk, because many of them work with pesticides. Varied results of this research appear below.

Studies of home exposure to radon are difficult to perform. How does one estimate how much radon a particular person has breathed in when everyone breathes in radon all the time? Results of such studies appear below with descriptions of their variously contrived investigation strategies. Some similar studies on aspects of home heating, and on automobile and industrial air pollution, also are reviewed below.

What else has been studied? You name it! Are you more at risk of lung cancer if you are tall? Or is it riskier to be short? Do you prefer to drink milk, or liquor? Either preference may actually be healthier in terms of lung cancer according to the "authorities"; edify yourself via the "scientific results" below. Do you like rice pudding? What's the lung cancer risk in that? Do you play mah jong? Learn here of your peril. Own a pet bird? Some researchers have a pet theory that the scent of bird droppings, which you might not even have noticed, may be killing you. Read the results and decide whether, in the cause of self-preservation, you need to toss dear Chirpy out into the wintry skies.

There is lots of such comedy amidst the intellectual tragedy of lifestyle epidemiology outlined on our table. Publication citations appear amongst appendices to this essay. In general we let results speak for themselves below, but as we did with our execrable but

fictitious Doctor Who study, where absolutely required in listing these sometimes even more execrable real-life study results, we will explain and suitably deride.

The next section of this paper deals distinctly with the "passive smoking" or ETS / Environmental Tobacco Smoke debacle. For that close analysis we present those ETS studies which previous compilers have defined as "standard" studies (general criteria for acceptability including that studies should be of never smoker lung cancer cases compared to controls unafflicted with smoking-related disease.) Some of the studies on the following chart make mention of ETS based on non-standard methods; such results are noted in "comments" sections for these individual studies.

On the table, as is a standard for such compendia as this, studies are referred to according to principal author's name. Where necessary for clarity first intial is also given. If more than one study on our tables has been performed by the same principal author the name designation is accompanied by a year designation: e.g. Boffetta (98) and Boffetta (99).

Studies typically present results similarly as we did for our Doctor Who study, giving RR followed by a confidence interval. This is not however always the case. Instead of a confidence interval some authors will give a P value indication, or sometimes just a statement within text, as to whether a given RR is statistically significant or statistically insignificant. Sometimes only a textual report to the effect of "no risk was found" or "no significant risk was found" will be the only report with no specific RR given.

In other cases a study author may not even mention any result for a particular factor but that result may be apparent from case/control cell counts provided in the study report. Where appropriate we will present such results by computation. In such cases cell counts will be shown in "comments" as: (cases exposed / cases unexposed / controls exposed / controls unexposed). For example, our Doctor Who workplace result "2 x 2" or "cell count" table was:

High Intensity / Work	Cases	Controls
Yes	8	2
No	12	18

In our abbreviated form this would be shown as: (8/12/2/18).

Where indication of no risk is given without specific RR we represent this on table as "NR"

(for "no risk") color coded red. Statistically significant results suggesting protection provided by a given exposure are color coded in pink. Statistically significant results suggesting risk entailed from a given exposure are color coded in brown. Never forget that statistical significance by no means suggests practical significance: all results must be viewed and reviewed critically. Results which are statistically insignificant, i.e. self-negating by definition, are color coded in blue on the table. Where RR is provided and statistical significance or insignificance is stated without a confidence interval the same color codes are used with "NS" indicating "not statistically significant" and "S" indicating "statistically significant".

Information is categorized on table by: Principal author; Year of publication; Location where study was performed (country or region); Sex(es) of lung cancer cases; Relative risk(s); Confidence interval(s). One or two results are included from each study. Indication as to where these results appear within the original study, or where within a secondary publication of the study's results, is provided in the "Comments" section for each entry. This may be given as a page number (P) within the report (e.g. P 2 of 6) or reference may be to a table (T) or tables provided within the report (e.g. T 13, T 17). Where appropriate fuller description of where particular results were located will be given. Some results have been culled from study "abstracts": a study's abstract is an abbreviated study report.

Most studies report results to two decimals. Some report to one decimal or to more than two decimals. For uniformity, in the relative risk column, we keep to two decimals (e.g. reporting 1.4 as 1.40), except for unusual cases as noted in context.

What can be made of the information below? In the more comical instances nothing. If you compare results, up and down on the table, you will find many instances where the very same factor will be "dangerous" at times and "protective" at others.

High exposure to cooking fumes is more often shown as risky, rather than not, but results regarding kitchen and general home ventilation (e.g. exhaust fan above the stove, opening doors and windows), as one might expect, suggest cooking smoke is ameliorable. Radon results, including a clear fluke or two, are perfectly underwhelming. Again, there is suggestion in studies that simple ventilation can obviate home atmosphere "peril". We do not live in uranium mines.

In fact the one factor we have not seen reported as perilous is basic ventilation. Perhaps it has been found perilous but the results did not get published. One thing is perfectly certain. Statistics, in the hands of fools, can "prove" anything.

You will best appreciate this, as suggested above, by comparing and contrasting the epidemiological hodge-podge of "scientific" results, up and down the rows and pages immediately following. If you enjoy laughing, also keep an eye out for some of the extra-wide confidence intervals, as unlimited in their potential as is lifestyle epidemiology psychosis itself.

A widely representative sampling of "authoritative research" is here provided. Table begins on following page. Warning: if you thought our fictitious Doctor Who was implausibly deranged: you were very wrong.

Principal Author	Year	Location	Sex	Relative Risk(s)	Confidence Interval(s)	Comments
Williams	1977	United States	M&F M&F	NR NR	NR (food service) NR (construction)	[Text throughout, T 6]. Comments extended below.
Williams 1977 Comments: No significant lung cancer (specific) risk relative to (generic) employment in the two industries based on controlled analyses. Occupational classifications are designated by then-prevailing US Census codes (067/077 for Construction and 910/916 for Food Service Workers) in Roger Williams's wide-ranging 1977 study of professional occupation and cancer. Both sexes were represented in both industries in total case group (study was of all cancers not just lung cancer) with females predominating in food service and in the lung cancer total from that profession while males constituted the majority in construction and the entirety of lung cancer incidence amongst construction industry workers. This study also includes an unrefined comparison to general population death rates, suggesting a significant risk of 1.65 for male construction workers, which was disconfirmed in the controlled analyses. The crude comparison was restricted to major industry classifications. Only controlled analyses were performed referent to food service workers.						
Lee, J.	1983	United States	M F	**3.70** 1.80	**1.20-11.50 (maximum)** 0.60-5.20 (minimum)	[Results published in letters section of *The Lancet*]. Highest and lowest computations reflecting risk for tall persons versus short persons for lung cancer. We suggest you start smoking to stunt your growth.
Buffler	1984	United States	M F	**2.56** 1.52	**1.49-4.41** 0.91-2.55	[T 13, T 17]. Results reflecting occupation in the construction industry, for males, and for females not personal employment but rather *living with* someone employed in the construction industry.
Tenkanen	1985	Europe	M M	0.54 **1.80**	NS (minimum) **S (maximum)**	[PP 1 through 4 of 6]. Lowest and highest alternative computations reflecting lung cancer risk for men who moved from the country to the city compared to men who lived in the city all along. The authors do not reveal who asked about this.
Coggon	1986	United Kingdom	M M	**2.50** **3.60**	**1.20-5.10 (cooks)** **1.30-10.40 (bakers)**	[From study abstract]. Comments extended below.
Coggon 1986 Comments: Lung cancer risks associated with occupation as professional cook and with specific occupation as professional baker or pastry cook. Case group for this study was exclusive to patients with centrally located lung tumors. David Coggon published a study in 1984, control-matched to represent a presumed rough parity of general lifestyle factors including smoking between cases and controls but lacking specific smoking histories, which suggested an elevated risk of lung cancer for certain occupations including a risk estimate for professional cooks of RR **6.7** within CI **1.8-24.2**. His separate follow-up study of 1986 was undertaken to confirm these results with specific analysis and adjustment for smoking histories.						

Gao	1987	China	F F	1.20 1.40	0.60-2.10 (cooks) 1.10-1.80 (rapeseed oil)	[PP 2, 4 of 6]. Comments extended below.
Gao 1987 Comments: Reports regarding occupation as professional cook and use of rapeseed oil for (home) cooking. The cooking oil result, based on Yu-Tang Gao's observation that 52% of the studied Shanghai lung cancer cases liked rapeseed best compared to just 45% of controls (who generally preferred soybean oil), has gotten an awful lot of play. The minuscule RR level between one and two, which Gao also reports as supported by a relative dose response of up to two to one when compared to soybean oil use amongst women who reported frequently smoky kitchens, has been alternately replicated and disconfirmed in subsequent research but twenty years on you will still hear health gurus discuss the possibility that Gao discovered the "secret" to the "mystery" of Asian female lung cancer incidence in rapeseed oil**:** after all, Western women almost never use rapeseed oil, some Asian women do, so there you are! This ignores quite a lot indeed, including the simple fact that, preferred as it may be amongst half of Shanghai or some other places, rapeseed oil is *not* commonly used in Asia at large, as regional researchers have noted in subsequent studies. Of course logic enters into health cultist blather rarely and never without force. Gao also notes that the result for professional occupation as a cook represented predominantly short-term rather than long-term employment amongst the available sample from this study group.						
Holst	1988	Netherlands	M&F	6.70 0.23	2.20-20.00 (birds) 0.10-0.60 (Vitamin C)	[T 3]. Lung cancer risks referent to ever keeping a pet bird of any type in one's home for a period of 6 months or longer in a lifetime and to higher versus lower Vitamin C intake.
Ives	1988	United States	F F	2.37 6.90	S (family history) 1.88-25.32 (husband's job)	[P 5 of 10, T 3]. Comments extended below.
Ives 1988 Comments: Risk report for ever-smokers and never-smokers collectively, referent to history of lung cancer in a near relative, and risk report for never-smokers specifically, referent to *husband's* occupation in the construction industry. Yikes! Get divorced! It is further explained that none of the cases or controls themselves (all females) ever worked in the construction industry. Janet Ives also textually reports no risk from ETS exposure relative to entire study group but does not qualify this point regarding never-smokers specifically**;** hence this report is not included on our charts of standard ETS studies.						
Wu	1988	United States	F F	NR 3.90	NR (household fuel use) 2.00-7.60 (family history)	[P 3 of 6, T 2]. Collective results reflecting history of using kerosene, coal, and/or wood for home heating and/or cooking and result for history of lung cancer in a near relative. This study was exclusive to patients with the adenocarcinoma lung cancer types.
Mettlin	1989	United States	M&F M&F	0.65 2.14	0.38-1.10 (booze) 1.13-4.07 (milk)	[T 3]. Lung cancer risks associated with heavy consumption of distilled liquor and with heavy consumption of whole milk. No, we never make these things up.

Zahm	1989	United States	M M	1.80 0.90	1.00-3.50 (food service) 0.70-1.00 (farmers)	[T 2]. Occupational risks relevant to employment in the food service industry generically and to farming.
Kalandidi	1990	Greece	F F	0.33 1.09	0.13-0.86 (fruits) 0.44-2.68 (vegetables)	[T 5]. Heavy consumption of fruits and heavy consumption of vegetables.
Wu-Williams	1990	China	F F	1.80 2.10	1.30-2.60 (cooking) 1.30-3.30 (pneumonia)	[T 5, T 6]. Comments extended below.
Wu-Williams 1990 Comments: Result reflecting smokiness in home kitchen (reported by respondent categorically as frequent versus never or rare eye irritation while cooking) relative to all types of cooking and result for personal history of pneumonia. In addition to cooking specifically Wu-Williams also analyzed household fuels and heating practices. While central heating is increasingly prevalent, room-specific heating as well as combination heating and cooking stoves, and wood and coal fuel use, remain common in rural China. Brick floors and walls directly heated by stoves are also traditional and remain fairly common. There were no statistically significant results regarding use of wood and coal generally while the use of any fuel type for direct heating of brick floors, walls, and "kang" (heated brick sleeping platforms) are reflected in results ranging up to 1.4 within CI 1.1-1.9 for heated walls and floors and up to 1.5 within CI 1.1-2.0 for kang.						
Liu, Z.	1991	China	M F	3.36 14.70	1.27-8.88 1.61-134.03	[T 2, T 3]. High versus low exposure to cooking smokes and fumes relative to all types of cooking and all fuel types.
Gardiner	1992	United Kingdom	M&F M&F	1.09 3.53	0.67-1.79 1.56-7.98	[T 1]. Top result is lung cancer risk from ever keeping a pet bird in one's home. Bottom result is for ever keeping pigeons outdoors in a coop. It is reported that no pigeon owners kept their birds indoors. A second test of the pigeon result via alternate comparison and adjustment techniques essentially confirmed the result shown, computing to a similar RR 3.90 within CI 1.20 - 12.62.
Ger (92)	1992	Taiwan	M&F	S	S	[From study abstract]. Comments extended below.
Ger 1992 Comments: Ownership of a pet dove was found to present a statistically significant risk of lung cancer. The full original publication in the Japanese language was not available. The brief available abstract in English does not provide figures. A subsequent publication in English by Luo-Ping Ger, presenting the lung cancer risk results in segregated form according to histologic types rather than generically as in the Japanese language original, but based essentially on the same patient data, is noted below on this chart – as Ger (93) – regarding occupational risk to professional cooks. The 1993 publication ignores the killing potential of pet doves entirely. Very possibly we should all do the same. Doves, incidentally, are white pigeons. With reference also to Gardiner 92, just above, it appears this species' carnage is a worldwide phenomenon.						

Kohlmeier	1992	Germany	M&F	2.14 0.24	1.35-3.40 (birds) 0.07-0.86 (carrots)	[T 2]. Comments extended below.
Kohlmeier 1992 Comments: Risk findings referent to ever keeping a pet bird of any kind and to eating carrots daily as opposed to seldom or never. Lenore Kohlmeier's questionnaire included queries regarding ETS exposure in childhood and adulthood at home and at work. Adulthood exposure produced no significant results. Childhood exposure is shown as significant in one form of analysis but insignificant in another. Results are not included on our charts of standard ETS studies since figures specific to never-smokers are not provided.						
Schoenberg	1992	United States	F F	0.91 8.70	NS (minimum) 1.30-57.80 (maximum)	[Results as cited on PP 5 and 6 of 16 and T 4 of 2001 review publication by William Field from proceedings of a 1992 symposium presented by the New Jersey Department of Health and US Department of Energy]. Comments extended below.
Schoenberg 1992 Comments: Lowest and highest computations from alternative tests reflecting moderate to high versus minimal exposure to radon gas at home. Radon measurements were in most instances monitored by sensors placed for a year's duration in the living areas and basements of cases' and controls' homes but the levels were only spot-measured or simply estimated for about a quarter of the total study population. This is considered the first direct study of residential radon and lung cancer. The NJ DOH suggested immediate remedial action in 1989 based on Janet Schoenberg's preliminary results and radon fear spiked internationally around this time. Although Schoenberg herself pointed with caution to the minuscule representation of four to five cases and but one control subject in her highest exposure categories (note the vast confidence interval accompanying the freak 8.70 RR), while reviewer Field points out additionally that the confidence interval computation is at the substandard 90% level (rather than the arbitrary and likewise dubious 95% level most often used), the scare was on and will never end for true believers.						
Alavanja (93)	1993	United States	F F	2.14 11.38	1.02-4.48 (minimum) 3.77-34.40 (maximum)	[T 7]. Lowest and highest results reflecting moderate to high saturated fat diet versus low fat diet. These results are specific to the adenocarcinoma lung cancer types.
Brownson	1993	United States	F	1.70	0.60-4.50	[T 1]. Occupation in the construction industry. Study was of females only.
Ger (93)	1993	Taiwan	M&F	5.55 10.42	1.39-22.10 (test one) 2.21-49.24 (test two)	[T 8]. Comments extended below.

Ger 1993 Comments: Results refer to lung cancer risk from professional occupation as a cook. Two tests were performed, the first a comparison with hospital-based controls, the second comparing the very same case respondents to community-based controls, and thus creating a risk report of about doubled magnitude. These results refer only to the adenocarcinoma lung cancer types. Ger reports no risk from occupation as a cook for squamous or small-cell lung cancer. This study also includes RR reports for various specifics of cooking (e.g. boiling, frying, use of coal stoves), these being statistically insignificant for the most part, although Ger suggests (with confidence intervals ranging hugely from 1.27 up to 45.61) that use of coal for cooking may be related to squamous and small cell lung cancer, but not in the least to adenocarcinoma. There are numerous ETS figures but none of these relate to never-smokers. As such this report is not included on our charts of standard ETS studies. Luo-Ping Ger's comments on ETS are nonetheless worth noting here for their amusingly extraordinary character. He finds no relationship with any form of lung cancer from living or working with smokers over an entire lifetime: not from smoking mothers, fathers, spouses, children, or co-workers. He does suggest, however, that *there is an approximate doubling of one's lifetime risk, selectively for squamous and small-cell lung cancer, consequent to having friends who smoke as occasional visitors in one's home*. This risk, Ger contends, applies only to squamous and small-cell cancer, not to adenocarcinoma, and he justifies this portion of his belief on the basis that active smoking is not related to adenocarcinoma, as he suggests is generally acknowledged, and as is also shown in his study's distinct analyses of active smoking. The specific problem with ETS emitted by friends who smoke, as opposed to that produced by constant cohabitants and regular workplace companions, Ger hypothesises, is that the occasional social visitors often come over to play mah-jong. In their excitement over the board game they tend to grow loud. This creates fear that neighbors will be disturbed. Therefore the rowdy mah-jong players are likely to close all the doors and windows, creating stuffy conditions, and thus lung cancer. With all his contemplative pipe-puffing, Einstein never thought of this, but Science marches on, with the like of Luo-Ping Ger to guide it.

Liu, Q.	1993	China	M F	**0.14** **0.02**	**0.04-0.51** **0.00-0.21**	[P 5 of 10]. Residence in well-ventilated homes (measured as adequate cross-ventilation of main living area through ordinary doors and windows) versus residence in poorly-ventilated homes. Results for kitchen ventilation specifically are similar at **0.15** within CI **0.05-0.44** for men and **0.06** within CI **0.01-0.32** for women.
Notani	1993	India	M	**4.48**	**1.20-16.90**	[From study abstract]. Occupation as professional cook.
Pershagen	1994	Sweden	M&F M&F	0.80 **2.60**	0.30-2.10 (open window) **1.50-4.40 (none)**	[T 5]. Relative lung cancer risk for persons living in homes with highest measured radon levels (meters were placed for a 3 month period during heating season in bedrooms and living rooms of case and control subjects), with results categorized for those who typically propped a bedroom window open when sleeping, and for those who did not.

Rosenberg	1995	United States	M&F M&F	0.80 1.00	0.60-1.20 (test 1) 0.70-1.40 (test 2)	[Results as cited on T 1 of Moysich 2002: see also below on this chart]. Regular aspirin usage. Test 1 compared lung cancer cases to alternative cancer patient control group, test 2 to non-cancer patient control group.
Alavanja (96)	1996	United States	F F	0.84 0.52	0.65-1.09 0.38-0.71	[T 2]. Top result reflects lung cancer RR from ever keeping a pet bird of any kind indoors. Bottom result reflects RR for patients who personally raised birds of any kind outdoors.
Axelsson	1996	Sweden	M M	1.60 1.89	0.72-3.54 (coffee) 0.94-3.80 (cheese)	[T 2, T 5]. Heavier versus lighter consumption of coffee and of cheese.
Modigh	1996	Sweden	M&F M&F	1.08 0.32	0.69-1.68 (current) 0.11-0.89 (never)	[P 2 of 3]. Lung cancer risk effect from ever owning a pet bird of any kind, for current smokers, and for persons who never smoked.
Wang, T.	1996	China	F F	4.02 3.07	2.38-6.78 (cooking) 1.30-7.26 (family history)	[PP 5, 6 of 6]. Comments extended below.
Wang, T. 1996 Comments: Results relevant to frequent versus infrequent reported exposure to cooking fumes in the home and to family history of cancer. Wang also discusses related matters. His unadjusted results suggested coal use was not a risk factor in itself, but that frequent reported exposure to coal smoke was a factor, however, the negative coal use result was confirmed while the positive coal smoke exposure result was disconfirmed, in adjusted analyses. Use of kang (heated sleeping platforms) was found insignificant in all forms of analysis. Results charted here, from adjusted analyses, confirmed and enlarged similar results in the unadjusted analyses.						
Zheng	1996	United States	F	1.05	0.71-1.55	[T 2]. Heavy consumption of black tea.
Zhou	1996	China	M F	4.50 5.50	2.26-8.97 2.04-12.00	[From study abstract]. Lung cancer risk associated with small versus large size of home kitchen.
Cardenas	1994-1997	United States	M&F M&F	1.20 1.40	0.90-1.50 (drop-out) 1.00-2.10 (non-white)	[1994 PP 89, 88 of 158]. Comments extended below.

Cardenas 1994-1997 Comments: Paper was prepared as a doctoral candidate's submission in 1994 and published in a shorter edition in 1997. Lung cancer risks for persons who did not graduate from high school versus those who did and for non-whites versus whites. Victor Cardenas also reports that his drop-out population reported above-average ETS exposure overall but below-average ETS exposure from a spouse specifically while non-whites reported below-average ETS exposure overall with about average exposure from their spouses specifically. Both education and race were adjusted for in Cardenas's ETS analyses, based on consensus opinions, despite that Cardenas notes: "schooling was not a meaningful confounder based upon the data at hand." All respondents were never-smokers. ETS-related results from Cardenas's research appear on tables in following section of this paper.						
De Stefani	1997	Uruguay	M M	1.09 **3.18**	0.68-1.72 (ice cream) **2.05-4.94 (rice pudding)**	[T 4]. Frequent versus infrequent consumption of the indicated delectables. De Stefani also assesses the wages of a general liking for dessert at RR **2.52** within CI **1.54-4.12**. You didn't think they'd overlook dessert did you?
Hebert	1997	United States	M	NR	NR	[From study abstract]. Comments extended below.
Hebert 1997 Comments: No heightened risk of lung cancer reported for tall versus short men. This study was of physicians and included no women. It also reports a risk for *all* cancer of a whopping and frighteningly fully 95% significant RR 1.21 within CI 1.05-1.39 for tall doctors. Our suggestion to begin smoking at least enough to stunt your growth still holds although US male physicians of a selectively daring nature may choose to ignore this advice.						
Ko (97)	1997	Taiwan	F	**8.30**	**3.10-22.7**	[T 6]. Comments extended below.
Ko 1997 Comments: Lack of exhaust fan above home kitchen stove relative to all types of cooking. Ying-Chin Ko's 1997 study results also show a sharp trend over time in Taiwan away from wood and coal fuels as being of marginal consequence, and a concurrent widespread switch from lard to vegetable oil for cooking of no consequence to lung cancer risk, while the lack of an exhaust fan is shown as a consistently statistically significant deleterious factor throughout all sub-group and time-based analyses. For analyses based on specific frying practices the presence of an exhaust fan neutralized all risks (ranging up to RR **13.3** within CI **3.4-52.4** for stir-frying) reported for cooking without an exhaust fan. Ko derived that, "Our interaction and multivariate analysis have indicated that the use of fume extractors in the kitchen, that were wider spread in Taiwan by 1970 but not on mainland China, explained the majority of differences between cases and controls, even if cooking fuels, oils and techniques (stir frying, frying, deep frying) were covered in the multivariate logistic regression model ...".						
Swanson	1997	United States	F F	1.46 **6.27**	0.86-2.51 (minimum) **2.68-14.60 (maximum)**	[T 1 within letters section of the *Journal of the National Cancer Institute*]. Lowest and highest results reflecting moderate to high saturated fat diet versus low fat diet. Figures reported by Swanson reflect lung cancer generically and are revised from data originally collected for the Alavanja 1993 study (see also above on this chart.)

van Loon	1997	Netherlands	M	0.53	0.34-0.84	[From study abstract]. Relative risk of lung cancer for men with high level of education compared to men with low level of education.
Beeson	1998	United States	M F	10.18 NR	2.44-42.45 NR	[P 6 of 10]. Risk from ambient air pollution measured as long-term residence amidst high ozone levels in high-density urban population centers within California between 1966-1992. Californian legislation mandating sex change operations may be in the offing.
Carpenter	1998	United States	M&F M&F	1.87 0.86	1.02-3.42 (booze) 0.44-1.75 (beer)	[T 2]. Comments extended below.
Carpenter 1998 Comments: Lung cancer risks associated with heavy consumption of distilled liquor and with heavy consumption of beer. We sympathize with strictly liquorish Mettlin (1989 study: see above) fans while noting that drunkards as a general class may still choose to rejoice. Curtis Mettlin may sanctify booze while Catherine Carpenter doesn't, but Carpenter once and for all puts the classic formula of shots *followed by* beers on a sure scientific basis, so simply consider the scholarly literature and order by the brace.						
Jöckel (98)	1998	Germany	M M	1.27 1.12	0.98-1.65 (construction) 0.85-1.48 (farmers)	[T 5, T 6]. Comments extended below.
Jöckel 1998 Comments: Results for occupational lung cancer risks. Karl-Heinz Jöckel also gives very limited analysis of female occupation with only one computation specific to females, RR 1.79 within CI 0.94-3.42, for a category of "suspected risk occupations" which is generally characterized or suggested as blue collar employment around smelly things but nowhere specifically delineated in the report.						
Matos	1998	Argentina	M M	0.90 1.50	0.30-2.50 (food service) 0.90-2.60 (farmers)	[T 4, T 3]. Occupational lung cancer risks.
Morabia	1998	United States	M F	1.28 1.17	0.88-1.86 0.83-1.64	[T 1]. Lung cancer risk from ever owning a pet parakeet, canary, finch, or parrot.
Nyberg	1998	Sweden	M&F M&F	0.50 1.27	0.24-1.06 (coffee) 0.73-2.21 (tea)	[T 3]. Heavy consumption of coffee and of tea (all types.)
Shen	1998	China	F F	2.45 4.36	1.06-5.66 (cooking) 1.03-23.85 (family history)	[T 6]. Results relevant to reported exposure to cooking fumes (all cooking methods) in the home and to family history of cancer. Fuel type was considered and was not found to be a significant factor. This study was of patients with the adenocarcinoma lung cancer types only.

Speizer	1999	United States	F F	1.35 1.10	1.00-1.80 (Vitamin C) 0.80-1.50 (saturated fat)	[T 4, T 3]. Heavy consumption of Vitamin C and heavy consumption of dietary saturated fat.
Yong	1999	United States	M&F M&F	0.48 1.16	0.28-0.82 (current) 0.56-2.39 (never/former)	[T 5]. Heavy consumption of both fruits and vegetables, results stratified for current smokers, and for never smokers and former smokers collectively.
Brüske-Hohlfeld	2000	Germany	M M	NR 1.31	NR (food service) 1.13-1.51 (farmers)	[P 5 of 12, T 4]. Occupational lung cancer risks.
Feskanich	2000	United States	M&F M&F	0.99 0.82	0.91-1.08 (generic) 0.59-1.16 (carrots)	[T 1, P 5 of 12]. Regular consumption of fruits and vegetables collectively and of carrots specifically.
Field	2000	United States	F F	1.34 1.79	0.81-2.22 (low exposure) 0.99-3.26 (high exposure)	[T 4]. Relative risk results for lowest and highest exposure categories, compared to minimal exposure category, relevant to radon gas exposure, collectively measured or estimated for home, area of residence, and workplace.
Ko (00)	2000	Taiwan	F F	3.20 12.20	1.40-7.30 (minimum) 4.50-33.10 (maximum)	[T 5]. Comments extended below.
Ko 2000 Comments: Lowest and highest computations relative to specific risk of preparing fried foods (all frying methods) without an exhaust fan above home kitchen stove. Results of this separate follow-up study are similar to those of Ko's 1997 study which also focussed on cooking. For this study residential heating or cooking fuel (wood, coal, gas or other) and type of cooking oil (lard or various vegetable oils) were found to be insignificant factors, while independently of specific cooking practice analyses, occupation as a professional cook was here considered and found to be a significant factor. In concluding remarks the author reflects that cooking risk and particularly the frying peril have continued and may persist despite a widely increased use of kitchen exhaust fans. Many Taiwanese women have switched from lard to Western-style processed vegetable oils for frying because of cholesterol scares but Ko opines that the good old lard fumes were probably healthier. He bases this pronouncement on his belief that lard contains fewer carcinogens. Statistical data from the professor's two cooking studies regarding use of particular cooking fats is notably neutral as a matter of fact but if you wanted a clinical reason to indulge in lardy fried foods there you are.						
Le Marchand	2000	United States	M&F M&F	1.10 0.90	0.70-1.80 (black tea) 0.50-1.60 (green tea)	[T 2]. Heavy consumption of black and of green teas.

Nyberg	2000	Sweden	M M	0.90 **1.60**	0.68-1.19 (minimum) **1.07-2.39 (maximum)**	[T 3, T 4]. Minimum and maximum computations reflecting moderate to high versus low exposure to ambient air pollution (based on analysis of historic nitrogen dioxide and sulfur dioxide levels) amongst males with long-term residence in various areas around Stockholm.
Pisa	2001	Italy	M&F M&F	2.00 1.00	1.00-3.90 (low exposure) 0.30-3.10 (high exposure)	[T 3]. Self-reversing relative risk results for lowest and highest exposure categories, compared to minimal exposure category, referent to radon gas levels in the home (measured for one year with meters placed in bedrooms of the case and control subjects.)
Lagarde	2001	Sweden	M&F M&F	1.08 1.44	0.79-1.47 (low exposure) 1.00-2.06 (high exposure)	[T 5]. Comments extended below.
Lagarde 2001 Comments: Relative risk results for lowest and highest exposure categories, compared to minimal exposure category, referent to radon gas levels in the home (alternately measured with meters placed for three months' time during heating seasons in bedrooms and living rooms of the case and control subjects, or estimated by extrapolation from regional data, where specific measurement was impracticable.) Frédéric Lagarde's questionnaire included a query regarding ETS exposure at home in adulthood, and he reports a heightened radon-related risk (RR 2.10 within CI 1.21-3.65 for the highest radon exposure category) amongst a subset of patients who reported ETS exposure, although overall computations of both ETS and radon exposure for this study produce uniformly insignificant results. Lagarde gives no overall computations, dividing all results between study population segments, or quartiles of exposure. He does not report any distinct ETS risk estimate at all but this is calculated from his cell counts as RR 1.15 within CI 0.93-1.43 for our charts of standard ETS studies. An overall computation of higher versus lower radon exposure for the ETS exposure group specifically (86/93/264/347) gives a result of RR 1.22 within CI 0.87-1.70. For the entire study group higher versus lower radon exposure (186/250/729/920) gives a result of RR 0.94 within CI 0.76-1.16. The Lagarde patient population was stitched together with data from six previous study groups including both re-use of data as well as re-interviews of subjects or often of surrogates. The entire selected study population was of reported never-smokers (a reference to "current smokers" on Table Five is a misprint for "current study.")						
Mao	2001	Canada	M F	**0.60** **0.60**	**0.50-0.70** **0.50-0.80**	[P 1 of 9]. Relative risk of lung cancer for persons with high level of education compared to persons with low level of education.
Nagano	2001	Japan	M&F	0.79	0.59-1.10	[T 4]. Heavy consumption of green tea.

Hu	2002	Canada	F F	**2.40** 1.00	**1.30-4.40** (cooking) 0.50-1.90 (milk)	[T 2]. Risk reports for frequent versus infrequent cooking (all methods) with shortening (all types) and for heavy consumption of milk. Study was of never-smoking cases from eight provinces of Canada. Specific heating and cooking fuels were not assessed.
Jöckel (02)	2002	Germany	M&F M&F	0.85 3.82	0.53-1.35 (all patients) 0.98-14.92 (young patients)	[T 2]. Lung cancer risk from ever owning a pet bird of any kind, computed for all patients included in the study, and more specifically for those who developed lung cancer at a young age (55 or younger.)
Kreuzer	2002	Germany	F F	**0.65** **0.34**	**0.44-0.95** (milk) **0.21-0.55** (cheese)	[T 4]. Heavy milk and cheese consumption.
Moysich	2002	United States	M F	**0.62** **0.52**	**0.43-0.90** **0.29-0.95**	[T 3]. Regular aspirin usage.
Sasco	2002	Morocco	M&F M&F	0.74 1.48	0.17-3.14 (minimum) 0.44-4.91 (maximum)	[T 3, T 2]. Risk from cooking or heating with coal as computed by alternate methods of covariate analysis. Both methods adjusted for personal smoking. Sasco notes that the smaller RR figure includes adjustment for inferior kitchen ventilation reflected in the larger RR figure. This study also reports no significant risk from ETS from any source at home or at work, according to both methods of analysis, but does not report distinct results for never-smokers, and as such is not included on our charts of standard ETS studies.

Zatloukal	2003	Czechoslovakia	F F	0.66 0.61	0.43-1.01 (adeno) 0.42-0.89 (others)	[T 6]. Relative risk of lung cancer amongst women who reported frequent lifelong mental or physical pain during menstruation, distinguished as specific risks of the adenocarcinoma types, and of the three other major types. Perhaps we can be thankful the ladies do not suffer for nothing.
Richiardi	2004	Italy	M F	5.70 2.60	1.40-24.00 (bakers) 0.50-13.00 (food service)	[PP 1, 5 of 10]. Occupational lung cancer risks.
Schabath	2004	United States	F	0.66	0.51-0.89	[P 1 of 11]. Effect of hormone replacement therapy (for menopause) on risk of lung cancer.
Behera	2005	India	F F	1,827.535 2,683.463	0.000-infinity (minimum) 0.000-infinity (maximum)	[T 2]. Comments extended below.
Behera 2005 Comments: *No* we do not "make up" any of these**:** maybe they do but we *don't*. Charted are minimum and maximum risk estimates reflecting use of kerosene-fuelled stoves versus stoves fuelled by liquefied petroleum *just as reported* on Table Two of this peer-reviewed study published most attractively (and in color!) by the Chandigarh Department of Pulmonary Medicine, Postgraduate Institute of Medicine, in the *Journal of the Association of Physicians of India*. Digambar Behera offers *no explanation whatsoever* for these thoroughly amusing kerosene conclusions but with all of 67 cases and 46 controls in this study there clearly must have been a quirk disparity in the 2x2 table which apparently became compounded by the increasingly prevalent problem of epidemiological psychosis. Incidentally kerosene fuel use has been and remains common in many areas both East and West and is not generally considered of particular risk, nor specifically as significantly risky compared to the other fuels Behera studies, named as liquefied petroleum, wood, cow-dung cake, agricultural waste, coal, "et cetera," and "mixed." Professor Behera, MD, also comments that adequate flues and household ventilation ameliorate risks from all household combustion though he makes no analysis on these points. The distinguished author additionally concludes from his data (without providing specific figures) that ETS exposure is not significantly related to lung cancer in non-smokers, but he does not define non-smokers specifically as never-smokers, while also choosing a control group composed entirely of respiratory disease patients**;** thus no result from this study is included on our charts of standard ETS studies.						
Chiu	2005	Hong Kong	F F	NR 2.52	NR (construction) 1.19-5.33 (housewife)	[From study abstract]. Risks referent to occupation as a construction worker and as a housewife.

Lissowska	2005	Europe	M&F	1.22	1.04-1.44	[T 3]. Lung cancer risk for persons who ever used wood and/or coal for cooking and/or heating versus those who never did. Alternative computations for population segments, and types and durations of wood or coal use, appear on three charts and range from 0.66 within CI 0.36-1.21 up to 2.23 within CI 1.45-3.44.
Liu, Y.	2005	Japan	F	2.40	1.07-5.40	[From study abstract]. Effect of hormone replacement therapy (for menopause) on risk of lung cancer.
Edwards	2006	United Kingdom	F F	1.38 2.13	0.59-3.26 (minimum) 1.34-3.38 (maximum)	[T 6]. Lowest and highest alternative risk computations regarding ambient air pollution, relative to long-term residence within 3 miles of heavy industry in Teesside, England.
Gorlova	2006	United States	M&F M&F	1.06 0.57	0.67-1.68 (HRT) 0.35-0.92 (hay fever)	[T 3]. Result reflecting history of hormone replacement therapy for menopause and result for personal history of hay fever. Gesundheit!
Neuberger	2006	United States	F F	NR NR	NR (food service) NR (construction)	[Sections 2, 3 and 4 of text. Commissioned by the US National Institutes of Health, this study is available free to the public.] Comments extended below.
Neuberger 2006 comments: John Neuberger performed distinct analyses on exposure to asbestos, specific industrial chemicals, and separately and more generally on "high-risk" occupations and industries. The high-risk occupational and industrial analyses (both unadjusted and adjusted) produced no risk indications, and are of pertinent interest regarding jobs we have focussed on, these being given noted consideration, but amidst a broader classification. We chart results here as a general finding of no risk regarding food service and construction work. Neuberger describes his high-risk occupations and industries as follows: "Occupations were classified as high risk if they included exposure to dusts, particulates, volatile organic compounds, or cooking fumes. These categories included bartender, metal worker, welder, cook, factory worker, machine operator, painter, gas station attendant, carpenter, waitress, and truck driver. High-risk industries included dry cleaning, restaurant, plastics manufacturing, ordnance plant, welding shop, construction, bar, café, foundry, battery factory, paint contractor, and trucking."						
Yu	2006	Hong Kong	F F	1.23 34.00	0.73-2.07 (minimum) 7.16-161.39 (maximum)	[T 3]. Comments extended below.

Yu 2006 Comments: Exposure to cooking smokes and fumes, lowest and highest risk computations reflecting frequent to very frequent versus infrequent preparation specifically of fried foods (all frying methods) in the home kitchen. Ignatius Yu's study was undertaken in metropolitan Hong Kong. Wood and kerosene fuels were sometimes used by study participants but coal use was not reported. Specific heating and cooking fuel use as well as specific animal or vegetable shortening use were investigated and reflected in several forms of multivariate analysis though these were not found to be significant factors in themselves.						
Khurana	2007	United States	M&F M&F	**2.32** **0.23**	**2.05-2.63** (low duration) **0.20-0.26** (high duration)	[T 2]. Self-reversing results pertinent to effect of statin drug regimen (for cholesterol control) on risk of lung cancer.
Ramanakumar	2007	Canada	M F	0.70 **2.50**	0.50-1.00 **1.50-3.60**	[T 3]. Use of wood, coal, and/or natural gas for residential heating and cooking.
Setoguchi	2007	United States	M&F M&F	1.18 1.02	0.72-1.92 (low duration) 0.59-1.74 (high duration)	[T 4]. Effect of statin drug regimen (for cholesterol control) on risk of lung cancer.
Veglia	2007	Europe	M&F M&F	**1.53** **1.45**	**1.10-2.10** (food service) **1.10-1.90** (farmers)	[T 2]. Occupational lung cancer risks.
Li	2008	China	F F	**2.51** NR	**1.80-3.51** (cooking oil) NR (fuel smoke)	[T 4, P 3 of 7]. Comments extended below.
Li 2008 Comments: Top result above reflects subjects who reported kitchens filled with oily cooking smoke "sometimes" or "frequently" as opposed to "seldom" or "never." Mingchuan Li states no risk for subjects with fuel smoke exposure, considered as those who reported using coal stoves or kang without flues. All cases were never-smokers. Li also reports no risk from passive smoking exposure at any age or from any source, at home, or at work. This study is not included on our charts of standard ETS studies. It uses a control group composed of patients with various lung diseases.						
Shen	2008	China	M&F	**2.39**	**1.28-4.48**	[From study abstract]. Comments extended below.
Shen 2008 Comments: Lung cancer risk resulting from consumption of green vegetables. Shen generously exonerates corn, buckwheat, radishes, peppers, melons, pickled vegetables, and salted meats from lung cancer culpability. He points out that coal use is common in the rural area where his study took place, and hypothesises that coal smoke may have a selective affinity for collecting onto green vegetables, as an explanation for their relative toxicity.						
Thompson	2008	United States	M&F	2.50 0.47	0.47-13.46 (test one) 0.11-2.04 (test two)	[T 3]. Comments extended below.

Thompson 2008 Comments: Both results shown reflect Richard Thompson's highest categories of home radon exposure, compared to lowest categories, according to two different models of measurement. Radon meters were placed in most commonly used living areas and in bedrooms of subjects' homes for a year. In general, risk of lung cancer *declined* with increasing exposure to radon, although most results, including those for highest exposures shown here, were statistically insignificant. Of the eleven exposure-graded results given for the two tests, the only two "statistically significant" results (**0.31** within CI **0.13-0.73** and **0.35** within CI **0.13-0.99**) reflected mid-range radon exposure.						
Liang	2009	China	F	**1.75** 1.64	**1.21-2.52 (cooking)** 0.93-2.86 (life crisis)	[T 2.] Result for exposure to cooking fumes (yes/no ten or more years' exposure) and for mental trauma in past 20 years described as "(e.g. failure of love affair or marriage, unemployment, death of relatives)".
Wang, X.	2009	Hong Kong	F F	**4.16** **5.19**	**2.06-8.41 (nonsmokers)** **1.64-16.40 (smokers)**	[T 3]. Results for women who most frequently stir-fried foods versus those who stir-fried most infrequently, stratified as pertinent to non-smokers and to smokers.
Lopez-Cima	2011	Spain	M&F M&F	1.33 1.59	0.86-2.06 (pollution) 1.00-2.51 (history)	[T 3, T 2. Results for urban versus rural residency considered to reflect exposure to air pollution, and result for family history of lung cancer.

3. Blood Libel

A definition of blood libel from Wikipedia online:

> Blood libel (also blood accusation) is a false accusation or claim that religious minorities, usually Jews, murder children to use their blood in certain aspects of their religious rituals and holidays. Historically, these claims—alongside those of well poisoning and host desecration—have been a major theme in European persecution of Jews.
>
> Blood libels typically allege that Jews require human blood for the baking of matzos for Passover, although this element was absent in the earliest cases that claimed (the contemporary) Jews reenacted the crucifixion. The accusations often assert that the blood of Christian children is especially coveted, and, historically, blood libel claims have been made to account for otherwise unexplained deaths of children.

A sacred tenet of the healthist cult is that, even if loathsome nicotine worshippers do not always kill themselves, they always and everywhere kill their families and neighbors, and especially, their own and their neighbors' children.

Those who never smoke, or who redeem themselves by quitting the evil practice before middle age, of course have the officially certified "zero" risk of lung cancer. They may have smoked heavily, and for years, but the risk of that is *more than* eradicable.

Yet one's sanctified standing amongst the smoke-free risk-free elite can be desecrated and destroyed by the Evil Ones Without. Did the risk-free ever have a family member, who purported to love them, but smoked in their presence? Why then, they have lost their sanctification!

They have been assaulted, dethroned, and in result shall forevermore suffer a positively ineradicable risk of lung cancer: there is no expiration date, not in a lifetime, not in eternity, for the foul taint of any association, of any duration and no matter how remotely in the past, with smokers. Loved ones? Dear friends and neighbors? Nay, filthy smokers! Killers! Bloody murderers!

It all gets taught to little children in school today, and has been, for decades now. The children must know that their parents, and relatives, and smiling neighbors who smoke are

fiends, threats, intolerable filth. We all must be made aware. The stinking others must be feared. They must be disgraced. They must be hated: all the time, everywhere, by everyone, under the force of righteous law. The vermin must be eradicated from our midst.

Secondhand smoke kills. There is no safe level of secondhand smoke. If there is any secondhand smoke anywhere it is by definition killing, potentially suddenly, immediately. Smokers kill. Smokers are sub-human filth. Even when not smoking they contaminate everyone and everything around them with their inherent filth. These are the most sacred mantras of all.

A couple of standard doctrinal chantings, from Sanjay Gupta, fresh upon his "zero risk" incantations, and also from an anti-smoker activist, were droned during the CNN broadcast of 2006 from which we have previously quoted.

> COLLINS: We know that people have questions out there, John. So we want to go ahead and get our first caller. Now, this is Frank in New Jersey. Frank, I believe you have a question to Dr. Sanjay Gupta?
>
> CALLER: Yes. Thank you very much for taking my call. I'm a 55-year-old person who has never smoked a day in his life, yet my paternal grandfather and father both died of lung cancer. Now, both were heavy smokers. And I'd like to know whether or not I run a risk, a genetic risk for developing this disease and whether or not there are any preemptive tests that I can undergo to determine an earlier diagnosis.
>
> GUPTA: Yes. You know, Frank, thanks for your question. A couple of things to think about. One is that I don't know how long you lived in the same household as your father when he was a heavy smoker, how much secondhand smoke you were exposed to, because that's something to consider as well.
>
> The genetic risk is there, although somewhat small, I think. You might be someone who qualifies for some – some earlier screening or someone who just pays more attention to the things Dr. Janne was just talking about, if you had a persisting cough, for example, or if you had some swollen lymph nodes, or something that seemed unusual to you, hoarseness of the voice that wouldn't go away. You might want to get that checked out earlier, rather than later.

The tests that we're talking about here, the most definitive test for you, would be probably a CAT scan of your lungs to actually see if there was anything unusual or abnormal there.

ROBERTS: Bonnie from Pennsylvania is on the line with us now. Bonnie, what's on your mind?

CALLER: Hi. I'm a 63-year-old nonsmoker with advanced lung cancer. And now that more emphasis will be placed on the fact that Dana Reeve was a nonsmoker and that you can get cancer, possibly get cancer from second-hand smoke, I would like to know what we can do to make smokers more aware of the seriousness of second-hand smoke.

ROBERTS: Susan Mantel, that sounds like that's one that's right up your alley. Do you want to take that?

SUSAN MANTEL, JOAN'S LEGACY LUNG CANCER FOUNDATION: Sure. Well, Bonnie, I think that there are two parts to your question, from our point of view.

One is making smokers more aware. A lot of states have put initiatives into place trying to establish clean air acts. And New York is one that I'm lucky enough to live in where that's been taken very seriously. Illinois has just put that into place in some places. Chicago in a thorough way.

So there's more and more of a push. Actually, the Lung Cancer Alliance has a great map where you can see the states that are moving on that and also how you can get involved with doing that.

And that said, you know, I think it's – it's up to all of us to make sure that our own environments are as clean as possible, and then know at some point, you know, it's about taking care of ourselves as patients whether we smoked or not.

COLLINS: Susan, thank you so much for that.

We must be clean. We must take care of ourselves as patients. We are vulnerable. We are being killed by the unclean others: the danger without. Make them more aware: tax them

to impoverishment: banish them, from employment, from housing, from any and all social milieux, indoors, outdoors too. Allow them not even any segregated quarters. Grant them no ghetto: make them aware: their very existence is intolerable.

They kill. They don't deserve to live. The attitude of orthodox healthists toward smokers was explained succinctly by the licensed nurse and anti-smoker activist Jane DeVille-Almond on British radio in June of 2009.

Despite their enormous tax burden, and lifetime contributions of health insurance premiums, Jane expressed that smokers should not receive care under the British National Health Service unless they could prove they had quit smoking.

The nurse cited the case of a smoker who was treated for a heart condition but had not quit smoking and declared that he should not receive further care unless he paid for it out of his pocket.

Her interviewer expressed that most people had enough trouble paying premiums over all of their working lives, and could not afford additionally to pay out of pocket for services, but caring nurse Jane was unmoved by this.

The interviewer pressed her, insisting, "Then what, if he can't afford it, what?" She replied calmly, "Well, then, they just have to die."

Of course, this is just another expression of the steely "quit or die" attitude, which has sustained amongst empowered fanatics over decades now, and also explains the morally reprehensible stonewalling against less hazardous cigarettes by the healthist elite, which we have previously discussed.

If the risk of smoking regularly and plentifully for years can be undone by quitting before middle age why would anyone think for a moment that having ever lived or worked with somebody else who smoked produces a risk that can never be undone? Why, for that matter, would anyone think that tiny puffs of smoke in the air present any risk at all?

The healthists do of course believe in all of this plain nonsense, because they are robustly skilled in groupthink and doublethink, and because such madly contorted belief is strictly necessary to the progress of their aim to eliminate smokers.

Propagating deluded and hysterical belief about ETS is necessary to recruiting the public

at large into agreement that smokers are absolutely worthless and intolerable so just have to die.

Many amongst the public swallow all the propaganda. Many of these might vehemently deny sharing the hateful sentiments of the cult while nevertheless and cheerfully supporting public policies advancing the smoker pogrom.

ETS "lifestyle epidemiology" studies began appearing in 1981. The stated aim of socially ostracizing smokers dated back a decade from this. In 1971 US Surgeon General Jesse Steinfeld wrote: "Nonsmokers have as much right to clean air and wholesome air as smokers have to their so-called right to smoke, which I would redefine as a 'right to pollute.' It is high time to ban smoking from all confined public spaces such as restaurants, theatres, airplanes, trains and buses. It is time that we reinterpret the Bill of Rights for the nonsmokers as well as the smoker."

The trouble was that smokers and nonsmokers got along well and did not want smoking banned. So few bans went into place. The thorny problems of general amity and social cohesion, operating under a widely sane perspective amongst the public, were addressed at the 1975 World Conference on Smoking and Health of the World Health Organization, held in New York city, under Chairman Sir George Godber, a British physician and health official.

A policy of "fostering the perception that secondhand smoke is unhealthy for nonsmokers" (as described by Doctor Gary L. Huber, et al., in *Consumers' Research*, July 1991) was initiated by Godber at the conference, with a specific aim "to emphasize that active cigarette smokers injure those around them, including their families and, especially, any infants that might be exposed involuntarily to ETS."

There was virtually no dissent amongst attendees at the 1975 conference as to the advisability of total dedication to smoking eradication, by any means necessary, or as to the utter worthlessness of persons who smoked. As Doctor Godber said:

> I imagine that most of us here know full well that our target must be, in the long-term, the elimination of cigarette smoking. ... We may not have eliminated cigarette smoking completely by the end of this century, but we ought to have reached a position where a relatively few addicts still use cigarettes, but only in private at most in the company of consenting adults.

> ... First, I think we must ask ourselves whether our society is one in which the major influences exercised on public opinion are such as would convey the impression that smoking is a dirty, anti-social practice, spoiling the enjoyment of youth and accelerating the onset of the deterioration of age.
>
> ... Need there really be any difficulty about prohibiting smoking in more public places? The nicotine addicts would be petulant for a while, but why should we accord them any right to make the innocent suffer?

There was no evidence in the nineteen-seventies, and there still is not, that "nicotine addicts" were making the "innocent" suffer. There was no perception either amongst the public that smokers injured anyone. Bars and restaurants certainly could, for example, have segregated or banned smoking, and individuals could certainly have refused to allow smoking friends to light up in their homes, but ashtrays were considered social necessities in most non-smokers' homes, and smoking was commonplace nearly everywhere people met, by common choice. This had been true, in most places and periods, for centuries across the world.

At the 1978 World Conference Godber further defined smoking as an infestation, to be wiped out, like head lice. In order to achieve the goal of creating such hateful perception of smoking and smokers the lifestyle epidemiologists, ever ready to "prove" anything they liked, soon began producing their usual quality of "scientific research" to the end of defining the verminous nature of smokers.

To the anti-smoking crusaders the implausibility of harm from wisps of tobacco smoke, in a world where combustion is and always has been common, and to which all animals have adapted, was simply an inconvenient truth. The point was to damn the truth in pursuit of the cause. The fanatics are highly adept at believing untruth, themselves, and the masses were sure to be malleable to "authoritative" suasion. As Christopher Snowdon has noted in his book *Velvet Glove, Iron Fist: A History of Anti-smoking*:

> Several of the biggest names in smoking research believed the threat from Environmental Tobacco Smoke (ETS), as it was known, to be illusory from the outset. Cuyler Hammond pointed out that even as GASP [Group to Alleviate Smoking Pollution] and ASH [Action on Smoking and Health] were promoting the passive smoking theory in the mid-1970s, there was "no shred of evidence" to support it. Ernst Wynder, who had spent twenty-five years researching smoking and health, told a cancer conference in 1975 that "passive smoking can

> provoke tears or can otherwise be disagreeable, but it has no influence on health [because] the doses are too small." Even some anti-smoking groups were sceptical. In 1973 ASH (UK) set up a committee to look at the evidence and concluded that "there is virtually no risk to the healthy nonsmoker, apart from exceptional exposure to tobacco smoke in an unventilated room or a closed car."
>
> Plausible or not, it was a theory with obvious and immediate appeal to the anti-smoking lobby and they were quick to spread it. John Banzhaf [founder of ASH] wrote in 1972: "I have little understanding for those men and women whose nasty nervous habit forces me to breathe carbon monoxide. Quite frankly – as well as literally – they make me sick." In the same year, *Reader's Digest* printed an article by one Max Wiener entitled "Nonsmokers Arise!" in which he opined that "smoking should be confined to consenting adults in private" because of the threat to nonsmokers' health.

Researcher Michael J. McFadden has described the hateful anti-smoking crusade in his book *Dissecting Antismokers' Brains*.

> While no one is suggesting that smokers are about to be rounded up and hauled off to crematoria, the intensity of the language used by some Crusaders against smokers is not far from that which was used against the Jews in the early days of Nazism. Remember, discrimination, ghettoization, and the building of hate came long before the ovens. And part of what buttressed that building of hate was the "findings" of Nazi scientists that purported to show the inferiority and depravity of the "lower races."
>
> Hate, by its very nature, tends to be irrational in its roots and expression; but when those who hate feel that they have objective and scientific basis for their feelings, the extremes to which it will grow are hard to predict.

> The top half of Figure 18 is an image [reproduced here at left] sent to one of the largest smokers' rights activist groups in the world: FORCES [Fight Ordinances and Restrictions To Control or Eliminate Smoking] International. The image seems to have been downloaded from a neo-Nazi site, altered, and then sent as an attached file to one of the FORCES mailboxes with the address and other sender

> identification electronically scrubbed out. The message attached to the image referred to smokers as being subhuman and an infestation ... language quite similar to that of previously cited British Health Minister Sir George Godber when he compared smoking in the home to an infestation of head lice ...

Vincent-Riccardo DiPierri describes the matter in psychological terms, assessing that anti-tobacco "authorities" have a "superiority syndrome (SS)" which produces crankiness, haughtiness, and an absolute insistence on their own ill-founded opinions, and their own rights, to the absolute exclusion of the opinions and rights of others, these others being perceived as inferiors, and as threats. The authorities impose this deluded and tyrannical perspective onto the public at large along with "environmental somatization syndrome (ESS)", in effect, an acute hypochondria which can perceive a common and harmless element of the environment – in this case, under suasion of debased authorities, the element of ETS – as terrifying and threatening. He writes regarding the ETS propaganda campaign:

> [H]ighly questionable information, manufactured by an incompetent, unstable mentality, can wreak havoc when it is propagated under the pretense of scientific credibility. As an SS and ESS pandemic spreads, the irrational fear and superiority become more acute and the demands for protection become more bizarre and socially dangerous.
>
> There are many nonsmokers who will happily sit around an open indoor-fire, or in a restaurant that obviously has an operating kitchen (i.e., cooking-smoke). Although ambient smoke can be quite visible in such settings, it produces no troubling. Yet, let a lit cigarette appear and panic and an eradication procedure ensue, and protected by the superiority syndrome. This reflects the deluded, superstitious, belief that tobacco smoke is somehow *very* different from these other sources of smoke, magically endowed with all manner of dangerous propensities: In typical settings none of this smoke, from whatever source, poses a danger to a normative range of functioning. These deluded beliefs are the result of a relentless healthist propaganda, i.e., iatrogenic.

The anti-smoking establishment has produced bizarre "scientific research" such as measuring smoke constituents in a room, which really require no measurement as they could be seen, and then defining the presence of smoke in the air as potentially fatal to humans, based on nothing more than the researchers' own deluded and panicked belief system.

The "scientists" have likewise measured blood pressure of persons in smoky rooms, and defined minute fluctuations over hours, these being consistent with rising and sitting, or laughing, or drinking a glass of water, as "caused by" ETS, and as signalling potential for a heart attack. The editors of medical journals, having typically the same neurotic belief system as do the fanatical researchers, publish such utter nonsense from time to time.

A particularly lunatic researcher along these unconventional lines, named James Repace, is a great darling of the fanatics. Repace, who has training in physics, worked as an anti-smoker activist while also working at the US Environmental Protection Agency decades ago. He now advertises himself as a "secondhand smoke consultant" and has a lucrative career advising/terrorizing government agencies and businesses toward harassment and ostracism of smokers.

By far the most favored form of pseudo-scientific nonsense, however, has long been, and remains, the debased practice of lifestyle epidemiology. It is primarily on this body of work that anti-smokers base their fear and hate campaign. We will present shortly a comprehensive listing of lifestyle epidemiology studies of the supposed effect of ETS on lung cancer risk in never smokers, from the beginnings of research, to the time of data collection for this paper, February of 2012.

The best-known previous compilations of ETS relative to lung cancer include the 1992 US Environmental Protection Agency report on ETS, "Respiratory Health Effects of Passive Smoking: Lung Cancer and Other Disorders", and statistician Allan Hackshaw's 1997 *British Medical Journal* article "The Accumulated Evidence on Lung Cancer and Environmental Tobacco Smoke". These compilations created a standard of listing studies relating never smokers' exposure to ETS and excluding studies which reported on ever smokers.

However, in that compilers of both collections took a clearly prejudicial perspective toward finding ETS to be "a cause of lung cancer", they did include a few studies they favored, which were not truly limited to never smoker lung cancer cases. This will be further described in due course. For our tables here we generally adhere to listing only the never smoker studies but also include all of the studies used by EPA and Hackshaw in their "meta-analyses" (pooling of existing studies' data as opposed to novel research).

The 1992 EPA report, which the aforementioned James Repace helped considerably to inspire, was a particularly shoddy piece of work. The tobacco industry sued against it and in 1998 a US federal judge officially vacated the EPA's findings on ETS and lung cancer.

The court's finding against the EPA was based, for the most part, on the EPA's doing meta-analysis on only some of the studies it compiled, rather than on all of them, which the court likened to "cherry-picking," and on the EPA's extraordinary move of switching from a conventionally used 95% "statistical significance" confidence level for its preliminary reports, to a rarely used 90% level for its final report.

The switch was necessary, in the biased eyes of the study's authors, because the final report's RR came out as "statistically insignificant" under conventional computation. The 90% confidence level produces a tighter "confidence interval", so on that unusual basis, the EPA result could be called "statistically significant."

The EPA appealed, and won in 2002, on the issue that the court which had ruled against it did not technically have proper jurisdiction for the case. The finding of the appeals court was based only on the jurisdictional issue and not on the substance of the original judge's finding.

Despite the EPA report's especially abominable methodology, and its reputation as a slapstick blunder and a grotesque farce amongst knowledgeable persons, the public are largely unaware of these things, and tend to be impressed by big agencies such as the US Environmental Protection Agency.

Thus, and despite all, the 1992 EPA finding, which was RR 1.19 within 90% CI 1.04-1.35, has been, ever since its first publication in December of 1992, and still remains a favorite amongst the tobacco control crowd, for its usefulness in inspiring fear and hatred of smokers across the world.

Other attempts at meta-analysis, including most famously Hackshaw's in 1997, have avoided the most comical pratfalls of the EPA, while coming up with similar results of marginal "statistical significance."

In plain, there is no reason to believe, and there is no rational evidence to support the idea, that ETS presents a risk of any ailment whatsoever. If we feared the common air, with all of its constituents, including cooking, heating, automobile, industrial, and myriad other sources of smoke, we would have to ban breathing itself. Fear of ETS is madness.

Recall the studies in the previous section of this paper. Simple ventilation, such as kitchen exhaust fans, or opening windows, is according to common sense, and by all evidence, perfectly sufficient for keeping household air fresh under normal conditions. All of lifestyle

epidemiology must be viewed with skepticism, but compare if you wish, the "scientists'" own results regarding cooking fumes, to the ETS results you'll see on tables below. If secondhand smoke kills, cooking breakfast must equate to a thermonuclear attack.

There is, in the real world as opposed to the dream world of statistically maddened fanatics, *no such thing* as a 1.19 relative risk, or anything like it. The ETS studies shown below reported on lung cancer patient groups ranging in size from a handful to a few hundred. Whether taken individually or collectively, and despite the very clear bias in the presentation of most of these studies, they suggest nothing. Lifestyle epidemiology does not have the power to suggest decimal risks. It does not have the power to suggest whole number risks with any remote accuracy.

Specific criticisms, widely voiced, of ETS lung cancer studies and meta-analyses have included the following:

1. Nothing was measured. As with our Doctor Who study the data comes from subjects' responses to interviews or on questionnaires. Two examples of original study questionnaires are included amongst appendices to this paper. Lung cancer patients are typically geriatric. Expecting precise memory of *other persons'* smoking in specific and varied periods dating back to childhood is essentially ridiculous. Likewise, there is generally no context, e.g. the size of a house one might have lived in decades previously, or whether it was well or poorly ventilated. Even when roughly accurate the subjects' memories *cannot* provide any precise or scientifically valuable index of exposure.

2. Smoker misclassification: smokers may deny smoking, particularly when diagnosed with lung cancer, therefore will be classified as never smokers. Even a small number of smokers, given the real risk of active smoking relative to lung cancer, if classified as never smokers, would throw off results enormously. Some studies try to adjust for this possibility. There is no sound basis for estimating how so to adjust. Most study authors simply ignore the question.

3. Publication bias has been previously described. It presents another question, pertinent to meta-analyses, which is usually simply ignored. Occasional attempts to adjust for this question – just as it is with smoker misclassification – have no sound basis. One cannot adjust for phantoms. They may well be there, but the exact shape of them, one cannot know.

4. Also specifically regarding meta-analyses, the ETS studies are by no means uniform, in

terms of classifying by age or place of exposure, or extent of exposure, or in presentation of data. They may show cell counts. They may not. Their results may be adjusted or may not be adjusted. If adjusted, the fashion of adjustment may be for one set of factors, or another altogether different set of factors, and this may be clearly delineated, or virtually not at all delineated. Some studies are quite detailed and may run to dozens or hundreds of pages. Others are no more than a paragraph or two. Most were published, but others are only available as poorly copied manuscripts, prepared by students of statistics. Meta-analysts are faced with trying to fit infinitely shaped results into one slot; they do so by whittling edges. Meta-analyses are attempts to build houses with mulch. They may serve the purposes of propagandists but cannot stand up to a single breath of scrutiny.

These criticisms, especially number 1, are enough in themselves to destroy any credibility for ETS studies, never mind, of a *decimal accuracy* of ETS studies. For further perspective we can do well to look at some results regarding active smoking, for ever smokers, from studies of recent years. Here we have a relatively clear case of a statistical association. Take a look at how accurate the active smoking studies are, all approaching the very same question, in essentially the same way.

What is an ever smoker's relative risk of lung cancer? You have to look at a whole lot of research to get even a general idea of what the whole number risk may be, and with these active smoking studies you have to consider region, as well, and as we have described. We provide six examples below, three from the West, and three from the East.

Table design for study results is the same in this section as for the previous section of this essay and the same scheme of abbreviation is used. Citations for these six studies are:

Wang, YC: "p53 Codon 72 Polymorphism in Taiwanese Lung Cancer Patients: Association with Lung Cancer Susceptibility and Prognosis": 1999: *Clinical Cancer Research*; 5; 129-134

Chan-Yeung, M: "Risk Factors Associated with Lung Cancer in Hong Kong": 2003: *Lung Cancer*; 40; 131-140

Franco-Marina, F: "Role of Active and Passive Smoking on Lung Cancer Etiology in Mexico City": 2006: *Salud Publica de Mexico*; 28; 3 (supplement)

Rylander, R: "Lung Cancer Risks in Relation To Vegetable and Fruit Consumption and Smoking": 2006: *International Journal of Lung Cancer*; 118; 739-743

Khurana, V: "Statins Reduce the Risk of Lung Cancer in Humans: A Large Case-control Study of US Veterans": 2007: *Chest*; 131; 1282-1288

Yun, B: "Sequence Variations in DNA Gene XPC Is Associated with Lung Cancer Risk in a Chinese Population: A Case-control Study": 2007: *BMC Cancer*; 7; 81; 31 pages

Principal Author	Year	Location	Sex	Relative Risk	Confidence Interval	Comments
Wang, Y-C	1999	Taiwan	M&F	1.54	0.89-2.66	T 1.
Chan-Yeung	2003	Hong Kong	M&F	3.78	1.11-12.92	T 2.
Franco-Marina	2006	Mexico	M&F	4.00	2.90-5.50	T 2.
Rylander	2006	Sweden	M&F	7.17	6.34-8.48	T 1 cell counts: (487/49/532/384)
Khurana	2007	United States	M&F	2.13	1.98-2.30	T 1.
Yun	2007	China	M&F	2.08	1.72-2.50	T 4 cell counts: (670/297/513/472)

So this is the clearest case in the statistical canon of a link with lung cancer, and what, would you say, is the RR for ever smokers? Two maybe? Would you say seven? Something roughly in between? Or would you call it 1.19 or 2.19 or 3.19 or 4.19 or 5.19 or 6.19 or 7.19? Or do the confidence intervals suggest the answer is really something less than one or closer to thirteen? Would you then call it, say 0.19, or 13.19? Does lifestyle epidemiology really have decimal accuracy? The cultists say so. Do you agree with them?

The cultists say so when it pleases them to say so. In 1992 the EPA decreed, based on its 1.19 RR finding with the jiggered confidence interval, that ETS merited classification as a Class A Carcinogen, or "known cause" of lung cancer, the highest risk classification in the EPA's armament. Also in the early nineteen-nineties the EPA had declined to classify electromagnetic radiation as a known (Class A) or even as a suspected (Class B) cause of cancer. Its rationale for that decision? That studies' RR results did not consistently exceed the whole number 3.

In biostatistics dealing with common lifestyle factors, consistent results over many studies are necessary for providing any inference, and relative risks below five are always very iffy. Those below two are absolutely meaningless; positive decimals mean nothing. The "authorities" ignore this when it suits them. They tell the public to ignore it when they wish to sway the public as it suits them. But they know it. The most prominent among them have said they know it. For example:

Professor James Enstrom, epidemiologist with the University of California at Los Angeles, regarding infinitesimal risks such as those claimed for ETS: "You're talking about ratios so close to 1.0 that it's really beyond the realm of epidemiology."

Professor Julian Peto of the International Agency for Research on Cancer: "Small associations below 2.0 may be beyond the limits of reliable epidemiological inference."

The US National Cancer Institute: "In epidemiological research, relative risks of less than 2 are considered small and are usually difficult to interpret. Such increases may be due to chance, statistical bias, or effects of confounding factors that are sometimes not evident."

The World Health Organization: "Relative risks of 2.0 may readily reflect some uperceived bias or confounding factor, those over 5.0 are unlikely to do so."

Doctor Sir Richard Doll of Oxford stated that "when 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."

New England Journal of Medicine editor Doctor Marcia Angell: "As a general rule of thumb, we are looking for a relative risk of 3 or more before accepting a paper for publication."

Doctor Robert Temple, Director of Drug Evaluation, US Food and Drug Administration: "My basic rule is if the relative risk isn't 3 or 4, forget it."

Looking at RR results for active smoking and lung cancer, as with examples above, and looking at biostatistics generally, makes the vast imprecision of lifestyle epidemiology obvious to any thinking person. Epidemiologists make themselves patently ridiculous when they pretend that ETS presents any credible risk for any medical condition whatsoever.

As mentioned, in pooling study results for compendia such as the 1992 EPA report and Hackshaw's 1997 publication, the compilers will assess studies' background data when such is available, and will "whittle"or alter some original RR results on various bases, in an attempt to provide a level of consistency for the stew they are creating. Some other study results, presented in original reports without background data, get put into the stew just as they are, into whatever category they come closest to fitting, and in whatever state of adjustment (or lack of adjustment) they may have had originally, regardless of whether

this has any consistency with the general concoction being presented.

For presentation here, study results are placed, as best they fit, onto tables representing five general categories: ETS exposure in childhood at home, beyond the childhood years at home, in the workplace, in social settings such as bars and restaurants, and (a smaller collection) in travel settings such as cars, planes, and trains. As with previous study result tables above, wherever practicable, results are reported according as data was originally published, with indication provided as to where the pertinent data appeared within original publications.

The original studies are inconsistent with each other in multifarious ways and one cannot generalize an overall exposed / not exposed category. As appropriate, and as with previous tables here, one or two results from each study, on each table for which they pertain, are provided. Dual results are categorized typically as representing lowest and highest results pertaining (a simple minimum and maximum), or by lower and higher exposure level findings, or by longer and shorter duration of exposure, or otherwise as discussed in Comments for individual entries.

Where necessary for clarity, or where results have been corrected or revised since their original publication, secondary sources consulted for results tabulated are referenced in Comments section. Confidence intervals are reported as they were published. The great majority are at the 95% level with a minority at a 90% level.

As we have discussed, it was long held as established that smoking was not related to the adenocarcinoma histological type of lung cancer. More recent contention that smoking has some effect on adenocarcinoma risk would seem to be nothing more than an artifact of generally increasing diagnostic bias regarding lung cancer. Adenocarcinoma is the most common type of lung cancer amongst never smokers. It accounts for an estimated 70% of all lung cancer amongst never smokers.

As the famous British doctor and medical writer James LeFanu has noted: “Passive smoking cannot conceivably cause lung cancer.” He ridicules the notion “that it allegedly causes a type of cancer in non-smokers, adenocarcinoma, known not to be related to smoking.”

“ETS kills” propagandists have tried to explain this away with the argument that smokers breathe smoke only shallowly into the lungs while people breathe generally deeply into the lungs, therefore ETS is breathed in to the deeper portions of the lung where

adenocarcinoma characteristically is located, therefore ETS causes adenocarcinoma.

This ignores, of course, that unless non-smokers spend most of their time sitting in smokers' laps, it is the smokers themselves who are most exposed to ETS, so if ETS "causes" adenocarcinoma it should always have been "causing" it in smokers. The propagandists' argument, in other words, assumes that smokers only smoke, and do not breathe. It assumes that smokers are only smokers and not human beings. Naturally, the cultists would think that, wouldn't they? They always do.

There have been two reactions to this adenocarcinoma stumbling block amongst lifestyle epidemiologists. Some of the earlier researchers eliminated some or all adenocarcinoma patients from their studies. Others have sought to reinforce the usual argument, essentially that people breathe while smokers don't, by limiting their choice of study subjects exclusively to never-smokers with adenocarcinoma.

ETS lifestyle epidemiology is a farce on any basis, but in reflection of this particular issue, following on our five general exposure tables below, we recapitulate the adenocarcinoma-only results collectively on a sixth chart.

Five among the studies presented require some explanation in advance.

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Hirayama 1981-1984:

Takeshi Hirayama (1927-1995) began his large long-term study of Japanese smoking in 1965 and published numerous reports on his work into the 1990s. He presented his current findings on active smoking as a lecturer at the World Health Organization's 1975 World Conference on Smoking and Health, and picked up the gauntlet Conference Chairman Sir George Godber threw, by subsequently creating the very first passive smoking figures.

Hirayama's final ETS reports came in 1984 but publication of his preliminary ETS findings in 1981 initiated the kinds of mainstream press headlines Sir George had sought. The 1981 advance publication stated generally, "The relative risk of developing lung cancer by passive smoking was about 1.8 compared with about 3.8 in direct smokers." For "direct" or active smokers Hirayama's research ultimately resulted in relative risk reports of 3.76 for males and 2.03 for females (as reported, without confidence intervals, on Table 4.2 within Chapter 4 of US EPA 1992).

The final 1984 ETS results were, for males, 2.25 (1.19-4.22), and for females, 1.45 (1.04-2.02). The staggering implausibility of ETS figures which rival or exceed actual smoking risks researched by Hirayama for the same study population was blindly ignored by the press in general. It still is ignored generally.

Hirayama's 1981 publication was, as one would expect, greeted by tobacco abolitionists as if it were the finding of the Holy Grail, and so of course, they retain a great affection for it. Hirayama's remains the ETS study most often cited by tobacco control activists. As such, the study has generally been given absolution for not being a study of never smokers exposed to ETS, and has been included in subsequent compendia.

For that reason we include results from Hirayama's research on our tables, but give notice which previous compilers have neglected, that this is not a study of never smoking lung cancer cases. Hirayama accepted subjects who were long term smokers for ETS analysis. His criterion was only that they did not smoke *daily*.

Trichopoulos 1981-1983-1984

Patients with the adenocarcinoma types of lung cancer were excluded from this study on the basis that, since the authors considered active smoking unrelated to those types, they likewise reasoned that ETS could not be so related.

The situation with the Trichopoulos study is similar as with that of the Hirayama study cited above. Its preliminary results were published shortly after Hirayama's in 1981 and were greeted by abolitionists as a sort of second coming of fondest hopes.

The adoration of the Trichopoulos study by tobacco control activists also has held fast over the years, so this study has been given similar absolution as does Hirayama's, for not being a study of never smokers.

As with the Hirayama study, we include the Trichopoulos study on our tables, because previous compilers have done so in reflection of the affection for this study amongst smoker pogrom enthusiasts, but we give notice generally neglected by others, that this is not a study of never smokers.

Trichopoulos studied what he called non-smokers, these being defined as persons who had not smoked within twenty years of lung cancer diagnosis. This definition would have included habitual long-term smokers.

According to age charts in the longer 1981 version of this report the average age of cases was sixty-two, the vast majority of all patients were beyond fifty, and more than a quarter are categorized in the highest bracket of seventy and beyond. With smoking initiation in the teen years being common, "non-smokers" as defined would in some cases likely have had substantial smoking habits lasting up to about four decades.

Apart from that there is much about the Trichopoulos study that confuses. The original publications do not provide any clear indication of precise relative risk. As variously published, its data does not in some respects correspond logically, or make any clear sense. Reference to outside sources is necessary to clarify.

Trichopoulos published originally in 1981 with an addendum appearing in 1983. Data was corrected in 1984 via "personal communication from Trichopoulos" (see US Surgeon General's Report of 1986 pg. 78 and US EPA report of 1992 pg. 5-43).

The 1983 report is brief, and includes more patients, but is similar in its reports, and in its obscurity, to the 1981 original. No confidence intervals are provided relative to ETS. Textual reports are muddled. Statistical significance is indicated for trends in exposure ranges listing relative risks of anywhere from 1.3 up to 3.4 in the two published reports (an unexplained spousal ex-smoker category also enters in this) but statistical significance or insignificance is never designated textually or otherwise for any individual ETS-related figure.

An overall RR of 2.4 from ETS is also given in the 1981 report, but this apparently applies to all smoking categories (including "current"), rather than to the "non-smoker" class specifically, while again statistical significance is not remarked upon. Furthering confusion, Trichopoulos reports a very similar relative risk based on his study's data (2.9 within CI 1.3-6.8) for active smoking, independently of his ETS results.

In short, "smokers" and "non-smokers" are nearly indistinguished in the early publications while all else is hazily defined at best, and as this story progresses from 1984, things actually get more confused.

Based on the 1984 personally communicated corrections, US EPA 1992 (Table 5-11) computes low to high exposure RR range for ETS among "non-smokers" as being between 1.95 (CI 1.13-3.36) and 2.55 (CI 1.31-4.93), while SGR 1986 (Table 9) had reported the 1984 corrected range as between 1.9 (CI 0.9-3.7) and 2.5 (CI 1.7-3.8). British biostatistician Peter N. Lee has computed an overall RR of 2.08 with a 95% confidence interval of 1.20-3.59

which we tabulate here.

Trichopoulos's 1984 data corrections are specifically cited in both the EPA and SG reports but are nowhere specifically described. Through comparison of the 1981-1992 published sources one can clearly deduce that parts of the 1983 report were misprinted (but never retracted in the *Lancet*) yet this still does not make the secondary report disparities comprehensible.

Various interpretations of Trichopoulos's evidently verbally reported but unpublished corrections may have been made by various parties at EPA and the Surgeon General's Department of Public Health. Nothing is clear about the Trichopoulos reports.

Since publication of this famous study Dimitrios Trichopoulos has advanced from the University of Athens to Harvard. He became the Director of the Department of Hygiene and Epidemiology at the Harvard School of Public Health in 1989 and still holds that post as of 2012.

Humble 1987:

Charles G. Humble reported ETS findings pertaining both to males and to females in his 1987 publication. He mentioned only generally in his original report that four of his total 28 never smoker cases had been found out as smokers subsequently to their original interviews but Humble published results for all 28 despite this. The situation was clarified years later.

In section A.4.14.3 Comments of the EPA's December 1992 ETS report we read in reference to Humble's study: "The ETS subjects (never-smokers) include 20 (4) female (male) [i.e. 20 female and 4 male] cases ... (the article reports 8 male cases, the number used in much of the analysis, but 4 of those 8 were found to be smokers, personal communication from Humble)."

Therefore, fully half of the males reported on in Humble's original report were smokers misrepresented as never smokers. We follow the example of subsequent compilers in reporting Humble's results only for females.

Schoenberg 1989:

Janet Schoenberg's 1989 study had a focus on radon exposure and has been cited in

subsequent literature on radon with regard to lung cancer. Schoenberg also did an analysis of ETS with regard to lung cancer in an addendum to her main report. This ETS data has been overlooked by subsequent compilers. A copy (nearly complete) of this study is included amongst appendices to this paper.

Schoenberg's study was performed in the US state of New Jersey, under the auspices of the state's Department of Health, and with funding from the US National Cancer Institute. The ETS data is specific to never-smoking lung cancer cases and includes textual reports reflecting childhood and adulthood exposure as well as cell counts and RR computations specific to exposure of never smoking women to smoking by their husbands.

We provide Schoenberg's results on our tables based on her textual reports, and specifically for spouse-related exposure as Schoenberg delineated them, according to whether the husband smoked cigars or a pipe (RR 0.52 within CI 0.22-1.30) or cigarettes (RR 1.20 within CI 0.75-1.80). Table B2 cell counts (73/43/303/196) can also be computed to an overall result for any form of smoking by the husband of 1.10 within CI 0.72-1.67.

With results reported for 116 never-smoking lung cancer cases the 1989 Schoenberg ETS study represented one of the larger such studies of the time. Had it been known of by the 1992 EPA authors, the spouse-related data would have qualified for their meta-analysis, and would have had a marginal effect in depressing the EPA's final result.

Wang, S. 1996:

On page 6 of 7 in Sheng-yong Wang's study there is discussion of never smokers and nonsmokers in which an ETS-related relative risk of 2.5 (CI 1.3-5.1) is mentioned. Previous compilers have evidently taken this to represent a unique computation specific to never smokers. We therefore follow precedent in including the finding on our table. However, the finding does not actually appear to be specific to never smokers. Rather, it seems more likely and simply to be an application to nonsmokers of Wang's Table Two ETS result (shown there as 2.54, $P < 0.05$) for all study subjects including smokers.

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Review of the corpus of ETS studies leaves one in awe of the power of propaganda. The tobacco control establishment has created enormous damage to society out of pure dross. The ETS studies are perfectly consistent in suggesting absolutely nothing. They are rife with reverse dose responses (more exposure creates less risk), with "statistical insignificance", and with plain defiance of common sense.

In the previous section of this paper, we saw some of the very worst of lifestyle epidemiology, and there is much more of the worst to come on tables below. The debasement of the reputation of science by lifestyle epidemiologists has produced scathing criticism from distinguished critics, and even a few *mea culpae* from within the debased profession itself, such as:

Doctor Charles Hennekens, Professor of Epidemiology, Harvard School of Public Health: "Epidemiology is a crude and inexact science ... We tend to overstate findings, either because we want attention or more grant money."

Doctor Dimitrios Trichopoulos, Director of the Department of Epidemiology, Harvard School of Public Health: "We are fast becoming a nuisance to society … People don't take us seriously anymore, and when they do take us seriously, we may unintentionally do more harm than good."

Epidemiologic icon Sir Richard Doll, on BBC radio, admitted, "The effects of other people smoking in my presence is so small it doesn't worry me."

Doctor Gio Gori has never saluted the flag of fanaticism. He summed up the pseudo-scientific research promoting what he calls "the ETS fraud" most aptly, in noting, "The emperor is stark naked."

The most comprehensive and up-to-date accounting of never smoker studies relating to lung cancer is that researched for the tobacco industry by British biostatistician Peter N. Lee. Lee's document "Epidemiological Evidence on Environmental Tobacco Smoke and Lung Cancer" (which, when referenced on tables below, shall be called the "P.N. Lee compendium") includes publishing citations for nearly all studies included on our tables, and also accounts for other studies, which may mention ETS in relation to lung cancer, but which do not report on never smokers, or are otherwise specifically deficient, or simply redundant of other publications. The most recent edition of this document (published in December of 2011) may be accessed at:

http://www.pnlee.co.uk/documents/refs/lee2011S.pdf

Publication citations for five additional never smoker ETS/lung cancer studies, which have not appeared in previous compilations, but which appear on our tables below, are as follows:

Schoenberg, J: "A Case-Control Study of Radon and Lung Cancer Among New Jersey

Women": August, 1989, publication of the New Jersey State Department of Health, Division of Epidemiology and Disease Control, Division of Occupational and Environmental Health.

Neuberger, J: "Risk Factors for Lung Cancer in Iowa Women: Implications for Prevention": 2006: *Cancer Detection and Prevention*; 30(2); 158-167

Pandey, A: "Lifetime Environmental Exposure to Tobacco Smoke and Primary Lung Cancer of Non-smoking Women in [a] Developing Country": 2008: *Epidemiology*; 19; 6; p S359

Wang, X: "The roles of smoking and cooking emissions in lung cancer risk among Chinese women in Hong Kong": 2009: *Annals of Oncology*; 20: 746–751

Kiyohara, C.: "Methylenetetrahydrofolate reductase polymorphisms and interaction with smoking and alcohol consumption in lung cancer risk: a case-control study in a Japanese population": 2011: *BMC Cancer*; 11:459; 10 pages

Tables of study results are preceded by an itemization of studies included, 94 in all, listed by size according to the number of never smoker lung cancer patients studied.

Principal Author	Year	Total Males	Total Females	Study Type	Total Number of Never-smoking Lung Cancer Patients Studied
94) Butler	1988	None	8	Cohort	8
93) Hole	1989	3	6	Cohort	9
92) Brownson (87)	1987	None	19	Case / Control	19
91) Humble	1987	None	20	Case / Control	20
90) deWaard	1995	None	23	Case / Control	23
89) Nishino	2001	None	24	Cohort	24
88) Inoue	1988	None	28	Case / Control	28
87) Wu	1985	None	29	Case / Control	29
86) Gallegos-Arreola*	2008	19	13	Case/Control	32
85) Svensson	1989	None	34	Case / Control	34
84) Speizer	1999	None	35	Cohort	35
83) Correa	1983	8	28	Case / Control	36
82) Liu, Q.	1993	None	38	Case / Control	38

81) Auvinen*	1996-98	41	3	Case / Control	44
80) Lee, P.	1986	15	32	Case / Control	47
79) Rylander	2006	18	31	Case / Control	49
78) Buffler	1984	11	41	Case / Control	52
77) Geng	1988	None	54	Case / Control	54
76) Liu, Z.	1991	None	54	Case / Control	54
75) Rachtan	2002	None	54	Case / Control	54
74) Neuberger	2006	None	56	Case/Control	56
73) Rapiti	1999	17	41	Case / Control	58
72) Vineis*	2005	17	42	Cohort	59
71) Lam, W.	1985	None	60	Case / Control	60
70) Layard	1994	21	39	Case / Control	60
69) Pershagen	1987	None	67	Case/Control	67
68) Shen	1998	None	70	Case / Control	70
67) Boffetta (99)	1999	4	66	Case / Control	70
66) Johnson	2001	None	71	Case / Control	71
65) Du	1993-96	None	75	Case / Control	75
64) Trichopoulos	1981-84	None	77	Case / Control	77 (*current* non-smokers)
63) Kabat (84)	1984	25	53	Case / Control	78
62) Jee	1999	None	79	Cohort	79
61) Hosseini	2010	26	55	Case / Control	81
60) Wang, S.	1996	None	82	Case / Control	82 (all smoking categories)
59) Chan	1982	None	84	Case / Control	84
58) Zatloukal	2003	None	84	Case / Control	84
57) Koo	1984-87	None	88	Case / Control	88
56) Choi	1989	13	75	Case / Control	88
55) Shimizu	1988	None	90	Case / Control	90
54) Kalandidi	1990	None	90	Case / Control	90
53) Franco-Marina	2006-09	22	72	Case / Control	94
52) Zheng	1997	25	69	Case / Control	94
51) Chuang*	2011	12	86	Cohort	98

50) Dalager	1986	29	70	Case/Control	99
49) Wen	2006	None	106	Cohort	106
48) Kurahashi	2008	None	109	Cohort	109
47) Kabat (95)	1995	41	69	Case / Control	110
46) Akiba	1986	19	94	Case / Control	113
45) Schoenberg	1989	None	116	Case/Control	116
44) Sobue	1990	None	120	Case / Control	120
43) Malats	2000	17	105	Case / Control	122
42) Kreuzer	2000-04	23	100	Case / Control	123
41) Tse	2009	132	None	Case / Control	132
40) Garfinkel (85)	1985	None	134	Case / Control	134
39) Hill (B)	2007	111	123	Cohort	134
38) Wang, T.	1996	None	135	Case / Control	135
37) Asomaning	2008	56	82	Case / Control	138
36) Jiang	2010	47	98	Case / Control	145
35) Hill (A)	2007	84	63	Cohort	147
34) Garfinkel (81)	1981	None	153	Cohort	153
33) Kiyohara*	2011	95	58	Case/Control	153
32) Zhang	2007	None	155	Cohort	155
31) Brenner	2010	46	110	Case/Control	156
30) Fang	2006	None	157	Case/Control	157
29) Zaridze	1998	None	189	Case / Control	189
28) Janerich	1990	45	146	Case / Control	191
27) Gorlova	2006	63	130	Case / Control	193
26) Lam, T.	1987	None	199	Case / Control	199
25) Seow	2002	None	176	Case / Control	176
24) Yu	2006	None	200	Case / Control	200
23) Stockwell	1992	None	210	Case / Control	210
22) Olivio-Marston*	2009	71	146	Case/Control	217
21) Ohno	2002	None	224	Case / Control	224
20) Liang	2009	None	226	Case/Control	226

19) Sun	1996	None	230	Case / Control	230
18) Wang, L.	2000	33	200	Case / Control	233
17) Gao	1987	None	246	Case / Control	246
16) Enstrom	2003	79	177	Cohort	256
15) Schwartz	1996	72	185	Case / Control	257
14) Hirayama	1981-84	64	200	Cohort	264 (never smoked *daily*)
13) Lee, C.	2000	None	268	Case / Control	268
12) Pandey	2008	None	268	Case / Control	268
11) Wang, X.	2009	None	279	Case / Control	279
10) Yang*	2008	151	134	Case / Control	285
9) McGhee	2005	145	179	Case / Control	324
8) Cardenas	1994-97	116	246	Cohort	362
7) Wu-Williams	1990	None	417	Case / Control	417
6) Brownson (92)	1992	None	431	Case / Control	431
5) Lagarde	2001	191	242	Case / Control	433
4) Lo	2010	123	339	Case / Control	462
3) Zhong	1999	None	504	Case / Control	504
2) Boffetta (98)	1998	141	509	Case / Control	650
1) Fontham	1994	None	653	Case / Control	653

* Specific male/female numerical breakdown for never-smokers was not provided in original study. Male/female breakdown is here represented as an approximation based on percentages reported for study's total patient population which included both smokers and never smokers.

ETS EXPOSURE AT HOME (SPECIFIC TO CHILDHOOD)

Principal Author	Year	Location	Sex	Base Relative Risk(s)	Confidence Interval(s)	Comments
Correa	1983	United States	M&F	NR	NR	[P 2 of 3].
Garfinkel (85)	1985	United States	F	0.91	0.58-1.42*	Comments extended below.

Garfinkel (85) Comments: RR shown is from Page 4 of 7. All of the confidence intervals shown in Lawrence Garfinkel's 1985 study are miscalculated and invalid (see retraction letter by A. Judson Wells and S. Jane Henley, *Journal of the National Cancer Institute* 1997; 89; 821-822). * Corrected CI shown here is from the P.N. Lee compendium.

Wu	1985	United States	F	0.60	0.20-1.70	[P 2 of 5]. In Wu's study, reports for never-smoking subjects relate only to the adenocarcinoma type of lung cancer rather than to all lung cancer.
Akiba	1986	Japan	M&F	NR	NR	[P 3 of 4].
Dalager	1986	United States	M&F	NR	NR	[P 2 of 4]. Dalager does not provide category-specific figures while reporting no significant risk from exposure at home in childhood and/or adulthood.
Gao	1987	China	F	1.10	0.70-1.70	[P 2 of 6].
Koo	1984-1987	Hong Kong	F	2.07	0.51-95.17	[1987 T 2]. Comments extended below.
Koo (87) Comments: Note that 2.07 is the RR report for childhood exposure *only*. This same study (as tabulated on adulthood chart below) also records an RR for ETS exposure *throughout* childhood *and* adulthood as 0.64 (CI 0.57-5.85). Don't just look at the ludicrous contradiction of the base RR figures though. Also note the two vast "confidence" intervals. Start with the blue CI within these comments, suggesting something like half the risk as for a non-exposed person (0.57 vs. 1.00), or is it about six times the risk (5.85), then keep going, look up to the charted blue CI, see the risk newly further below the norm (0.51) or, wait a minute now, is it (95.17) nearly *a hundred times* the norm? (This would translate to at least a 200% chance, in other words a cosmic certainty, of contracting lung cancer from ETS.) Bear in mind the mantral contention that the lung cancer risk from ETS exposure is *fractionally* above 1.00. You will see many comically wide confidence intervals in ETS studies to follow. Error in computation or typography is inferentially indicated in this case, but no correction apparently ever was made by the authors or publishers, which reflects the plain shoddiness evident in much so-called "peer reviewed" and "authoritative" lifestyle epidemiology. In a general context, and we dare say at a minimum, common sense tells us a CI range that so much as halves or doubles its base RR is enough by itself utterly to extinguish all credibility. A coin flip would be as accurate. In fact most items on these charts cannot even pass the heads or tails test, but as you move along here, keep an eye out for extra-wide confidence intervals. They are among the more prominent holes that shine the light through rotten apple statistics.						
Pershagen	1987	Sweden	F	1.00	0.40-2.30	[T 5].
Geng	1988	China	F	NR	NR	[P 2 of 4].
Schoenberg	1989	United States	F	NR	NR	[Text throughout, T B 2, T B 6]. Schoenberg does not provide category-specific figures while reporting no significant risk for never smokers from exposure at home in childhood and/or adulthood.
Svensson	1989	Sweden	F F	3.30 0.90	0.50-18.80 (mother) 0.40-2.30 (father)	[T 7]. Did you notice the confidence interval accompanying the motherly figure? We told you to watch for that didn't we?

Janerich	1990	United States	M&F M&F	1.09 2.07	0.68-1.73 (low duration) 1.16-3.68 (high duration)	[T 2].
Sobue	1990	Japan	F F	1.71 0.60	0.95-3.10 (mother) 0.40-0.91 (father)	[P 4 of 12, T 2].
Brownson (92)	1992	United States	F F	0.50 0.80	0.20-0.80 (minimum) 0.50-1.40 (maximum)	[T 1].
Stockwell	1992	United States	F F	1.60 1.20	0.60-4.30 (mother) 0.60-2.30 (father)	[T 2].
Fontham	1994	United States	F F	0.99 0.88	0.73-1.35 (low duration) 0.67-1.16 (high duration)	[T 5].
Kabat (95)	1995	United States	M F	0.90 1.55	0.43-1.89 0.95-2.79	[T 2].
Sun	1996	China	F	2.29	1.56-3.37	The Sun study has only been published as a single-paragraph abstract. Comments extended below.
Sun Comments: The Xei-wi Sun study has only been published as a single-paragraph abstract and presents a conflicting hodge-podge of results with minimal descriptive detail. For these charts we have tabulated best-appropriate results by category from the confusing and conflicting array of results mentioned in the brief and poorly presented original publication. Some of the results displayed in the report explicitly relate only to subgroups with compounded category exposures, these results omitting single-category exposure respondents, while other results rearrange category focus, in some cases explicitly omitting compounded exposures: in short, Sun tends for the most part to display small portions of his data picture while neglecting the wider view. Charted childhood result is presented here by virtue of general pertinence to the childhood category, although it is not clear whether this may be an overall computation, or alternatively may refer to subgroup computation contingent on specific inclusion or exclusion of exposures in categories other than childhood. The short piece is simply unclear on this and other points, while relating both significant and insignificant results, interpretable as pertaining to various or multiple exposure categories, and conflicting in import. For example, with specific regard to household exposure, evidently related to whole lifetime and specifically absent workplace exposure, Sun textually reports no significant risk, and for the specific workplace computation, which excludes household exposure, Sun also reports insignificant findings. On the other hand some explicitly combined-category analyses relating to home and workplace exposure, and others which seemingly may refer somehow or other to home and/or workplace, with specific inclusions or exclusions left undefined, are reported as producing significant results. Sun furthermore reports conflicting dose response results in text, while only displaying one (negative) dose response with numbers, this being relative to spousal exposure. Textually, Sun suggests that “long-term” ETS exposure may increase lung cancer risk, while complaining that previous studies produced “inconsistent data,” a description well-befitting his own perplexingly bouncing reports.						
Wang, T.	1996	China	F	0.91	0.55-1.49	[T 1]. Figures shown are from unadjusted analyses. Adjusted figures are not specified although Wang states his conclusion that lung cancer is not related to ETS exposure from any reported exposure in childhood and/or adulthood as based on the confirmation of his adjusted analyses.

Boffetta (98)	1998	Europe	M&F M&F	0.78 0.81	0.64-0.96 (test 1) 0.66-0.99 (test 2)	[Figure 1]. Comments extended below.
Boffetta (98) Comments: Paolo Boffetta's 1998 study was based on reports from numerous European centers. Methods of apportioning cases and controls were inconsistent between centers. Boffetta provides two tests for this result, the first a general reconciliation of data, the second reflecting specific standards of case/control matching.						
Zaridze	1998	Russia	F	0.92	0.64-1.32	[T 3]. Responses were collected regarding both parents but only results referent to father's smoking, shown here, are reported.
Boffetta (99)	1999	Europe	M&F M&F	1.00 0.30	0.40-2.40 (low duration) 0.10-0.90 (high duration)	[T 2]. This study was of patients with the adenocarcinoma form of lung cancer only.
Rapiti	1999	India	M&F M&F	1.30 12.00	0.50-3.50 (minimum) 4.20-34.00 (maximum)	[T 2]. Are you still watching the "confidence" intervals? Is it half? Is it 3,400%? Don't forget to laugh.
Speizer	1999	United States	F	NR	NR	[PP 4, 6 of 8]. Speizer does not provide category-specific figures while reporting no significant risk based on data collected for childhood and adulthood exposures.
Zhong	1999	China	F F	0.90 0.90	0.50-1.80 (low duration) 0.50-1.90 (high duration)	[T 3].
Lee, C.	2000	Taiwan	F F	0.90 1.70	0.30-3.10 (mother) 1.10-2.60 (father)	[T 3].
Wang, L.	2000	China	M&F	1.52	1.10-2.20	[T 2].
Johnson	2001	Canada	F	0.54	0.10-2.70	[T 2].
Ohno	2002	Japan	F	NR	NR	[P 3 of 12, T 5]. Comments extended below.
Ohno Comments: Yoshiyuki Ohno provides two tests of each result on a stratified list of specific early life exposures ranging from young childhood through adolescence, none of which are generally applicable, and results for all but one of which are statistically insignificant. The single statistically significant result of RR 0.65 within CI 0.47-0.91 applies with amusing specificity to smoking by the father during the child's high school years.						
Rachtan	2002	Poland	F	2.53	1.45-4.41	[T 3].

Zatloukal	2003	Czechoslovakia	F F	1.35 2.10	0.75-2.45 (minimum) 1.02-4.33 (maximum)	[T 3]. The 1.35 RR relates to adenocarcinoma and the 2.10 to the other three major types (squamous, large, and small cell.) Zatloukal does not provide result for lung cancer generically but this is computed in P.N. Lee compendium as 1.61 within CI 1.01-2.57.
Kreuzer	2000-2001-2002-2004	Germany	M F	0.97 0.90	0.40-2.30 0.50-1.40	Kreuzer published data between 2000 and 2002 which overlapped with the Boffetta 1998 data. Kreuzer's unique data, reflected here, was first published in Table 2.6 of the 2004 monographs on carcinogenesis (Volume 83) of the International Agency for Research on Cancer.
Vineis	2005	Europe	M&F M&F	1.08 3.63	0.45-2.59 (low exposure) 1.19-11.11 (high exposure)	[T 4].
Neuberger	2006	United States	F	NR	NR	[Sections 2,3 and 4 of text, T 1.] Comments extended below.
Neuberger Comments: John Neuberger reports both statistically significant and statistically insignificant childhood at home and adulthood home and/or workplace exposure RR figures below nullity for larger case group and textually reports no risk for never-smokers specifically but does not provide separate calculations exclusive to never-smokers.						
Wen	2006	China	F	0.21	0.03-1.61	[T 4].
Yu	2006	Hong Kong	F	NR	NR	[PP 1 through 4 of 7]. Yu does not provide category-specific figures while reporting no significant risk from lifetime exposure in childhood and/or adulthood at home and/or at work.
Zhang	2007	Japan	F	NR	NR	[Text throughout, T1]. Comments extended below.
Zhang Comments: Yawei Zhang, who queried patients regarding their whole lifetimes, reports that: "The percentage reporting ever exposure to passive smoking at home and/or work was lower in lung cancer patients than in the cohort" without providing specific calculation or category-specific data. The RR is calculable by cell count division from Table One data [127/28/57207/9025] as 0.72 within CI 0.47-1.08 relating to ever exposure at home and/or at work.						

Asomaning	2008	United States	M&F	1.03	0.70-1.54	[Computed from T 2 cell counts]. Comments extended below.
Asomaning Comments: Kofi Asomaning's study divides categorical ETS results for never, lighter and heavier smokers in an unorthodox and convoluted manner. It does not report conventionally categorized exposed/unexposed RR results for any smoking category but those for never smokers are here calculated from cell counts which are provided in study Table Two. "First exposure" in young life is defined by Asomaning as coming before age 25 with cell counts inclusive of all 138 never-smoking case subjects of 89/49/297/169 computed to results shown in row above representing home exposure for this approximate "childhood" category. Study Table Three reports results for more exposures versus fewer exposures relevant to never smokers, throughout life and at any combination of home, work, and/or social settings, as between 0.87 (CI 0.22-3.38) and 1.29 (0.82-2.02).						
Kurahashi	2008	Japan	F	0.93	0.52-1.66	[P 3 of 5].
Pandey	2008	Nepal	F	**1.80**	**1.20-2.90**	Published only as a brief abstract.
Liang	2009	China	F	0.78	0.49-1.25	[T2.]
Olivio-Marston	2009	United States	M&F	1.47 **2.25**	1.00-2.15 (minimum) **1.04-4.90 (maximum)**	[T 2.] Study reported on two distinct patient groups.
Brenner	2010	Canada	M&F	1.00	0.60-1.80	[T 2.]
Chuang	2011	Europe	M&F	0.97	0.64-1.50	[T 2.]

ETS EXPOSURE AT HOME (EXTENDING BEYOND CHILDHOOD)

Principal Author	Year	Location	Sex	Base Relative Risk(s)	Confidence Interval(s)	Comments
Garfinkel (81)	1981	United States	F F	1.27 1.10	0.85-1.89 (low exposure) 0.77-1.61 (high exposure)	[T 4]. Males included in study but ETS reports only given for females.
Chan	1982	Hong Kong	F	NR	NR	[P 1 of 4, T 3].
Correa	1983	United States	M F	2.00 2.07	NS NS	[T 1]. Patients with alveolar type lung cancer were excluded from the Correa study on the basis that, since the authors considered active smoking unrelated to that adenocarcinoma sub-type, they likewise reasoned that ETS could not be so related.

Buffler	1984	United States	M F	0.52 0.78	0.15-1.74 0.34-1.81	[T 7].
Hirayama (Basis: never smoked *daily*.)	1981-1984	Japan	M F	**2.25** **1.45**	**1.19-4.22** **1.04-2.02**	[T1, T6]. See comments preceding these tables.
Kabat (84)	1984	United States	M&F	1.00	0.50-2.01	[T 3 cell counts (22/56/22/56).]
Trichopoulos (Basis: *current* non-smokers.)	1981-1983-1984	Greece	F	**2.08***	**1.20-3.59***	* Results as computed for the P.N. Lee compendium. See also comments preceding these tables.
Garfinkel (85)	1985	United States	F	1.31	0.87-1.98*	Comments extended below.
Garfinkel (85) Comments: RR shown is from Table 5. All of the confidence intervals shown in Lawrence Garfinkel's 1985 study are miscalculated and invalid (see retraction letter by A. Judson Wells and S. Jane Henley, *Journal of the National Cancer Institute* 1997; 89; 821-822). * CI shown here is from Table 5 corrections accompanying the 1997 retraction letter.						
Lam, W.	1985	Hong Kong	F	**2.01***	**1.09-3.72***	Comments extended below.
Lam, W. Comments: This is an unpublished study of which only excerpts are generally available to this day. US EPA reported on it based on an incomplete draft they requested and received in the early 1990s. Since then the EPA analysis has been cited by researchers, so we report on this study here based on comparison of EPA's descriptions with available excerpts from the original. Wah Kit Lam's hand-typed doctoral thesis takes a specific focus on the prevalence of adenocarcinoma amongst female non-smokers with lung cancer and it is in this regard that subsequent researchers have cited his observations. His researches encompassed males and females but only female subjects were studied relative to ETS and ETS was one of three factors studied alongside cooking smoke and incense exposure common to Hong Kong households. Lam notes inconsistency in his ETS data and states in particular that his data was sparse regarding lung cancer other than of the adenocarcinoma types, given the marked predominance of those types amongst his female patients, "and did not therefore afford meaningful statistical analysis." Based on data regarding a subset of patients, textual suggestion of a possible relationship specifically between husband's smoking and peripherally located adenocarcinoma in women is made, with some data and general analysis provided, but no odds ratio is computed in the original. * Relevant RR and CI for adenocarcinoma only, pertaining to spousal exposure, are shown here as computed on page A-100 of US EPA 1992.						
Wu	1985	United States	F	1.20	0.50-3.30	[P 2 of 5]. In this study, reports for never-smoking subjects relate only to the adenocarcinoma type of lung cancer rather than to all lung cancer.
Akiba	1986	Japan	M F	1.80 1.50	0.50-5.60 1.00-2.50	[T 2].

Dalager	1986	United States	M&F	NR	NR	[P 2 of 4]. Dalager does not provide category-specific figures while reporting no significant risk from exposure at home in childhood and/or adulthood.
Lee, P.	1986	United Kingdom	M&F M&F	0.98 0.86	NS (low exposure) NS (high exposure)	[P 4 of 10, T 4].
Brownson (87)	1987	United States	F	1.68	0.39-6.90*	Comments extended below.
Brownson 1987 Comments: This study was of patients with the adenocarcinoma form of lung cancer only. It included both male and female subjects but does not include RR report for male never-smokers. RR is from study Table 4. The originally reported CI of 0.39-2.97 has been noted in subsequent literature as being at variance with background data. * Corrected CI is from P.N. Lee compendium.						
Gao	1987	China	F	0.90	0.60-1.40	[P 2 of 6].
Humble	1987	United States	F F	1.80 1.20	0.60-5.60 (low exposure) 0.30-5.20 (high exposure)	[T 4]. Note discussion of this study, in narrative section, preceding these charts.
Koo	1984-1987	Hong Kong	F F	1.68 0.64	0.62-5.45 (low duration) 0.57-5.85 (high duration)	[1987 T 2]. The RR of 1.68 is for subjects exposed only in adulthood. The 0.64 figure is for subjects exposed in both childhood and adulthood.
Lam, T.	1987	Hong Kong	F	**1.65**	**1.16-2.35**	[T 4].
Pershagen	1987	Sweden	F	1.20	0.70-2.10	[P 4 of 8].
Butler	1988	United States	F	2.02	0.48-8.56	[P 12 of 22]. Males included in some category reports but ETS at home report is only given for females.
Geng	1988	China	F	**2.16**	**1.08-4.29***	Comments extended below.
Geng Comments: Guan-Yi Geng's 1988 study has been cited in subsequent literature, so we include it here, although it is plagued with errors. Geng's raw data appears coherent if sketchy and unrefined, and the spousal exposure RR of 2.16 (from study Table 5) corresponds with sparse data provided, but attempts at closer analysis shown on Table 6 are clearly incompatible with the raw numbers of patients and controls, i.e mathematically impossible, and not subject to correction given the paucity of base data. Another overall RR for spousal exposure of 1.86 is given on Table 7 but this is not explained or explainable with reference to background data. The confidence interval accompanying the Table 5 RR is also erroneously computed. (See "Simple Methods for Checking for Possible Errors in Reported Odds Ratios, Relative Risks and Confidence Intervals", Peter N. Lee, *Statistics in Medicine* 1999; 18; 1973-1989). * Corrected CI is from the 1999 article.						
Inoue	1988	Japan	F	2.25	0.77-8.85*	Comments extended below.

Inoue 1988 Comments: RR is from study page 2 of 3. The originally reported confidence interval of 0.91-7.10 has been noted in subsequent literature as seemingly at variance with background data. * Corrected CI is from the P.N. Lee compendium.						
Shimizu	1988	Japan	F F	0.80 **4.00**	NS (minimum) **S (maximum)**	[T 1]. Comments extended below.
Shimizu Comments: Figures shown are low and high among an unusual array provided, some indicated as statistically significant, others as not. The low figure is for cohabiting with husband's smoking mother in adulthood and the high figure is for cohabiting with one's own smoking mother in adulthood. Maybe one of the old ladies rolls her own out of hemlock. Incidentally, some subsequent publications have taken the figures shown here as representative of childhood exposure, but the figures clearly relate to women cohabiting with parents and/or in-laws in adulthood (extended households are common in the Orient.) Hiroyuki Shimizu's study is of only 90 patients, and is unconventionally presented, providing limited textual description, and no confidence intervals. P.N. Lee computes the Shimizu data to an overall RR of 1.08 within CI 0.64-1.82.						
Choi	1989	Korea	M F	2.70 1.60	NS NS	[T4].
Hole	1989	Scotland	M&F	2.41	0.45-12.83	[T 7].
Schoenberg	1989	United States	F F	0.52 1.20	0.22-1.30 (minimum) 0.75-1.80 (maximum)	[T B6]. Schoenberg states no significant risk from any exposure at home in childhood and/or adulthood and provides results shown here specific to exposure from the husband. The smaller RR figure pertains to never-smoking women whose husbands smoked cigars or pipes and the larger for those whose husbands smoked cigarettes.
Svensson	1989	Sweden	F	NR	NR	[T 7]. Svensson does not provide category-specific figures while reporting no significant risk from adulthood exposure at home and/or at work.
Janerich	1990	United States	M&F M&F	0.16 1.80	0.04-0.62 (minimum) 0.83-3.90 (maximum)	[T 3, T 2].
Kalandidi	1990	Greece	F	**1.92**	**1.02-3.59**	[P 4 of 7].
Sobue	1990	Japan	F F	0.94 1.45	0.62-1.40 (minimum) 0.94-2.23 (maximum)	[T 1].
Wu-Williams	1990	China	F	0.70	0.60-0.90	[PP 2, 3 of 6. T 3].

Liu, Z.	1991	China	F	0.77	0.30-1.96	[P 1 of 6]. Males also included in some category reports but ETS report only given for females.
Brownson (92)	1992	United States	F F	0.70 1.30	0.50-1.00 (minimum) 1.00-1.80 (maximum)	[T 2]. Comments extended below.
Brownson Comments: Results above are for lung cancer generically. Ross Brownson's Table Three also provides specific RR reports by lung cancer type. The cancer types associated with active cigarette smoking are squamous and small cell lung cancer. For non-smoking women living with smokers, Table Three provides RR reports as low as 0.2 within CI 0.1-1.1 (at highest level of ETS exposure) for squamous cancer, and 0.5 within CI 0.0-4.8 (at lowest level of ETS exposure) for small cell cancer. Note that the 0.0 end of the CI spectrum provided in these reports would suggest a cosmic certainty of freedom from disease thanks to ETS exposure while the 4.8 extreme exceeds many reported risks from decades of *active* smoking. If any of this makes sense to Brownson, he is alone, or at least as we hope, he soon shall be.						
Stockwell	1992	United States	F	1.60	0.80-3.00	[T 2].
Liu, Q.	1993	China	F F	0.70 **2.90**	0.23-2.20 (low exposure) **1.20-7.30** (high exposure)	[T 3]. Males also included in some category reports but ETS report only given for females.
Fontham	1994	United States	F	1.23	0.96-1.57	[T 6].
Layard	1994	United States	M F	1.47 0.58	0.55-3.94 0.30-1.13	[T 4].
deWaard	1995	Netherlands	F F	2.70 2.40	0.80-9.10 (low exposure) 0.70-8.30 (high exposure)	[T 2]. Regular ETS exposure, at home and/or elsewhere, was the criterion used for this result.
Kabat (95)	1995	United States	M F	1.13 0.95	0.53-2.45 0.53-1.67	[T 2].
Du	1993-1995-1996	China	F	NR	NR	[1996 P 26 of 29]. Comments extended below.
Du Comments: Ying-xiu Du reports not on a single study but on his wide series of researches into various aspects of lung cancer aetiology in the 1980s. These studies included both sexes but Du analyzes ETS only regarding women and reports uniformly insignificant results both generally in text and in various RR figures given with either CI or P value indications of statistical insignificance. Du discusses ETS at some length in his several reports. He concludes that a lack of association between ETS and lung cancer is not surprising, particularly in light of the questionable association between active smoking and adenocarcinoma, the form of lung cancer predominant amongst never smokers. Du summarizes his team's ETS researches in stating: "A lack of correlation was obtained, regardless of whether smoking spousal status, the number of cigarettes smoked per day, or smoking duration (in years), was considered."						
Schwartz	1996	United States	M&F	1.10	0.80-1.60	[T 2].
Sun	1996	China	F F	1.16 0.86	0.80-1.69 (low duration) 0.45-1.65 (high duration)	The Sun study has only been published as a single-paragraph abstract. Comments extended below.

Sun Comments: The Xi-wei Sun study has only been published as a single-paragraph abstract and presents a conflicting hodge-podge of results with minimal descriptive detail. Charted results, the best category-specific match, are for living with a smoking spouse, at top the overall figure, with a long-term exposure figure (i.e. more than 35 years) below it. This computation may or may not specifically include or exclude other sources of exposure: this is simply not made clear. Sun specifically reports no significant risk for either household exposure absent workplace exposure, generally and evidently pertaining to whole lifetime, or for workplace exposure absent home exposure, but additionally reports a risk of **1.83** within CI **1.20-2.80** as pertinent to adulthood exposure, this being unspecific as to ETS source and also unclear as to whether computed to include or exclude particular locational or age-specific exposures. Results as presented on these charts make the best sense possible of Sun's sparse and multiply conflicting report, conforming as well with his closing statement that "the risk seems to be higher when exposure occurs in childhood and adolescence than in adulthood." Refer also to comments following the Sun 1996 entry on our childhood chart (above) for more background on this peculiarly presented study.						
Wang, S. (Basis evidently included smokers)	1996	China	F	**2.50**	**1.30-5.10**	P 6 of 7. See comments preceding these tables.
Wang, T.	1996	China	F	1.41 1.08	0.68-1.94 (low duration) 0.37-3.14 (high duration)	[T 2]. Figures shown are from unadjusted analyses. Adjusted figures are not specified although Wang states his conclusion that lung cancer is not related to ETS exposure from any reported exposure in childhood and/or adulthood as based on the confirmation of his adjusted analyses.
Cardenas	1994-1997	United States	M F	1.10 1.20	0.60-1.80 0.80-1.60	[1997 T 3].
Zheng	1997	China	M&F	1.04	0.59-1.85	[T 1].
Auvinen	1996-1998	Finland	M&F	0.69	0.28-1.74	[T 3, revised]. The Auvinen study was originally published in 1996 with data republished in corrected form in 1998.
Boffetta (98)	1998	Europe	M&F M&F	1.16 1.15	0.93-1.44 (test 1) 0.89-1.37 (test 2)	[Figure 1]. Comments extended below.
Boffetta (98) Comments: Paolo Boffetta's 1998 study was based on reports from numerous European centers. Methods of apportioning cases and controls were inconsistent between centers. Boffetta provides two tests for this result, the first a general reconciliation of data, the second reflecting specific standards of case/control matching.						
Shen	1998	China	F F	0.65 0.70	0.19-2.12 (low exposure) 0.27-1.76 (high exposure)	[T 3, P 3 of 3]. This study was of patients with the adenocarcinoma form of lung cancer only.
Zaridze	1998	Russia	F F	**1.66** 1.35	**1.09-2.52 (low exposure)** 0.84-2.18 (high exposure)	[T 3].

Boffetta (99)	1999	Europe	M&F	1.00	0.50-1.80	[T 3]. This study was of patients with the adenocarcinoma form of lung cancer only.
Jee	1999	Korea	F F	1.30 1.90	0.60-2.70 (low duration) 1.00-3.50 (high duration)	[T 1].
Rapiti	1999	India	M&F M&F	0.10 **5.10**	0.01-1.20 (minimum) **1.50-17.00 (maximum)**	[T 3]. Ha, **ha**.
Speizer	1999	United States	F	1.50	0.30-6.30	[PP 4, 6 of 8].Speizer's criterion for this result, adulthood exposure at home and/or at work was considered to pertain primarily to home exposure.
Zhong	1999	China	F F	1.20 1.00	0.80-1.70 (low duration) 0.70-1.60 (high duration)	[T 2]. RR of 1.20 is for home exposure in adulthood only. RR of 1.00 is for home exposure in both childhood and adulthood.
Lee, C.	2000	Taiwan	F F	1.20 **3.30**	0.70-2.20 (minimum) **1.70-6.20 (maximum)**	[T 3].
Malats	2000	Europe & Brazil	M&F	1.50	0.80-2.60	[T 4]. This study considered subjects' gene types. Overall figure included here is related to home exposure specifically and encompasses all study subjects.
Wang, L.	2000	China	M&F M&F	0.81 0.86	0.50-1.30 (low duration) 0.50-1.50 (high duration)	[T 2].
Johnson	2001	Canada	F	1.21	0.50-2.80	[T 3].
Lagarde	2001	Sweden	M&F	1.15	0.93-1.43	[Computed from T 6 cell counts]. Comments extended below.
Lagarde 2001: Frédéric Lagarde's study has a primary focus on radon gas exposure in the home and does not give a distinct ETS risk estimate but does provide cell counts [178/254/611/997] relevant to ETS exposure at home in adulthood. Resulting relative risk with confidence interval are charted above.						
Nishino	2001	Japan	F F	0.39 1.90	0.11-1.40 (minimum) 0.81-4.40 (maximum)	[T 3, T2]. Comments extended below.

Nishino Comments: Yoshikazu Nishino considered the dreaded ETS effect on cancer of the lung and also on cancers of numerous other parts of the body. Forty-eight RR reports are given on three charts. The highest RR charted is 2.60 (CI 0.97-6.80) for cancer of the rectum. The study specifically defines rectal cancer, incidentally, as being unrelated to active smoking. If non-smokers are sitting down on live cigarettes we implore them to stop. In fact every one of the forty-eight RR reports is statistically insignificant, with the exceptions, of three reports for breast cancer. The three "confident" and "significant" results suggest that ETS exposure cuts a woman's risk of breast cancer in half.						
Ohno	2002	Japan	F F	0.76 1.00	0.52-1.11 (test 1) 0.67-1.49 (test 2)	[T 6]. Comments extended below.
Ohno Comments: Yoshiyuki Ohno presents relative risks based on comparison of his lung cancer patient population with not one but two "control" or comparison populations unafflicted with lung cancer, one control group derived from the general population, the other comprised of hospital patients only. The risk estimates rarely coincide between the two analyses. In this instance, marriage to a smoker produced a base relative risk estimate of 0.76 based on analysis comparing to the general population but 1.00 when the hospital population was compared, while the lower end of the confidence interval alters from 0.52 to 0.67 and the higher end from 1.11 to 1.49.						
Seow	2002	Singapore	F	1.30	0.90-1.80	[P 2 of 7]. Sparse description of ETS exposure was here assumed to relate primarily to spousal smoking.
Enstrom	2003	United States	M F	0.63 0.94	0.33-1.22 0.66-1.33	[T 7, T 8].
Zatloukal	2003	Czechoslovakia	F F	0.36 0.66	0.11-1.22 (minimum) 0.22-1.96 (maximum)	[T 3]. Regular ETS exposure, at home and/or elsewhere, was the criterion used for these results. The 0.36 RR relates to adenocarcinoma and the 0.66 to the other three major types (squamous, large, and small cell.) Zatloukal does not provide result for lung cancer generically but this is computed in P.N. Lee compendium as 0.48 within CI 0.21-1.09.
Kreuzer	2000-2001-2002-2004	Germany	M F	0.40 0.80	0.10-3.00 0.50-1.30	[T 2.2 IARC 2004].
McGhee	2005	Hong Kong	M&F	**1.39**	**1.03-1.88**	[T (on P 2 of 2)].
Vineis	2005	Europe	M&F	NR	NR	[T3]. Vineis does not provide category-specific figures for never-smokers while textually reporting no significant risk for never smokers from exposure at home and/or at work.
Fang	2006	China	F	**1.77**	**1.07-2.92**	From study abstract.

Gorlova	2006	United States	M&F	1.64	0.94-2.88	[T 3].
Neuberger	2006	United States	F	NR	NR	[Sections 2,3 and 4 of text, T 1.] Comments extended below.
Neuberger Comments: John Neuberger reports both statistically significant and statistically insignificant childhood at home and adulthood home and/or workplace exposure RR figures below nullity for larger case group and textually reports no risk for never-smokers specifically but does not provide separate calculations exclusive to never-smokers.						
Rylander	2006	Sweden	M&F	1.37	0.72-2.61	[P 2 of 5].
Wen	2006	China	F	0.89	0.42-1.92	[T4].
Yu	2006	Hong Kong	F	NR	NR	[PP 1 through 4 of 7]. Yu does not provide category-specific figures while reporting no significant risk from lifetime exposure in childhood and/or adulthood at home and/or at work.
Hill (A)	2007	New Zealand	M F	0.97 1.00	0.53-1.77 0.49-2.01	[P 1 of 11, T 3]. Comments extended below.
Hill (B)	2007	New Zealand	M F	1.45 1.16	0.75-2.81 0.70-1.92	[P 1 of 11, T 3]. Comments extended below.
Hill (A) and (B) Comments: Sarah E. Hill's 2007 paper reports on two separate cohort studies, the first (A) dating to the 1980s, the second (B) to the 1990s. Standardized RR results are provided separately for males and for females for both cohorts, with alternative adjusted RR results provided for some computations, but not for others. The consistently computed standardized results are shown here. The selectively computed adjusted RR results all lie within the same range and are likewise shown as statistically insignificant, null results.						
Zhang	2007	Japan	F	NR	NR	[Text throughout, T1]. Comments extended below.
Zhang Comments: Yawei Zhang reports that: "The percentage reporting ever exposure to passive smoking at home and/or work was lower in lung cancer patients than in the cohort" without providing specific calculation or category-specific data. The RR is calculable via cell counts in Table One data [127/28/57207/9025] as 0.72 within CI 0.47-1.08 relating to ever exposure at home and/or at work.						
Asomaning	2008	United States	M&F	0.93	0.39-2.19	[Computed from T 2 cell counts]. Comments extended below.
Asomaning Comments: Kofi Asomaning's study divides categorical ETS results for never, lighter and heavier smokers in an unorthodox and convoluted manner. It does not report conventionally categorized exposed/unexposed RR results for any smoking category but those for never smokers are here calculated from cell counts which are provided in study Table Two. Cell counts specific to adulthood home exposure can only be extracted from figures as presented on Table Two for Asomaning's subgroup of subjects whose "first exposure" to ETS occurred after age twenty-five, with cell counts 25/9/96/32 computed to results shown in row above. Cell counts representing entire case group of 138 never smokers of 114/24/393/73 can be computed as RR 0.88 within CI 0.53-1.46 representing lifetime home exposure (i.e. in childhood and/or adulthood.)						
Gallegos-Arreola	2008	Mexico	M&F	**8.00**	**1.83-34.92**	[T 1 cell counts (30/2/90/48).]

Kurahashi	2008	Japan	F	1.02 1.61	0.51-2.04 (minimum) 0.83-3.11 (maximum)	[T2, T3].
Pandey	2008	Nepal	F	**2.20**	**1.40-3.70**	Published only as a brief abstract.
Yang	2008	United States	M&F	1.20	0.80-1.90	[T3]. Comments extended below.
Yang Comments: The focus of Ping Yang's study was on gene types and medical history relating to both smokers and never-smokers. RR and CI shown reflect the entire group of 285 never-smokers irrespective of gene type (18% of total 1,585 respondents.) The author's sparse description of ETS analysis – "ETS exposure was modeled as a dichotomized covariate (yes vs. no)" – is here assumed to refer primarily to spousal smoking.						
Franco-Marina	2006-2009	Mexico	M&F	**1.80**	**1.10-3.00**	[IARC 2009.] Original 2006 publication does not give figures pertinent to never smokers. Figures shown here are those relevant to never smokers as reported in International Agency for Research on Cancer 2009 handbook on page 16.
Liang	2009	China	F	1.05	0.69-1.60	[T 2.]
Tse	2009	Hong Kong	M M	0.93 0.77	0.57-1.51 (low exposure) 0.32-1.81 (high exposure)	[T3].
Wang, X.	2009	Hong Kong	F	NR	NR	[PP 1,5, T2].
Wang, X. Comments: This study had a focus on active smoking and on cooking fumes. Xiao-rong Wang reports that: "We also made an attempt at evaluating the effect of ETS among women who were lifelong nonsmokers and found no evident elevated risk." RR calculations relevant to ETS on Table Two, variously adjusted or not adjusted and unspecifically pertaining to exposure at home and/or at work, are uniformly null ranging from 0.95 within CI 0.54-1.68 to 1.16 within CI 0.66-2.0.						
Brenner	2010	Canada	M&F	1.00	0.50-2.00	[T 2.]
Hosseini	2010	Iran	M F	1.50 1.50	0.60-3.60 0.80-3.00	[T 2.] Results based on lifetime exposure pertaining mainly to the home.
Jiang	2010	China	M&F	**2.46**	**1.53-3.94**	[T 3.]
Lo	2010	Taiwan	M&F	**2.20**	**1.64-2.95**	[T1 cell counts (366/96/293/169).] Results based on lifetime exposure considered to pertain mainly to home in adulthood.
Kiyohara	2011	Japan	M&F	1.00	0.65-1.55	[T1 cell counts (99/54/135/74).]

ETS EXPOSURE IN THE WORKPLACE

Principal Author	Year	Location	Sex	Base Relative Risk(s)	Confidence Interval(s)	Comments
Kabat (84)	1984	United States	M&F	1.11	0.59-2.09	[T3 cell counts (44/34/42/36).]
Garfinkel (85)	1985	United States	F F	0.88 0.93	0.46-1.67 (minimum)* 0.55-1.55 (maximum)*	Comments extended below.
Garfinkel (85) Comments: RR shown is from Table 7. All of the confidence intervals shown in Lawrence Garfinkel's 1985 study are miscalculated and invalid (see retraction letter by A. Judson Wells and S. Jane Henley, *Journal of the National Cancer Institute* 1997; 89; 821-822). * Confidence intervals shown here are from Table 7 corrections accompanying the 1997 retraction letter.						
Wu	1985	United States	F	1.30	0.50-3.30	[P 2 of 5]. In this study, reports for never-smoking subjects relate only to the adenocarcinoma type of lung cancer.
Lee, P.	1986	United Kingdom	M&F	NR	NR	[P 4 of 10, T 4]. Lee provides six variables for workplace exposure with detailed response data, results for which are uniformly statistically insignificant and not generally applicable, while both the minimum and maximum (0.19 and 3.24) are related only to male and not to female response data.
Koo	1984-1987	Hong Kong	F	0.91	NS	[1984 P 1 of 8, T 2].
Geng	1988	China	F	NR	NR	[P 2 of 4].
Shimizu	1988	Japan	F	1.20	NS	[PP 1, 3 of 9, T 1].
Svensson	1989	Sweden	F	NR	NR	[T 7]. Svensson does not provide category-specific figures while reporting no significant risk from adulthood exposure at home and/or at work.
Janerich	1990	United States	M&F	0.91	0.61-1.35*	Comments extended below.

Janerich Comments: Charted RR of 0.91 for workplace exposure is from Page 3 of 5 in Dwight Janerich's original report. The original confidence interval given with it (0.80-1.04) is reportedly at variance with source data and has been corrected in subsequent literature (see "Lung Cancer from Passive Smoking at Work", A. Judson Wells, *American Journal of Public Health* 1998; 88; 1025-1029). * Corrected CI shown here is from the 1998 article.						
Kalandidi	1990	Greece	F	1.08	0.24-4.87	[P 5 of 7].
Wu-Williams	1990	China	F	1.20*	0.90-1.60	Comments extended below.
Wu-Williams Comments: Workplace RR of 1.1 (shown on Table 3 and Page 3 of study) was subsequently corrected by Anna Wu-Williams. The originally reported CI is correct. (See "Lung Cancer from Passive Smoking at Work", A. Judson Wells, *American Journal of Public Health* 1998; 88; 1025-1029). * RR shown here is from the 1998 article.						
Brownson (92)	1992	United States	F	0.98*	0.74-1.31*	Comments extended below.
Brownson Comments: Ross Brownson discusses workplace data selectively in text but overall RR and CI for never-smokers are not presented. These have been calculated from the original Brownson data for subsequent literature (see "Lung Cancer from Passive Smoking at Work", A. Judson Wells, *American Journal of Public Health* 1998; 88; 1025-1029). * RR and CI shown here are from the 1998 article.						
Stockwell	1992	United States	F	NR	NR	[P 4 of 5].
Fontham	1994	United States	F	**1.39**	**1.11-1.74**	[T 6].
Kabat (95)	1995	United States	M F	1.02 1.15	0.50-2.09 0.62-2.13	[T 2].
Schwartz	1996	United States	M&F	1.50	1.00-2.20	[T 2].
Sun	1996	China	F	1.38	0.94-2.04	The Sun study has only been published as a single-paragraph abstract. Refer also to home exposure tables (childhood and adulthood) above for more detail on this peculiarly presented study.
Wang, T.	1996	China	F	0.89	0.45-1.77	[T 1]. Figures shown are from unadjusted analyses. Adjusted figures are not specified although Wang states his conclusion that lung cancer is not related to ETS exposure from any reported exposure in childhood and/or adulthood as based on the confirmation of his adjusted analyses.
Cardenas	1994-1997	United States	M&F M&F	0.80 1.20	0.50-1.40 (low exposure) 0.80-2.00 (high exposure)	[1994 T 33].
Boffetta (98)	1998	Europe	M&F M&F	1.17 1.10	0.94-1.45 (test 1) 0.89-1.37 (test 2)	[Figure 1]. Comments extended below.

Boffetta (98) Comments: Paolo Boffetta's 1998 study was based on reports from numerous European centers. Methods of apportioning cases and controls were inconsistent between centers. Boffetta provides two tests for this result, the first a general reconciliation of data, the second reflecting specific standards of case/control matching.						
Zaridze	1998	Russia	F	0.88	0.55-1.41	[T 3].
Boffetta (99)	1999	Europe	M&F	1.50	0.80-3.00	[T 4]. This study was of patients with the adenocarcinoma form of lung cancer only.
Rapiti	1999	India	M&F	1.10	0.30-4.10	[P 4 of 7].
Speizer	1999	United States	F	NR	NR	[PP 4, 6 of 8]. Speizer does not provide category-specific figures while reporting no significant risk from childhood or adulthood exposure at home and/or work.
Zhong	1999	China	F	1.90	0.90-3.70	[T 2]. The 1.90 RR is for exposure in workplace only. An RR for exposure at work, and at home in adulthood, and at home in childhood as well, is reported as 1.60 within CI 0.90-2.70.
Lee, C.	2000	Taiwan	F	1.20	0.50-2.40	[T 3].
Malats	2000	Europe & Brazil	M&F	NR	NR	[T 4]. Malats does not provide a category-specific figure while reporting statistically insignificant risk (for all never smoker study subjects) from ever exposure via spouse or in the workplace.
Wang, L.	2000	China	M&F	1.56	0.70-3.30	[P 2 of 7]. Comments extended below.

Wang, L. Comments: Note that the Longde Wang study appears on three charts here. It produced statistically insignificant results for home exposure to ETS in childhood, at home in adulthood, and in the workplace. In fact a look at the study's Table Two reveals a vast array of thirty-five ETS RR reports encompassing compounded variables but only two RR figures of marginal and eccentric statistical significance (the risk from lower ETS exposure shown as significant while that from higher exposure is not, risk to both sexes individually shown as insignificant but to both sexes combined as significant.) However, if crowning absurdity interests you, don't miss Table One of this study. Table One reports larger and statistically significant deviations from nullity in lung cancer risk results for "lifestyle factors" other than ETS exposure. Results include an RR for lung cancer of **0.25** for male subjects who own black and white TV sets as opposed to an RR of **3.64** for men who own color sets. Ladies with color TV sets are somewhat safer with an RR of **1.97** but nearly as bad off if they own a refrigerator (RR **3.29**.) The imperiled refrigerator owners can offset their risk by owning "large animals" (RR **0.44** for owning one such beast, or even better, **0.43** for owning two or more.) Yes: large animals: we are told only that these may or may not be cattle. Wang does explain that appliance and beast ownership were conceived as indicators of relative affluence amongst rural residents which turned out to be self-refuting regarding lung cancer. Of course, based on such "confident" and "significant" results, epidemiological believers are free to conclude that watching black and white TV in a crowded barn may render sure immunity against lung cancer, while installing a color set in one's living room, or God forbid heading out to the refrigerator for a snack during commercial breaks, may clearly spell doom. This audaciousness underscores what good statisticians have always known**:** in reflection of debased methodology and sheer chance RR differences in studies such as this ranging from small fractions to compounding multiples of "normal" risk are *fully to be expected between any two groups*. Statistical association is quirky, unreliable, and must never be treated in itself as suggesting "causation." Nevertheless Wang and his co-authors, while making no suggestions regarding TV sets, animals, or refrigerators, do state that their overwhelmingly statistically insignificant results conform with the *possibility* of a *fractional* increase in lung cancer risk from ETS exposure. Propagated claims of "risk" from ETS are thoroughly ridiculous. There is nothing scientific about them. They are irresponsible and vicious scare-mongering

Johnson	2001	Canada	F	1.27	0.40-4.00	[T 3, P 2 of 5].
Ohno	2002	Japan	F F	0.80 1.38	0.56-1.15 (test 1) 0.92-2.05 (test 2)	[T 7]. Comments extended below.

Ohno Comments: Yoshiyuki Ohno presents results based on comparison of his lung cancer patient population with not one but two "control" or comparison populations unafflicted with lung cancer, one control group derived from the general population, the other comprised of hospital patients only. The risk estimates rarely coincide between the two analyses. In this instance, workplace ETS exposure produced a base relative risk estimate of 0.80 based on analysis comparing to the general population and 1.38 when the hospital population was compared, while the lower end of the confidence interval alters from 0.56 to 0.92 and the higher end from 1.15 to 2.05.

Kreuzer	2000-2001-2002-2004	Germany	M F	0.50 1.40	0.20-1.30 0.80-2.20	[T 2.5 IARC 2004].
Vineis	2005	Europe	M&F	NR	NR	[T 3]. Vineis does not provide category-specific figures for never-smokers while reporting no significant risk for never smokers from exposure at home and/or at work.
Gorlova	2006	United States	M F	**3.84** **11.66**	**1.04-14.17** **1.26-107.60**	[T 5]. Comments extended below.

Gorlova Comments: Are you still spotting wide confidence intervals? And still laughing? There does not appear to be technical error in these calculations. The error is in judgement. Such calculations should never be contemplated, let alone, published. Note these "statistically significant" workplace (only) figures and also note that for persons exposed *both at home and at work* Olga Gorlova reports RR figures for males of 2.56 within CI 0.69-9.47 and for females of 1.88 within CI 0-87-4.07. A non-smoking working girl with a cigar-chomping boss may face an absolute cosmic certainty of lung cancer – but if her husband smokes too – well then of course her risk is more than iffy. If you believe in that sort of thing.

Neuberger	2006	United States	F	NR	NR	[Sections 2,3 and 4 of text, T 1.] Comments extended below.
Neuberger 2006 Comments: John Neuberger reports both statistically significant and statistically insignificant childhood at home and adulthood home and/or workplace exposure RR figures below nullity for larger case group and textually reports no risk for never-smokers specifically but does not provide separate calculations exclusive to never-smokers.						
Rylander	2006	Sweden	M&F	2.26	0.93-5.48	[P 2 of 5].
Wen	2006	China	F	2.23	0.95-5.27	[T 4]. Comments extended below.
Wen Comments: Wanqing Wen's 2.23 RR is for subjects exposed exclusively at work. Exposure in childhood and via the spouse and at work is lower in the land of Wen at 2.17 within CI 0.94-5.03. Exposure only in adulthood but both at home and work is lower still at 1.24 within CI 0.44-3.51. Exposure both in childhood and at the workplace but not via the spouse is healthiest of all at 0.77 within CI 0.17-3.43. Color TV sets or large animals might just explain this mess (see L. Wang above) but Wen makes no attempt to do so.						
Yu	2006	Hong Kong	F	NR	NR	[PP 1 through 4 of 7]. Yu does not provide category-specific figures while reporting no significant risk from lifetime exposure in childhood and/or adulthood at home and/or at work.
Zhang	2007	Japan	F	NR	NR	[Text throughout, T1]. Comments extended below.
Zhang Comments: Yawei Zhang reports that: "The percentage reporting ever exposure to passive smoking at home and/or work was lower in lung cancer patients than in the cohort" without providing specific calculation or category-specific data. The RR is calculable by cell count division from Table One data [127/28/57207/9025] as 0.72 within CI 0.47-1.08 relating to ever exposure at home and/or at work.						
Asomaning	2008	United States	M&F	1.21	0.82-1.78	[Computed from T 2 cell counts]. Comments extended below.
Asomaning Comments: Kofi Asomaning's study divides categorical ETS results for never, lighter and heavier smokers in an unorthodox and convoluted manner. It does not report conventionally categorized exposed/unexposed RR results for any smoking category but those for never smokers are here calculated from cell counts which are provided in study Table Two. Cell counts 85/53/266/200 compute as odds ratio and confidence interval shown in row above and relate to exposure in the workplace relative to all 138 never-smoking case subjects.						
Kurahashi	2008	Japan	F	1.32	0.85-2.04	[T3].
Tse	2009	Hong Kong	M M	2.08 1.41	0.49-8.84 (low exposure) 0.84-2.36 (high exposure)	[T4].
Wang, X.	2009	Hong Kong	F	NR	NR	[PP 1,5, T2].
Wang, X. Comments: This study had a focus on active smoking and on cooking fumes. Xiao-rong Wang reports that: "We also made an attempt at evaluating the effect of ETS among women who were lifelong nonsmokers and found no evident elevated risk." RR calculations relevant to ETS on Table Two, variously adjusted or not adjusted and unspecifically pertaining to exposure at home and/or at work, are uniformly null ranging from 0.95 within CI 0.54-1.68 to 1.16 within CI 0.66-2.0.						

Brenner	2010	Canada	M&F M&F	1.30 1.20	0.8-2.20 (low duration) 0.7-2.00 (high duration)	[T 2.]

ETS EXPOSURE IN SOCIAL SETTINGS (e.g. RESTAURANTS & BARS)

Principal Author	Year	Location	Sex	Base Relative Risk(s)	Confidence Interval(s)	Comments
Garfinkel (85)	1985	United States	F F	1.42 1.77	0.75-2.70 (minimum)* 0.86-3.64 (maximum)*	Comments extended below.
Garfinkel (85) Comments: Relative risks are from Table 7. All of the confidence intervals shown in Lawrence Garfinkel's 1985 study are miscalculated and invalid (see retraction letter by A. Judson Wells and S. Jane Henley, *Journal of the National Cancer Institute* 1997; 89; 821-822). * Confidence intervals shown here are from Table 7 corrections accompanying the 1997 retraction letter.						
Lee, P.	1986	United Kingdom	M&F M&F	1.06 0.59	NS (low exposure) NS (high exposure)	[P 4 of 10, T 4].
Janerich	1990	United States	M&F	0.59	0.43-0.81	[P 3 of 5].
Stockwell	1992	United States	F	NR	NR	[P 4 of 5].
Fontham	1994	United States	F	**1.50**	**1.19-1.89**	[T 6].
Kabat (95)	1995	United States	M F	1.39 1.22	0.67-2.86 0.69-2.15	[T 2].
Cardenas	1994-1997	United States	M&F M&F	0.70 1.10	0.50-1.10 (low exposure) 0.60-2.00 (high exposure)	[1994 T 33].
Boffetta (98)	1998	Europe	M&F M&F	0.24 2.32	NS (minimum) NS (maximum)	[P 5 of 11].
Ohno	2002	Japan	F F	1.22 **2.55**	0.62-2.38 (minimum) **1.16-5.58** (maximum)	[T 8].
Asomaning	2008	United States	M&F	0.97	0.64-1.48	[Computed from T 2 cell counts]. Comments extended below.
Comments: Kofi Asomaning's study divides categorical ETS results for never, lighter and heavier smokers in an unorthodox and convoluted manner. It does not report conventionally categorized exposed/unexposed RR results for any smoking category but those for never smokers are here calculated from cell counts which are provided in study Table Two. Cell counts (99/39/337/129) compute as RR and confidence interval shown in row above and relate to exposure in social settings relative to all 138 never-smoking case subjects.						

ETS EXPOSURE IN TRAVEL SETTINGS (e.g. CARS, PLANES, TRAINS)

Principal Author	Year	Location	Sex	Base Relative Risk(s)	Confidence Interval(s)	Comments
Lee, P.	1986	United Kingdom	M&F	0.64	NS	[P 4 of 10, T 4].
Kabat (95)	1995	United States	M F	0.27 5.17	0.01-13.99 (minimum) 1.46-18.24 (maximum)	[T 2].
Boffetta (98)	1998	Europe	M&F	1.14	0.88-1.48	[P 5 of 11].
Rapiti	1999	India	M&F	5.20	1.90-14.00	[P 4 of 7].
Ohno	2002	Japan	F F	0.53 1.25	0.25-1.15 (minimum) 0.74-2.13 (maximum)	[T 8].

ADENOCARCINOMA-SPECIFIC RESULTS / ALL CATEGORIES

Principal Author	Year	Location	Sex	Base Relative Risk(s)	Confidence Interval(s)	Comments
Lam, W.	1985	Hong Kong	F	2.01	1.09-3.72	Home/Adulthood
Wu	1985	United States	F	0.60	0.20-1.70	Home/Childhood
Wu	1985	United States	F	1.20	0.50-3.30	Home/Adulthood
Wu	1985	United States	F	1.30	0.50-3.30	Workplace
Brownson (87)	1987	United States	F	1.68	0.39-2.97	Home/Adulthood
Shen	1998	China	F F	0.65 0.70	0.19-2.12 (low exposure) 0.27-1.76 (high exposure)	Home/Adulthood
Boffetta (99)	1999	Europe	M&F M&F	1.00 0.30	0.40-2.40 (low duration) 0.10-0.90 (high duration)	Home/Childhood
Boffetta (99)	1999	Europe	M&F	1.00	0.50-1.80	Home/Adulthood
Boffetta (99)	1999	Europe	M&F	1.50	0.80-3.00	Workplace
Zatloukal	2003	Czechoslovakia	F	1.35	0.75-2.45	Home/Childhood
Zatloukal	2003	Czechoslovakia	F	0.36	0.11-1.22	Home/Adulthood

Epilogue: The Provenance of Lifestyle Epidemiology

Sir Francis Galton, born 16 February 1822 in Birmingham, England, was a pioneer in producing statistical studies via questionnaire responses. He founded the statistical principle of standard deviation and is considered the principal founder of the general field of research now called biostatistics or lifestyle epidemiology.

Galton was born to a privileged family. He was a clever but neurotic boy, prone to physical and nervous breakdowns. Such infirmity repeatedly interrupted his collegiate career at Cambridge, ruining his chances of the honors degree he coveted. He did receive his ordinary degree in mathematics but ultimately abandoned medical training begun at King's College.

In adulthood he became a scientific dabbler, an early developer of techniques in fingerprint classification, and most infamously, the creator of the pseudo-science for which he coined the name (deriving from the Greek for "well-born") of "eugenics" in 1883.

Eugenics employed tortured statistics and biased analogies to the end of "proving" which sorts of humans were desirable and which were undesirable. Galton was a cousin of Charles Darwin, and his theories of eugenics have been described as "social Darwinism", in that they aimed at eliminating unfit persons to enhance evolution and what was called the human "germ-plasm".

Galton and an ever-growing contingent of eugenicists across the world defined their own sort as "well-bred": the worthy ones, the elite, the supermen, who got to decide what other sorts of persons should not exist. Naturally, the world's many racists and social snobs were greatly attracted to eugenics. It was, essentially, a sham science based on nothing more than narcissism.

Eugenics has become a dirty word since the World War Two era and worlwide knowledge of the Nazis' Holocaust of Jews and other perceivedly "unfit" persons. The use of the term eugenics gradually became extinct across the globe in the decades following the war. Eugenics also emphasised "racial purification" through behavior modification and Germany instituted an anti-smoking campaign during the Nazi era, strikingly similar if not quite so dictatorial, as that existing in much of the world today. Modern health dictators have picked up where Adolf Hitler left off.

Previous to the war, appallingly, the name of eugenics was considered a proud one. Practice in eugenics was considered highly progressive, principles of eugenics were taught in universities and medical schools across much of the world, and advocates of eugenics held great sway in influencing public policy.

Under the leadership of Adolf Hitler, Germany became the world leader in promoting eugenics, but prior to that, the United States was considered the most "progressive" nation in the world in implementing eugenics-based policies.

Although eugenics included a racist philosophy, particularly extolling what Americans called Nordics and the Nazis called Aryans (i.e. light-skinned and light-haired persons deriving from Northern Europe), practitioners of eugenics sought to eliminate from their populations what they considered defective and inferior genetic types of any race.

Therefore, in the US in the early twentieth century, while racial segregation and miscegenation laws of the South were maintained with the pseudo-scientific endorsement of eugenics, and exclusionary immigration policies were implemented(which closed the US door to many Jews seeking to escape the Nazis), policies of coerced sterilization aimed at perceived defectives of any race or background also advanced throughout the nation, particularly in California. American eugenicists became both impressed by and then jealous of Germany.

For example, as Hitler's anti-semitic, "euthanasia", and sterilization policies advanced, Joseph Dejarnette, eugenics advocate and superintendent of Virginia's Western State Hospital, complained in a 1934 letter to the the *Richmond Times-Dispatch*: "The Germans are beating us at our own game." Virginia, a slave state of the old Confederacy, remained a segregation state until the mid-twentieth century and had passed a eugenics-based sterilization law in 1924.

Policies inspired by the debased moral outlook of the eugenicists also outlived the Nazi era, particularly in their original home base, the United States. For example, the infamous Tuskegee Experiment was begun by the US Public Health Service (PHS), the bailiwick of the Surgeon General, in 1932. PHS workers wished to study the progress of syphillis in males.

Hundreds of poor rural black men, from areas surrounding Tuskegee, Alabama, were recruited for the study under the pretense of their receiving free medical examinations and care. Many of the men had syphillis. They were not told the nature of their illness. When the curative power of penicillin for syphillis was established in the nineteen-forties, the

Tuskegee study subjects still were not told of what they suffered, and were not given penicillin. Instead, their disease was allowed to progress and they were allowed to die of it.

The Tuskegee experiment was not ended until 1972. It had begun under the administration of Surgeon General Hugh S. Cumming (an ardent supporter of eugenics who had been a member of the Advisory Council of the American Eugenics Society) and continued under six of his successors to that office. Decades later, as details of the experiment leaked out, victims or their families received settlements typically of tens of thousands of dollars each.

As it was under the Nazis, amongst eugenics admirers in the US, persons of any race could be deemed "unfit" and therefore suitable for misuse and abuse. For example, in the nineteen-forties and 'fifties, researchers at the Massachusetts Institute of Technology enrolled retarded children housed at the Walter E. Fernald State School in Waltham, Massachusetts, in what was called the "Fernald Science Club."

Science club members attended no classes but had fun meetings at which they got snack treats and also were treated with free trips to Boston Red Sox baseball games. Meanwhile, without informed consent of the children or their guardians, and regularly for years on end, for the purposes of studying digestion, the "science club" members were fed radioactive cereal and injected with radioactive materials.

The state of Massachusetts, the Massachusetts Institute of Technology, and the Quaker Oats company were sued for damages decades later as details of the eugenics-inspired experiment came to public light. Victims and their families received varying amounts in settlements.

Racial segregation was phased out in the US in the nineteen-sixties but vestiges of it remained long after. Despite a 1967 ruling of the US Supreme Court that states' anti-miscegenation laws (banning marriage or intimacy between whites and non-whites) then existing – there were still sixteen such state laws at the time – were unconstitutional, the more racist states were slow to remove these laws from their state registers. Alabama was the last to do so. That was in the year 2000. Racism is no longer *en vogue*. Hate is though.

The creator of eugenics, Francis Galton, died in 1911, but his pseudo-scientific legacy survived him, having its British seat at the University College of London where he had endowed a Galton Chair of Eugenics. The University removed the Galton Chair of Eugenics in 1994 and removed Galton's name from its laboratory devoted to genetics in 2000 in reflection of public distaste for memories of eugenics. Under influence from admirers of

Francis Galton, however, the University re-instated the Chair in 2009, now calling it the Galton Chair of Genetics. Professor Nicholas Wood holds the chair as of 2012.

Galton had two protégés who became the first two men to hold the Galton Chair of Eugenics at University College London. Karl Pearson held the chair from its creation in 1904 until 1933. Pearson was an Englishman, named Carl rather than Karl at birth, but studied in Germany, and changed the spelling of his name reportedly out of combined admiration for German racial pride and for Karl Marx. Pearson was offered a knighthood in 1935 but turned it down in reflection of his distaste for Britain's capitalist system.

Sir Ronald Aylmer Fisher, Galton's other protégé, succeeded Pearson. Fisher is commonly considered the single most important developer of biostatistical techniques from the time of Francis Galton, and to have established practice in the field fundamentally as it exists today, for better or worse. He has frequently been called the "father" of modern statistical practice.

Statistical practice can be either better or worse. We have discussed some of both in this essay. There is no doubt that the originators of biostatistical practice, Galton, Pearson, and Fisher, had technical ability or even brilliance. Did they apply it wisely? Would you think them great philosophers?

Galton's biographer Martin Brookes (*Extreme Measures: The Dark Visions and Bright Ideas of Francis Galton*), an evolutionary biologist who worked in recent times at the Galton Laboratory at University College in London, describes the creator of eugenics thus:

> When mountaineers are asked why they risk their lives to scale a precipitous peak they often reply, "Because it's there". Galton seemed to apply a similar philosophy towards counting and measuring. Many of his studies were measuring for measuring's sake, the product of an obsessive drive he possessed from childhood. Galton's obsession unwittingly turned him into one of the Victorian era's chief exponents of the scientific folly. Experiments in tea-making, for instance, were a particular favourite. Galton could not just accept what came out of the pot. Instead, he had to devise complex mathematical equations to work out the best way of making a good brew, based on such crucial considerations as the temperature of the water and the time taken for stewing. Slicing a cake was also seen as a mathematical challenge, and his solution, "Cutting a round cake on scientific principles", was no doubt eagerly devoured by readers of a 1905 issue of *Nature*.

> Galton was a talented inventor. Many of his scientific measurements were obtained using apparatus of his own design. But here again, things sometimes took a peculiar turn. Fearful, for example, that his mind was in perennial danger of overheating, he added a hinged lid to a top hat to provide the necessary ventilation. The lid was raised and lowered by means of a rubber bulb that dangled stylishly from the brim. Other wayward inventions included a pair of spectacles designed for underwater reading, and a bicycle "speedometer" that consisted of nothing more than a sandglass which the rider was supposed to hold while counting the revolutions of the wheel. It never caught on.
>
> Galton possessed a potent mix of wisdom and whimsy. But other aspects of his character were less appealing. An immense snob, perennially occupied with distinctions of race, class, and social status, he was routinely dismissive of those he considered beneath him – women, black people, and the poor. He could be charming and tolerant to family and friends, but heartless and cruel to others.
>
> His cheerful, witty exterior concealed an exceptionally private man haunted by mental illness. His diaries – each less than two inches square – chronicled an outline of his life in minuscule, barely legible, handwriting. His most intimate thoughts were recorded in code and then destroyed.

Galton held it amongst his scientific opinions that Jews were "specialized for a parasitical existence" and across the world such views were widely respected by scientific and governmental "authorities" of Galton's day. Galton's protégés certainly held and promoted similar "scientific" opinion.

Karl Pearson held it as unimpeachable fact, based on his eugenical knowledge, that superior persons of superior races must be strongly influenced to fecundity, including all possible influence of government policy and law, and that inferior races are better eliminated than merely subjugated, with war being a noble and highly desirable pursuit for proud races. From the 1905 printed edition of his lecture entitled "National Life from the Standpoint of Science":

> ... [N]o community of men can trust blindly to heredity to preserve their racial characters. Every nation is an agglomeration of good and bad elements, and each new generation is born from a relatively small portion of the whole. The greatness of a nation depends on the dominant fertility of its fitter stocks, and fluctuates with the extent of this dominance. Love of ease, a mistaken sense of duty, insidious new social habits, may tamper with the preponderating fertility

of the fitter and more capable racial constituents before we have realized their effects. Some only of these things can be touched by the legislator; in the aggregate they are subject alone to social feeling and to an enlightened national pride. Is it possible to arouse a consciousness in the folk that the parentage of the next generation is not a personal but a national problem? – that a nation which has ceased to insure that its better elements have a dominant fertility has destroyed itself far more effectually than its foes could ever hope to destroy it in the battlefield?

... What I have said about bad stock seems to me to hold for the lower races of man. How many centuries, how many thousands of years, have the Kaffir or the negro held large districts in Africa undisturbed by the white man? Yet their intertribal struggles have not yet produced a civilization in the least comparable with the Aryan's. Educate and nurture them as you will, I do not believe that you will succeed in modifying the stock. History shows me one way, and one way only, in which a high state of civilization has been produced, namely, the struggle of race with race, and the survival of the physically and mentally fitter race.

... Let us suppose we could prevent the white man, if we liked, from going to lands of which the agricultural and mineral resources are not worked to the full; then I should say a thousand times better for him that he should not go than that he should settle down and live alongside the inferior race. The only healthy alternative is that he should go and completely drive out the inferior race. That is practically what the white man has done in North America.

... But I venture to say that no man calmly judging will wish either that the whites had never gone to America, or would desire that whites and Red Indians were to-day living alongside each other as negro and white in the Southern States, as Kaffir and European in South Africa, still less that they had mixed their blood as Spaniard and Indian in South America.

... I venture to assert, then, that the struggle for existence between white and red man, painful and even terrible as it was in its details, has given us a good far outbalancing its immediate evil. In place of the red man, contributing practically nothing to the work and thought of the world, we have a great nation, mistress of many arts, and able, with its youthful imagination and fresh, untrammelled impulses, to contribute much to the common stock of civilized man.

> ... You will see that my view – and I think it may be called the scientific view of a nation – is that of an organized whole, kept up to a high pitch of internal efficiency by insuring that its numbers are substantially recruited from the better stocks, and kept up to a high pitch of external efficiency by contest, chiefly by way of war with inferior races, and with equal races by the struggle for trade-routes and for the sources of raw material and of food supply.

Ronald Aylmer Fisher, also an ardent racist who insisted that "scientific knowledge provides a firm basis for believing that the groups of mankind differ in their innate capacity for intellectual and emotional development", spoke of the needs to suppress societal inferiors and to increase fecundity of a superior elite toward creating a race of supermen (i.e. of persons as similar as possible to Ronald Aylmer Fisher.) From his 1914 *Eugenics Review* article "Some Hopes of a Eugenicist":

> From the moment that we grasp, firmly and completely, Darwin's theory of evolution, we begin to realise that we have obtained not merely a description of the past, or an explanation of the present, but a veritable key of the future ...
>
> At the present time in this country the evidence appears to be conclusive that we are breeding more from the worse than from the better stocks ...
>
> ... We do not dub ourselves knights of a new order. But necessarily, inevitably, it might be unconsciously, we are the agents of a new phase of evolution. Eugenicists will, on the whole, marry better than other people – higher ability, richer health, greater beauty. They will, on the whole, have more children than other people. Their biological type, characterised by their solicitude for human betterment, their scientific insight, above all their intense appreciation of human excellence, has a strong tendency to improve and to survive [as] a new natural nobility of worth and birth.

The progenitors of biostatistics were philosophically and morally blind. Today's "lifestyle epidemiology" is nothing more or less than the modern incarnation of eugenics, in its techniques, and in its basic intention: the same old end of creating miserable social division, of pitting an elite against inferiors defined by the elite. Francis Galton expressed a hope that eugenics would become "a new religion", and so it has, as a debased cult belief.

The "statistics madness" Vincent-Riccardo DiPierri describes was evident from the very beginning, in Francis Galton, the mad creator of eugenics. The name of eugenics has been buried, but the methods of eugenics, and its spirit of hate, persist in the debased practices

of today's "health" establishment.

So it goes on as before: the crazed statistics and propaganda, the disturbed narcissistic psychology and its viciousness toward others, the contempt for social cohesion in preference for a righteous war of the worthy against the inferior and threatening others, the haughtiness, the self-superiority, the insistence of infallibility based on anti-science.

How can the "authorities" be so foolish, so hateful, and blind? Because too many amongst the public have been so foolish as to permit this. The situation *can* be changed. But it never *will* be changed until *force* is brought to bear against empowered and very hateful fanatics.

Appendix 1

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Appendix 2

Original questionnaires used for the Lam 1985 and Boffetta 1998 ETS studies

FIG. 7.1. LUNG CANCER QUESTIONNAIRE

Name: _____ Sex/Age: _____ Date _____

Address (District): _____ How Long? _____ Dx: _____

Born in ☐ Hong Kong ☐ China In Hong Kong for ___yrs. Dialect Gp. _____

Occupation: _____ for _____ yrs. Schooling ☐ ≤ 6 yr. ☐ > 6 yr.

Marital Status: ☐ Single ☐ Married ☐ Widowed _____ yrs.

Husband: occupation _____

SMOKING: ☐ non-smoker ☐ ex-smoker _____ yrs. ☐ smoker

☐ cigarettes ☐ hand-rolled ☐ water-pipe ____/day x ____ yrs.

PASSIVE SMOKING:

Home: Size of home: _____ No. family members: _____

	Husband	Others		Father	Mother
Non-smoker	☐	☐	☐	☐	☐
Smoker: Amount	____/day	____/day	____/day	____/day	____/day
Exposure/d	____ hr.	____/hr.	____/hr.	____/hr.	____/hr.
Duration	____ yr.	____/yr.	____/yr.	____/yr.	____/yr.

AT WORK: Size of working place _____ No. of smoker workmates _____

Exposure time/day _____ hr. for _____ yrs.

DOMESTIC COOKING: ☐ Never/seldom cooks

☐ Cooks regularly

☐ Kerosene _____ yrs. ☐ Electricity _____ yrs.

☐ Coal gas _____ yrs. ☐ Gas _____ yrs. ☐ wood ___ yrs.

Chinese pan cooking ☐ yes ☐ no ____ times/wk for ____ yrs.

HOME INCENSE BURNING: ☐ Yes ☐ No for _____ yrs.

☐ Daily ☐ Festivals only

☐ Inside house ☐ Outside house

HISTORY OF PULMONARY TB: ☐ Yes ☐ No

Chest x-ray: (No.)

INTERNATIONAL AGENCY FOR RESEARCH ON CANCER
WORLD HEALTH ORGANIZATION

European Multicentre Case-Control Study of Lung Cancer in Non-smokers

Detailed Results on Exposure to Environmental Tobacco Smoke

Paolo Boffetta, Antonio Agudo, Wolfgang Ahrens, Ellen Benhamou, Simone Benhamou, Sarah C. Darby, Gilles Ferro, Cristina Fortes, Carlos A. Gonzalez, Karl-Heinz Jöckel, Martin Krauss, Lothar Kreienbrock, Michaela Kreuzer, Anabela Mendes, Franco Merletti, Fredrik Nyberg, Göran Pershagen, Hermann Pohlabeln, Elio Riboli, Giovanni Schmid, Lorenzo Simonato, Jean Trédaniel, Elise Whitley, Heinz-Erich Wichmann, Carlos Winck, Paola Zambon, Rodolfo Saracci

IARC Technical Report No. 33

Lyon, 1998

Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

Tobacco smoking

- **Did you ever smoke for longer than one year?**

  Yes ..1; No ..2

  1 or more cigarettes/day ☐
  or half a pack of cigarettes/week ☐
  or 2 or more packs/month ☐
  or 4 or more cigarillos/week ☐
  or 3 or more cigars/week ☐
  or 3 or more pipes/week ☐

  if YES to at least one of the questions, go to "Active smoking" section
  if NO, continue with the next question

- **Did you ever try to smoke?** Yes ..1; No ..2 ☐

- **Have you ever smoked cigarettes, cigar or pipe, even very few occasionally during a social occasion and/or at a particular period of your life?** Yes ..1; No ..2 ☐

If yes, from what age did you smoke occasionally?		**What did you smoke?**			**Frequency**
From	**To**	Cigarettes Filter ..... 1 Non filter ..... 2 Cigar ..... 3 Pipe ..... 4 Cigarette & pipe 5	**If during this period you smoked mainly the same brand, indicate the brand** *(otherwise skip)*	**How many?**	Per day ..... 1 Per week ..... 2 Per month ..... 3 Per year ..... 4 Per lifetime ... 5
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- **Did you inhale tobacco smoke?** ☐

  Not at all, only in the mouth 1
  A little, just in the throat 2
  Deeply into the lung 3

Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

ETS during childhood and until subject left parents' home

- **Going back to your childhood, I would like to know if you ever lived with any person who used to smoke in your presence. First of all, I would like to know whether your father or your mother used to smoke when you were a child.**

if the subject is aware that one or both parents smoked since his/her birth, starting age=0.

Relationship	**What did he/she smoke?** Cigarettes..... 1 Cigars..... 2 Pipe..... 3 Cigarette & pipe ..... 4 Don't remember ..... 8 Non-smoker..... 9	**Did he/she smoke in your presence?** Every day .....1 Almost every day .2 *(5-6 days/week)* 2-4 days/week.....3 Rarely.....4 Never.....5	**From age**	**To age**
Father	☐	☐	☐☐	☐☐
Mother	☐	☐	☐☐	☐☐

- **Now I would like to know about any other person who lived in your home or with whom you lived for longer than six months or who was a very regular visitor to your home, who used to smoke in your presence (brother, sister, other relatives, friends, visitors, etc...).**

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- **I would like you to try to remember how long you used to spend in a smoky room (outside the home, including school but excluding the workplace and vehicles).**

From age	**To age**	**How many hours in a room with tobacco smoke?**	**Frequency** Per day.....1 Per week .....2 Per month.....3	**Would you say that the room was usually:** Very smoky .....1 *(you could see clouds of smoke in the air)* Fairly smoky .....2 *(you could see diffuse smoke in the air)* A little smoky .....3 *(you could only smell the smoke)*
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- **At what age did you leave your parents' home?** *(still leaving there =99)* ☐☐

Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

ETS from the spouse

The person with whom the subject lives/lived in a marital type relationship.

- **Are you or have you ever been married or living with someone in a marital type relationship?**
  Yes ..1; No ..2 ☐

  If the subject has been married more than once, start with the first marriage and add additional lines for subsequent marriages.

- **Did your first spouse ever smoke?** Yes ..1; No ..2 ☐

  if NO, go to next spouse or next page.

- **Starting with your marriage (or the time you lived in marital status), I would like to know how much and how often you were exposed to his/her tobacco smoke at home, in vehicles or anywhere else indoors. I would also like to know whether the smoking habits of your spouse ever changed.**

  Two types of changes need probing:
  1. *Change in spouse smoking habits: Type of product, amount smoked, change to non filter/filter.*
  2. *Change in subject's exposure to spouse: Moving house, spouse away from home.*

  Use more than one line when spouse smoked more than one tobacco product in the same period.
  Start on first line with subject's age when he/she was first exposed.

Period		**What did he/ she smoke?**	**How many cigarettes (cigars, pipes) smoked per day?**	**Did he/she smoke in your presence?**	**How many cigarettes (cigars, pipes) did he/she smoke in your presence on average?**		**For how many hours/day were you exposed to tobacco smoke from your spouse on average?**	
From age (years)	**To age (years)**	Cigarettes............1 Cigars..................2 Pipe.....................3 Cigarette & pipe ..4		Every day.............1 Almost every day ..2 *(5-6 days/week)* 2-4 days/week.......3 Rarely ....4 Never 5	Weekdays	Holidays Weekends	Weekdays	Holidays Weekends
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Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

ETS from other cohabitants

Smokers who lived with you in the same house or visited your house regularly, other than your spouse.

Use more than one line for subjects who smoked more than one tobacco product in the same period.

	Period: From age (years)	Period: To age (years)	What did he/she smoke? Cigarettes .... 1, Cigars .... 2, Pipe .... 3, Cigarette & pipe.. 4	Did he/she smoke in your presence? Every day .... 1, Almost every day .... 2 *(5-6 days/week)*, 2-4 days/week .... 3, Rarely .... 4, Never 5	How many cigarettes (cigars, pipes) did he/she smoke in your presence on average? Weekdays	How many cigarettes (cigars, pipes) did he/she smoke in your presence on average? Holidays Weekends	For how many hours/day were you exposed to tobacco smoke from him/her on average? Weekdays	For how many hours/day were you exposed to tobacco smoke from him/her on average? Holidays Weekends
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Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

ETS in vehicles

- **Have you ever (lifetime) traveled daily or at least a couple of times per week by car, train, bus or another enclosed vehicle which was smoky (or where you could at least smell tobacco smoke) most of the time?**
  Yes ..1; No ..2 ☐

if NO, go to next page.
if YES, please state from what age to what age, on which type of vehicle and for how many hours per day or per week you were exposed to tobacco smoke.

Period From age (years)	 To age (years)	Type of vehicle Car.......................1 Train ....2 Bus/tram ....3 Other (specify).....4	How many hours per day or week were you in this vehicle while you were exposed to smoke? Per day	 Per week	Would you say that the vehicle was usually: Very smoky...... 1 Fairly smoky .... 2 A little smoky ... 3
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Exposure already covered, e.g. occupational and from spouse should be excluded. Exposure during daily commuting to work should be reported in this section.

Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

ETS indoors

In places other than the home, work premises or vehicles e.g. restaurants, bars, pubs, cinemas, theatres, friends' homes, etc.

- **I would like to know if you have ever spent regularly (at least once a week) some time in a smoky place (or where you could at least smell tobacco smoke) indoors other than at home or at work.**
  Yes ..1; No ..2 ☐

If NO, go to next page.

Specify where exposure took place	Period: From age (years)	Period: To age (years)	How many hours per week were you exposed to tobacco smoke in this place?	Would you say that the place was usually: Very smoky .... 1; Fairly smoky .... 2; A little smoky 3
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Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

ETS at the workplace

- **I would like to know if you ever worked in an indoor place where you were exposed to tobacco smoke.**
  Yes ..1; No ..2 ☐

If NO, go to next page.
If YES, fill in one line for each job in which the subject was exposed to ETS.

Job number (refer to job history)	Exposure to ETS From age (years)	Exposure to ETS To age (years)	Would you say that the place was usually: Very smoky.....1 Fairly smoky ...2 A little smoky ..3	How many hours/day on average were you exposed to tobacco smoke at work, including time spent at the canteen or during breaks?	How many days per week were you exposed to tobacco smoke at work?	Approximately how large was the room? Small (under 40 m^2) ...1 Medium (40-80 m^2).....2 Large (80-200m^2) ....3 Very large (>200 m^2) ..4
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Questionnaire on occasional smoking and environmental tobacco smoke (ETS)

Residential history

- **I would like to know about all houses/residences where you lived for longer than two years, starting with the home where you were born.**

Period: From age (years)	Period: To age (years)	Town, village	County *(if abroad, the country)*	In your opinion, is/was it a rural or urban area? Rural....1 Urban...2	What is/was the principal means of cooking? Gas....1 Electricity....2 Coal stove....3 Wood stove....4 Other (specify)..5	What is/was the principal means of heating? (no more than 2 methods for each residence) Central heating*....1 Gas stove or similar....2 Wood stove....3 Coal stove....4 Kerosene or other fuel.5 Other (specify)....6
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Central heating: a system for which there is no combustion unit in the living or sleeping area of the house.

WEIGHTS

Tobacco product (WT1)

Cigarette	= 1
Cigarette, filter	= 1
Cigarette, non filter	= 1
Cigar	= 3
Pipe	= 3
Cigarette & pipe	= 1
Cigarette & cigar	= 1
Cigar & pipe	= 4
Cigarette & cigar & pipe	= 1
Cigarillo	= 2
Cigarette & cigarillo	= 1
Pipe & cigarillo	= 4
Cigar & cigarillo	= 3
Pipe & cigar & cigarillo	= 4
Cigarette & cigar & cigarillo	= 1
Cigarette & pipe & cigarillo	= 1
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Cigarettes consumption frequency (WT2)

Every day	= 1
5-6 days/week	= 0.75
2-4 days/week	= 0.5
Rarely	= 0.1

Level of smokyness (WT3)

Very smoky	= 1
Fairly smoky	= 0.5
A little smoky	= 0.2

Consumption frequency (WT4)

Per day	= 365
Per week	= 52
Per month	= 12
Per year	= 1

Smoker equivalents (WTSE)

Each single relative (Father, mother, other)	= 1

Smoking parent equivalents (WTSPE)

Mother	= 1
Father	= 0.75
Other relatives	= 0.25

OCCASIONAL SMOKING

OSTR **Ever tried to smoke**

OSOC **Ever smoked occasionally**

OSUD **Duration (years)**

$$= \sum_{1}^{n} \Delta age \text{ , } non\ overlapping$$

OSCU **Cumulative exposure (packs/lifetime)**

$$= \left(\sum_{1}^{n} \left(\Delta age \times WT1 \times WT4 \times No.smoked \right) \right) / 20$$

$$= \left(\sum_{1}^{n} \left(WT1 \times No.smoked \right) \right) / 20 \text{ , } when\ WT4 = Lifetime$$

ETS during CHILDHOOD

Childhood 0-18 years

C1EV **Ever exposed**

C1EM **Ever exposed to mother**

C1EF **Ever exposed to father**

C1TP **Exposure to different tobacco products**

C1UN **Number of smokers**

$$= \sum_{1}^{n} WTSE$$

C1WN **Weighted number of smokers**

$$= \sum_{1}^{n} WTSPE$$

C1US **Smoking parent years**

$$= \sum_{1}^{n} (\Delta age \times WTSE)$$

C1WS **Weighted smoking parent years**

$$= \sum_{1}^{n} (\Delta age \times WTSPE)$$

C1UD **Duration (years)**

$$= \sum_{1}^{n} \Delta age \text{ , } non\ overlapping$$

C1WD **Weighted duration (years)**

$$= \sum_{1}^{n} (\Delta age \times WT2)$$

ETS from SPOUSE

S1EV **Ever exposed**

S1TP **Exposure to different tobacco products**

S1UD **Duration (years)**

$$= \sum_{1}^{n} \Delta age \text{ , non overlapping}$$

S1DH **Weighted duration (hours/day × years)**

$$= \sum_{1}^{n} \left(\Delta age \times WT2 \times \frac{(5 \times hours/WD) + (2 \times hours/WE)}{7} \right)$$

S1CU **Cumulative exposure (packs/day × years)**

$$= \left(\sum_{1}^{n} \left(\Delta age \times WT1 \times WT2 \times \frac{(5 \times amount/WD) + (2 \times amount/WE)}{7} \right) \right) / 20$$

S1AV **Average exposure (cigarettes/day)**

$= 20 \times (Cumulative\ exposure/Duration)$

S1CS **Cumulative exposure of smoking spouse (packs/day × years)**

$$= \left(\sum_{1}^{n} (\Delta age \times WT1 \times amount/day) \right) / 20$$

S1TS **Time since last ETS exposure (years)**

WD=Weekday
WE=Weekend

ETS from SPOUSE and OTHER COHABITANTS

S3EV **Ever exposed**

S3TP **Exposure to different tobacco products**

S3UD **Duration (years)**

$$= \sum_{1}^{n} \Delta age \text{ , } non\ overlapping$$

S3CU **Cumulative exposure (packs/day × years)**

$$= \left(\sum_{1}^{n} \left(\Delta age \times WT1 \times WT2 \times \frac{(5 \times amountWD) + (2 \times amountWE)}{7} \right) \right) / 20$$

S3AV **Average exposure (cigarettes/day)**

$= 20 \times (Cumulative\ exposure/Duration)$

S3TS **Time since last ETS exposure (years)**

WD=Weekday
WE=Weekend

ETS from WORKPLACE

WOEV **Ever exposed**

WOUD **Duration (years)**

$$= \sum_{1}^{n} \Delta age \text{ , } non\ overlapping$$

WOH **Duration (hours)**

$$= \sum_{1}^{n} \left(\Delta age \times 52 \times \left(hours / day\right) \times \left(days / week\right)\right), \ non\ overlapping$$

WOWD **Weighted duration (hours × level)**

$$= \sum_{1}^{n} \left(\Delta age \times 52 \times WT3 \times \left(hours / day\right) \times \left(days / week\right)\right)$$

WOTS **Time since last ETS exposure (years)**

ETS from SPOUSE and WORKPLACE

SWEV **Ever exposed**

SWUD **Duration (years)**

$$= \sum_{1}^{n} \Delta age \text{ , } non\ overlapping$$

SWDH **Weighted duration (hours/day × years)**

$$= \sum_{1}^{n} \left(\Delta age \times WT2 \times \frac{(5 \times hours/WD) + (2 \times hours/WE)}{7} \right)$$

SWTS **Time since last ETS exposure (years)**

WD=Weekday
WE=Weekend

ETS from VEHICLES

VEEV **Ever exposed**

VEUD **Duration (years)**

$$= \sum_{1}^{n} \Delta age \text{ , } non\ overlapping$$

VEWD **Weighted duration (days × level)**

$$= \sum_{1}^{n} \left(\Delta age \times 365 \times WT3 \times \frac{hours\ /\ day}{24} \right)$$

ETS from OTHER INDOOR SETTINGS

INEV	**Ever exposed**
INUD	**Duration (years)** $= \sum_{1}^{n} \Delta age$, *non overlapping*
INWD	**Weighted duration (days × level)** $= \sum_{1}^{n} \left(\Delta age \times 365 \times WT3 \times \frac{hours/day}{24} \right)$

Appendix 3

The Schoenberg 1989 Never Smoker ETS / Lung Cancer Study

A CASE-CONTROL STUDY OF RADON AND LUNG CANCER AMONG NEW JERSEY WOMEN

TECHNICAL REPORT - PHASE I

AUGUST, 1989

NEW JERSEY STATE DEPARTMENT OF HEALTH

DIVISION OF EPIDEMIOLOGY AND DISEASE CONTROL
DIVISION OF OCCUPATIONAL AND ENVIRONMENTAL HEALTH

THOMAS H. KEAN
GOVERNOR

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EXECUTIVE SUMMARY

The New Jersey State Department of Health (NJDOH) has been conducting an epidemiologic study of radon and lung cancer in New Jersey women. This study focuses on the questions of whether and to what extent radon in homes is associated with increased lung cancer risk. The importance of this question arose from the 1985 finding of very high levels of radon, a known cause of lung cancer among underground miners, in some Eastern Pennsylvania residences.

The New Jersey research reported here is the first large-scale epidemiological study of radon and lung cancer based on actual measurements in homes and detailed smoking histories for individual subjects. It is an extension of a case-control study of lung cancer which previously had been conducted among New Jersey women. The cases in that study were women newly diagnosed with lung cancer from August 1982 through September 1983, while the controls were women without lung cancer but similar in age and race to the cases. Information on smoking, residential, occupational and dietary histories was collected for 994 cases and 995 controls.

The radon substudy initially focused on those New Jersey dwellings which met a residence criterion, i.e., where subjects had lived the longest and for at least 10 years during the period from 10-30 years prior to lung cancer diagnosis or control selection. Both long-term and short-term radon measurements were made in these houses. Radon exposures for subjects were estimated by year-long alpha track detector measurements in the living areas. Four-day measurements of radon were made using charcoal canisters in basements to provide quick screening measurements for current residents, in case radon levels were so high that immediate remediation was needed, and to provide back-up data in case year-long measurements of radon were not completed.

This report is based on radon exposure data from 433 cases and 402 controls. Some of the original cases and controls were not included in the radon substudy because address-specific information could not be collected, because no house met the

residence criterion, or because radon tests could not be conducted at a house which did meet this criterion.

The overall distribution of radon exposure was generally low: only 24 cases (5.6%) and 12 controls (3.0%) had year-round living area radon concentrations of 2 pCi/L or greater. After smoking, age and occupation were taken into account, the estimated lung cancer risk for those exposed to the highest radon category (2-11 pCi/L) was 80% greater than the risk for those at the lowest exposure level (less than 1.0 pCi/L). Because the number of subjects in the higher exposure category was small, however, the relative risk estimate was not statistically significant. In contrast, the trend for increasing risk with increasing radon exposure was statistically significant; the probability that this trend was due to chance alone was only 4%.

When duration of exposure was also taken into account, similar patterns of increasing risk with increasing cumulative radon exposure were seen. The estimated lung cancer risk for those exposed to the highest cumulative radon category (50-155 pCi/L-years) was 40% greater than the risk for those at the lowest exposure level (less than 25 pCi/L-years). Furthermore, the increase in lung cancer risk over background risk per unit of cumulative exposure was consistent with that generally found in the studies of underground miners.

Study analyses also showed that lung cancer risk for women who smoked about one pack a day was 1,000% greater than risk for lifetime nonsmokers. This again confirmed that **smoking is the major cause of lung cancer.**

Some of the results of this study must be interpreted cautiously because of the small number of subjects in the highest radon exposure categories. Extensive data analyses and discussion throughout the technical report and its appendices are designed to consider the extent of any possible biases introduced by reduction of the potential study population to those with actual radon exposure estimates.

Nevertheless, the study suggests that the findings of radon-related lung cancer in miners can be applied to the residential setting. Excess radon exposures typical of homes may increase risk of lung cancer; extremely high residential exposures would be associated with very serious lung cancer risks. These results support the comprehensive interdepartmental radon-related effort initiated in 1985 by the NJDOH and the New Jersey Department of Environmental Protection, including provision of technical information and services, citizen education, and research activities. The study also confirms that smoking avoidance education should be strongly emphasized along with radon reduction activities.

The exposure data yielded by this study also suggest that the relationship between screening measurements and year-round living area measurements need better characterization for public policy purposes and clearer understanding by the public before remediation decisions are made. In addition, building code modification to prevent radon entry may be an effective means for reducing overall population risks from radon exposure.

Further data analyses may refine the results of this study. A second, still ongoing phase of data collection will add more subjects to the substudy, and will result in more complete exposure histories from additional houses for those subjects already included.

The findings of this study also need to be corroborated by other residential radon studies currently underway worldwide. In the meantime, existing actions to reduce radon exposure to the lowest feasible levels should be maintained. Remedial action should be taken in residences when follow-up testing indicates that typical exposures of occupants are above 4 pCi/L. This recommendation is not based upon the absence of any risk below 4 pCi/L; rather, it is based upon the limited feasibility of remediating residences below that level.

A CASE-CONTROL STUDY OF RADON AND LUNG CANCER AMONG NEW JERSEY WOMEN

TECHNICAL REPORT, PHASE I - AUGUST, 1989
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This report on the New Jersey Radon-Female Lung Cancer Case-Control Study was written by Janet Schoenberg (NJDOH, Division of Epidemiology and Disease Control) and by Judith Klotz (NJDOH, Division of Occupational and Environmental Health). Other persons who made important contributions to this study include Homer Wilcox, Maria Gil-del-Real, and Annette Stemhagen (NJDOH, Division of Epidemiology and Disease Control), Gerald Nicholls and Mary Cahill (NJDEP), Zdenek Hrubec (National Cancer Institute, Radiation Epidemiology Branch), and Thomas Mason (National Cancer Institute, Environmental Epidemiology Branch).

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NEW JERSEY RADON STUDY - METHODS

This radon study is an extension of a statewide population-based incident case-control interview study of lung cancer previously conducted among New Jersey women (Schoenberg et al., 1989; see Appendix B).

Original subjects: selection and data collection.

The original study cases included all female New Jersey residents who were newly diagnosed with histologically confirmed primary cancer of the lung from August 1982 through September 1983. For cases who were interviewed themselves, controls (frequency matched to cases by 5-year age groups and race) were selected during the same time period from New Jersey drivers' license files (age <65) and from Health Care Financing Administration files of persons enrolled for Medicare (age 65+). For cases with next of kin interviews, individually matched controls were selected from state death certificate files.

During the original study, personal interviews were completed for 994 (76.1%) of the 1,306 cases identified and for 995 (68.7%) of the 1,449 controls identified. Details on the reasons for nonresponse are summarized in Appendix B. Fifty-three percent of the interviews were conducted with the subjects themselves. The remaining interviews were conducted with the spouse (17%) or with other next of kin (30%), mainly daughters, sons and sisters.

The questionnaire included a lifetime brand-specific smoking history, information on smoking habits of other household members, lifetime residential and occupational histories, and a history of consumption of foods containing vitamin A. These data have been analysed in some detail, specifically with respect to lung cancer risk associated with active and passive smoking, occupational exposure, and diet (vegetable consumption), after adjusting for age, race, respondent type, education, and county of

RESULTS

Inclusion in the radon study.

The status of the original 994 female lung cancer cases and 995 controls in the current radon substudy was examined (Table 3) in order to determine the extent of any possible bias in the composition of the radon substudy population relative to the original study population. Cases and controls did not differ significantly with respect to their status in the substudy. However, slightly more controls did not have measurements at the index residences, particularly due to refusal by the current occupant to participate. More often than for cases, original control subjects were still the occupants of the index residences. Some of these controls, who had already spent considerable time being interviewed, did not want any further involvement with the study.

Analyses in this radon study include the 411 cases and 385 controls whose index residence was successfully tested for radon with alpha track detectors and/or charcoal canisters. In addition, most analyses also include the 22 cases and 17 controls whose index residence was an apartment above the second floor or a trailer, for whom radon exposures were estimated (see Appendix J). Therefore, a total of 835 subjects (433 cases, 402 controls) are included in the radon study.

Table 4 shows the distribution of these subjects by age, respondent type and race, and by active smoking (lifetime average number of cigarettes per day, total years smoked, years since smoking cessation, tar content of cigarettes smoked from 1973-1982), vegetable consumption, occupation, education, county of residence at diagnosis, and passive smoking (for non-smokers only: exposure to spouse tobacco smoke). These variables were considered as possible confounders in the analysis of any association between radon exposure and lung cancer. Odds ratios which were estimated for these variables in the original female lung cancer study are summarized in Appendix B. These

analyses confirm the importance of smoking as the most significant risk factor for female lung cancer (particularly, number of cigarettes per day and years since smoking cessation). In addition, age, occupation, vegetable consumption, and respondent type (i.e., differences in smoking-related odds ratios by respondent type) also contribute signficantly to the observed lung cancer risk.

More detailed analyses on the characteristics of those women included in the radon substudy, compared to those women not included, are presented in Appendix C. Overall, controlling for all the potential confounders, there was significantly greater participation for cases from the original study than for controls. Moreoever, radon study subjects were more often older, whites, either nonsmokers, light smokers, or ex-smokers, residents of counties with higher radon levels, and more highly educated. However, there were relatively few significant case-control differences between those included and not included, except among heavy smokers, who showed some unusual risk factor distributions (Appendix C).

Type of measurement results.

One or more alpha track measurements were completed for 719 (90%) of the 796 index residences tested in the radon study. One or more charcoal canister measurements were obtained for 788 (99%) addresses. The canister measurements were not used for analyses, except to estimate the year-round living area radon concentration when no alpha track measurements were completed (see below, p. 17).

The charcoal canister results were also used to determine how the sample of houses included in this study compared to other New Jersey houses. In Appendix G, the distribution of basement or lower floor charcoal canister measurements from the 788 houses tested in this study has been compared, by county, to the distribution of basement or lower floor charcoal canister measurements obtained in a statewide survey of 5,727 homes conducted for the NJDEP. There was relatively good agreement

"other histologic types" at 1-1.9 pCi/L (OR=2.1, 90% CI = 1.2, 3.8), but insignificant at higher radon levels.

Passive smoking. Analyses were also conducted taking into account passive smoking exposure by lifetime nonsmokers (see Appendix B for background from the orginal case control study). Table 13 shows the distribution of the 274 nonsmokers (61 cases, 213 controls) by year-round living area radon concentrations, as well as by exposure to spouse tobacco smoke (no exposure, exposure to spouse cigarette smoke, exposure to spouse tobacco smoke only from pipes and cigars). Adjusting for exposure to spouse tobacco smoke had very little effect on the odds ratios estimated for radon exposure among nonsmokers, or on the trend statistic in this subgroup.

Similar analyses (not shown) were also conducted considering exposure to tobacco smoke from any household member, not just the spouse. Adjusting for any household tobacco exposure had no effect on the odds ratios estimated for radon among nonsmokers. Therefore, neither of the nonsmoker-passive smoking exposure variables were considered in the overall model for all subjects.

Analyses of cumulative radon exposures.

All of the analyses described above have considered only the radon concentration measured in the living area of the index residence. The number of years of residence at the index address had not yet been taken into account. A **cumulative exposure index multiplies the radon concentration by years of residence.** In the development of the cumulative exposure index used in these anslyses below, several assumptions have been made:

(1) A minimum period of five years since relevant radon exposure has been assumed, rather than ten years, making the exposure period of interest the years from 5-30 years prior to case diagnosis or control selection. This assumption is based on the

TABLE 13

Distribution of lung cancer cases and controls (LIFETIME NONSMOKERS ONLY), by radon level (year-long living area alpha track measurements, n=221; estimates, n=53) and by exposure to spouse tobacco smoke

New Jersey radon-female lung cancer case-control study, 1982-1988

	Radon (pCi/L)				
Passive smoking status	<1.0 [a]	1-1.9	2-3.9	4-11.3	Total
No exposure					
Cases	17	0	1	0	18
Controls	54	15	1	0	70
Exposure to spouse cigarette smoke					
Cases	29	8	0	1	38
Controls	96	17	2	1	116
Exposure to spouse tobacco smoke (pipes/ cigars only)					
Cases	2	3	0	0	5
Controls	18	7	2	0	27
Total nonsmokers					
Cases	48	11	1	1	61
Controls	168	39	5	1	213
Unadjusted OR	1.0	0.99 (0.53,1.8)	0.70 (0.11,4.3)	3.5 [c] (0.34,36.4)	
Adjusted OR b	1.0	1.0 (0.55,1.9)	0.79 (0.13,5.0)	3.1 [d] (0.30,32.7)	

[a] Includes subjects whose index address was an apartment above the second floor or a trailer.

[b] Adjusted by exposure to spouse tobacco smoke (no exposure, exposed to spouse cigarettes, exposed to spouse pipes/cigars only)

[c] OR for 2+ pCi/L: 1.2 (0.30,4.6); trend (Zcat) = 0.43, p = 0.33.
[d] OR for 2+ pCi/L: 1.2 (0.31,5.0); trend (Zcat) = 0.51, p = 0.32.

TABLE 14

Distribution of lung cancer cases and controls by cumulative radon exposure[a] and by lifetime average daily cigarette consumption, New Jersey radon-female lung cancer case-control study, 1982-1988

Smoking status	Cumulative radon (pCi/L-years) <25	25-49	50-99	100-155	Total
Nonsmokers					
Cases	52 (85.2%)	8 (13.1%)	0 (-)	1 (1.6%)	61
Controls	175 (82.2%)	33 (15.5%)	4 (1.9%)	1 (0.5%)	213
Unadjusted OR	**1.0**[b]	**0.82**	**0.0**	**3.4**	**1.0**[c]
<15 cigs/day					
Cases	64 (77.1%)	14 (16.9%)	3 (3.6%)	2 (2.4%)	83
Controls	82 (91.1%)	8 (8.9%)	0 (-)	0 (-)	90
Unadjusted OR	**2.6**	**5.9**	**∞**	**∞**	**3.2**
15-24 cigs/day					
Cases	146 (82.0%)	24 (13.5%)	8 (4.5%)	0 (-)	178
Controls	58 (86.6%)	7 (10.4%)	2 (3.0%)	0 (-)	67
Unadjusted OR	**8.5**	**11.5**	**13.5**	**—**	**9.3**
25+ cigs/day					
Cases	99 (89.2%)	10 (9.0%)	1 (0.9%)	1 (0.9%)	111
Controls	25 (78.1%)	4 (12.5%)	3 (9.4%)	0 (-)	32
Unadjusted OR	**13.3**	**8.4**	**1.1**	**∞**	**12.1**
Total					
Cases	361 (83.4%)	56 (12.9%)	12 (2.8%)	4 (0.9%)	433
Controls	340 (84.6%)	52 (12.9%)	9 (2.2%)	1 (0.2%)	402
Unadjusted OR	**1.0**[d]	**1.0**	**1.3**	**3.8**	

[a] Cumulative radon exposure during 25 years from 5-30 years prior to case diagnosis or control selection; assumes exposure of 0.6 pCi/L (median for controls) for any of the 25 years during which the subject did not live in the index address where the measurements were made.

[b] Unadjusted odds ratio (an estimate of the lung cancer risk associated with radon exposure and smoking, but not adjusted for any other factors), relative to nonsmokers with < 25.0 pCi/L-years cumulative radon exposure.

[c] Unadjusted odds ratio (an estimate of the lung cancer risk associated with smoking, but not adjusted for radon exposure or any other factors), relative to lifetime nonsmokers.

[d] Unadjusted odds ratio (an estimate of the lung cancer risk associated with cumulative radon exposure, but not adjusted for smoking or any other factors), relative to subjects with < 25.0 pCi/L-years cumulative radon exposure.

APPENDIX B

Original NJ Female Lung Cancer Case-Control Study: Methods

Cases included all female state residents who were newly diagnosed with histologically confirmed primary cancer of the lung, trachea or bronchus (code 162, International Classification of Diseases, 9th Revision) from August 1982 through September 1983. They were ascertained through a rapid reporting system that the NJ State Department of Health (NJDOH) established with local hospital pathology departments, and by periodic review of hospital pathology records and of State Cancer Registry and death certificate files. Pathology reports, from which the histologic type was determined, and other medical records were reviewed by NJDOH physicians to verify the diagnosis. No slide review was conducted. Some adenocarcinoma cases were designated as "probably in-scope," rather than "in-scope," if there was insufficient documentation to rule out completely the possibility of another primary site, particularly breast cancer.

Population-based controls were selected using one of three files. For cases who were themselves interviewed, controls were selected using a random sample of either New Jersey drivers' license files (for ages less than 65) or Health Care Financing Administration Files (for ages 65 or older), and were frequency matched to the cases within race and 5-year age groups. For deceased or incapacitated cases, with next-of-kin respondents, New Jersey State mortality files were used to select controls who were individually matched to the cases by race, age, and closest date of death (or date of death closest to date of diagnosis, for incapacitated cases). Controls selected from mortality files were excluded if lung cancer or any other respiratory disease was mentioned on the death certificate.

Subjects or their next-of-kin were personally interviewed in their homes by trained interviewers. Questionnaire items included demographic data, a detailed

brand-specific smoking history, a history of passive exposure to smoking by other household members, a dietary history to determine consumption of foods containing vitamin A, and lifetime residential and occupational histories.

In the cigarette use section of the questionnaire, a smoker first was asked to recall the years in which she smoked cigarettes for any period 6 months or longer. Second, she was asked to recall the brands of cigarettes she smoked during each period, the specific years in which she smoked each brand, the number of cigarettes of each brand smoked per day, and the depth of inhalation for each. The interviewer probed for any changes in number per day of a particular brand; a change greater than 10 per day generated a new data entry. The sequence of temporal episodes yielded the summary measures of years actually smoked and years since cessation (if any). The collection of brand name and intensity records for each episode yielded a lifetime intensity measure, or average number of cigarettes smoked per day. The tar content per cigarette for any brand in any year was determined from historical estimates (Tobacco Merchants Association, 1978; USDHHS, 1981) and test data (Federal Trade Commission, 1976; Federal Trade Commission,1983).

Time-weighted average tar levels were calculated for the interval 1973-1982. This period was selected because (a) precise figures for tar content of all domestically produced cigarettes were available; (b) except for the latter part of the interval, when ultra-low-tar cigarettes became available, this period did not show as sharp a decline in tar content as the two previous decades; and (c) this proximal portion of the smoking history was assumed to be recalled more accurately by both self- and next of kin respondents.

Diet was assessed by asking about the usual frequency of consumption, approximately 4 years earlier, of 59 food items, including major sources of preformed retinol and carotenoids. For fruits and vegetables that the respondent

said were eaten primarily in certain seasons, frequency of consumption both in season and out of season and the length of season were obtained. In these analyses, the average frequency of consumption was calculated for the food group "vegetables", which in an earlier study of lung cancer among New Jersey white males (Ziegler, 1986) had shown the strongest, most consistent inverse association with lung cancer risk. The variable used in these analyses was calculated using exactly the same food items as had been used in the earlier study. However, it should be noted that the questionnaire used in this study also asked about consumption of several additional vegetables; this additional information is not yet included in the analyses. Therefore, the dietary associations presented here are preliminary. However, it is doubtful that this will strongly affect the degree to which diet confounds any association with radon.

In the occupational history section of the questionnaire, information was obtained on each full-time or part-time job held for 3 months or more since age 12. This included the name and address of employer; type of business; job title; duties performed; materials handled; exposure to solvents, fumes, or dust; and time period of employment. All industry and job title information was coded using the 1970 census index system (US Bureau of Census, 1971). Job title categories and industry-job title categories (selected job titles from specified industries; only those potentially exposed, excluding most clerical, administrative, and sales personnel) were chosen for analysis after an extensive literature review, with particular attention to Dubrow and Wegman's summary of occupational surveillance studies (Dubrow and Wegman, 1983). For this study, categories were also chosen if they represented occupations in which women were frequently employed.

In the passive smoking exposure section of the questionnaire, a subject was asked whether any member of her household ever smoked. This included parents and other members of her family while she was growing up, and her spouse if she

was ever married. This was followed by questions relating to who these individuals were, how long the subject lived with them while they smoked, what they smoked (cigarettes, cigars, pipes, or a combination of these), and how much they smoked.

Original NJ Female Lung Cancer Study: Results

Interviews were successfully completed for 994 (76%) of the 1306 female lung cancer cases identified, and for 995 (69%) of the 1449 female controls identified. Reasons for non-response are detailed in Table B1. Response rates were similar for all major histologic types. Of the cases interviewed, 269 (27%) were squamous cell carcinoma, 220 (22%) were small cell carcinoma, 290 (29%) were adenocarcinoma, and 215 (22%) were other histologic types. Of the cases with adenocarcinoma, 191 were judged "in-scope" and 99, "probably in-scope."

Table B2 shows the distribution of the 994 cases and 995 controls by various risk factors and subject characteristics. The overall distributions of cases and controls by age, race, and respondent type were by design very similar. The median ages for cases and controls were both 65 years. [However, the age distributions varied significantly among cases by histologic type, with a greater proportion of adenocarcinoma cases in the younger age stratum. The median ages for squamous cell, small cell, adenocarcinoma, and other histologic types were 65, 67, 63, and 63, respectively.] Ten per cent of the cases and 9 per cent of the controls were non-white. Interviews for 54 per cent of the cases and 53 per cent of the controls were conducted with the subjects themselves. The remaining interviews were conducted with next of kin, either the spouse (16% for cases, 19% for controls), or other next of kin (31% for cases, 28% for controls). The majority of the other next of kin respondents were daughters, sons, or sisters (e.g., for controls, 13%, 8%, and 3%, respectively). The distribution of respondent type among controls and among

cases varied significantly with age. For the younger subjects, a larger percentage were interviewed themselves; for the older subjects, a larger percentage had next of kin interviews, with an increasing proportion of other (nonspouse) respondents.

The percentages of cases and controls varied significantly with several different measures of cigarette smoking, including lifetime average number of cigarettes smoked per day, total duration of smoking, number of years since smoking cessation, and average tar content of cigarettes smoked during 1973-1982 (Table B2). More cases than controls were heavy smokers (25+ cigarettes/day) or moderate smokers (15-24 cigarettes/day) than light smokers (<15 cigarettes/day) or lifetime nonsmokers. More cases than controls had smoked for 35+ years rather than <35 years. More cases than controls were current smokers (quit 0-1 years) or recent ex-smokers (quit 2-9 years) than long-term ex-smokers (quit 10+ years). More cases than controls were smokers of high tar (21+ mg) cigarettes than lower tar (<21 mg) cigarettes.

More cases than controls were low consumers of vegetables (<35 servings/month) than high consumers (75+ servings/month).

In analyses of occupation, many of the job title categories considered to be high risk based on our literature review and/or found to be high risk for New Jersey males (Schoenberg et al, 1987) were not represented in the female data set. For example, there were no women ever employed as blacksmiths, boilermakers, brickmasons, automobile mechanics, plasterers, plumbers and pipefitters, roofers, or stationary engineers and firemen. There were only one or two metal molders, sheetmetal workers, asbestos insulation workers, furnacemen, and construction laborers. The smoking adjusted odds ratios (OR) were significantly high for professional and photographic equipment manufacturing workers [22 cases, 9 controls; OR=2.3; 95% confidence interval (CI) = 1.0, 5.1] and for laundry and dry cleaning workers [73 cases, 41 controls; OR=1.5; 95% CI = 1.0, 2.3]. The smoking

adjusted OR was also high for plastics manufacturing workers [29 cases, 15 controls; OR=1.9; 95% CI = 0.98, 3.9] and for food counter workers [36 cases, 21 controls, OR=1.8; 95% CI = 0.98, 3.3]. In addition, women who had worked as restaurant or food service workers for more than 20 years had a significantly high smoking adjusted risk [28 cases, 9 controls; OR=2.8; 95% CI = 1.2, 6.8].

Some of the industry and job title categories showed an excess of cases over controls, but the numbers of subjects were very small. These categories included petroleum industry [2 cases, 0 controls], construction industry [2 cases, 0 controls], lumber and wood products manufacturing [3 cases, 2 controls], asbestos products manufacturing [4 cases, 2 controls], primary iron and steel manufacturing [4 cases, 1 control], transportation industry workers excluding drivers [4 cases, 3 controls], drivers (irrespective of industry) [10 cases, 3 controls], gas stations and garage workers [4 cases, 1 control], painters [7 cases, 5 controls] and bartenders [3 cases, 0 controls]. It is not possible, given the limited number of subjects in these latter categories, to attribute any statistical significance to the findings of excess cases. However, in carrying out the analyses for the radon study, it was important to control adequately for any potential confounding by occupation in these data. Therefore, it was decided to include these small categories, along with the five larger categories mentioned above (professional and photographic equipment, laundry and dry cleaning, etc.) in an overall a posteriori high-risk occupation category, which would represent the potential influence of occupation. As shown in Table B2, 203 cases and 103 controls were represented in this high-risk occupation category. However, because this category was constructed after looking at the data, it is not possible to attribute any statistical significance to a derived risk estimate.

There was little difference between cases and controls in the percentage of subjects by educational level (Table B2). Slightly more cases than controls lived in the "low radon" counties (Atlantic, Cape May, Essex, Hudson, Ocean), while slightly

more controls than cases lived in the "moderately low radon" counties (Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union). [See footnote e, Table B2, for further details on the grouping of counties according to radon level.]

Among lifetime nonsmokers only [116 cases, 499 controls], there were slightly more cases than controls who were exposed to spouse cigarette smoke, and fewer cases than controls who were exposed to spouse smoke from only pipes or cigars.

Table B3 shows the odds ratios and 95% confidence intervals estimated for these risk factors and subject characteristics using multiple logistic regression analysis (Breslow and Day, 1980) as carried out using the microcomputer-based LOGRESS program (McGee, 1986). Of the several variables for smoking, only lifetime average number of cigarettes smoked per day was considered in this initial analysis. Smoking was the major risk for lung cancer among these women, with risks ranging from 4.4 for light smokers to 14.4 for heavy smokers. After controlling for smoking, the age variable (72+ years compared to 58-71 years)[1], the vegetable consumption variable (both low and moderate consumption) and the occupation variable showed moderate increases in risk. Respondent type, race, education, and county group had little influence on lung cancer risk, after adjusting for smoking, age, diet, and occupation.

Even though respondent type was not an appreciable confounder for the associations between lung cancer and smoking, there were significant differences in the smoking-related risk by respondent type, i.e., there was significant interaction (Schoenberg et al, 1989). Table B4 shows results of an analysis similar to that shown in Table B3, but including interaction terms between respondent type and each of the three variables for the smoking groups according to cigarettes per day.

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[1] The risk associated with increased age emerges despite the fact the the original case and control series were age matched. This reflects reverse confounding by smoking. It also reflects a cohort effect, in that subjects age 72+ were more often nonsmokers than were subjects younger than age 72.

The OR for the three cigarettes per day groups (i.e., the main effects in the model) now reflect the smoking-associated risk in subjects who were self-respondents, with risks ranging from 6.2 in light smokers to 27.7 in heavy smokers. The OR for the three respondent type* cigarettes per day groups (i.e., the interaction terms), when multiplied by the OR for the main effects, yield the OR for subjects with next of kin respondents, with risks ranging from 3.2 for light smokers to 9.1 for heavy smokers. The hypothesized reasons for this significant interaction have been discussed extensively elsewhere (Schoenberg et al, 1989). Actual differences in smoking between living and deceased controls may explain some of the risk differences by respondent type. However, misclassification by next of kin respondents seems as likely an explanation, given the significantly lower percentage of smokers reported by next of kin for cases. The possibility of misclassification is also consistent with differences in the degree of respondent type heterogeneity observed in histologic type specific smoking risks.

Because smoking is such an important risk factor, it was not considered sufficient to control only for lifetime average number of cigarettes smoked per day. Table B5 shows the results of analyses considering total duration of smoking, number of years since smoking cessation, or average cigarette tar content (1973-1982), in addition to lifetime average daily cigarette consumption. All of these analyses are adjusted for age, race, respondent type, vegetable consumption, occupation, and education (but not for the interaction between smoking and respondent type). Inclusion of any of the three detailed smoking variables shows a highly signficant improvement in the overall fit of the model, as measured by the increase in the likelihood ratio statistic [duration of smoking, Chi-square=40.9, 4 df, $p < 0.0001$; years since smoking cessation, Chi-square=78.6, 6 df, $p < 0.0001$; cigarette tar content, Chi-square=59.0, 6 df, $p < 0.0001$]. Within light, moderate, or heavy smokers, risk increases systematically with increasing number of years

smoked, with decreasing number of years since smoking cessation (if any), or with increasing cigarette tar content. The maximum risk is 18.8 for heavy smokers who smoked 35+ years, 20.5 for heavy current smokers, or 33.2 for heavy smokers of high tar cigarettes.

Logistic regression analyses for passive smoking were limited to lifetime nonsmokers (Table B6). After adjusting for age, race, respondent type, vegetable consumption, occupation, and education, there was a very slight, non-significant increase in risk associated with exposure to spouse cigarette smoke, and a nonsignificant decrease in risk associated with exposure to spouse smoke only from pipes or cigars. Previous analyses (not shown) gave the same results when exposure to smoke from any household member, not just the spouse, was considered. Also, previous analyses according to the duration of exposure to spouse cigarette smoke, or the reported intensity of exposure (number of cigarettes smoked per day in the house) did not show any systematic relationship, after adjusting for age. Therefore, only the spouse smoking variable shown in these tables has been used in the radon study analyses.

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TABLE B2

Number of lung cancer cases and controls in original study, by various risk factors and subject characteristics, New Jersey female lung cancer case-control study, 1982-1983

	No. of cases (%)		No. of controls (%)	
TOTAL	994		995	
AGE AT DIAGNOSIS[a]				
<58 years	255	(25.7%)	249	(25.0%)
58-71 years	489	(49.2%)	485	(48.7%)
72+ years	250	(25.2%)	261	(26.2%)
RESPONDENT TYPE				
self	532	(53.5%)	528	(53.1%)
spouse	155	(15.6%)	188	(18.9%)
other next of kin	307	(30.9%)	279	(28.0%)
RACE				
white, including hispanic	899	(90.4%)	910	(91.5%)
nonwhite	95	(9.6%)	85	(8.5%)
CIGARETTES/DAY [b]				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
< 15 cigarettes/day	198	(19.9%)	210	(21.1%)
15-24 cigarettes/day	414	(41.7%)	195	(19.6%)
25+ cigarettes/day	266	(26.8%)	91	(9.2%)
TOTAL NUMBER OF YEARS SMOKED				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
< 35 years	219	(22.0%)	235	(23.6%)
35+ years	659	(66.3%)	261	(26.2%)
NUMBER OF YEARS QUIT SMOKING				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
Current smoker, quit 0-1 years	712	(71.6%)	289	(29.1%)
Ex-smoker, quit 2-9 years	103	(10.4%)	78	(7.8%)
Ex-smoker, quit 10+ years	63	(6.3%)	129	(13.0%)
AVERAGE CIGARETTE TAR CONTENT, 1973-1982				
Lifetime nonsmoker	116	(11.7%)	499	(50.2%)
Nonsmoker, 1973-1982	60	(6.0%)	116	(11.7%)
Smoker, tar <21 mg/cigarette	664	(66.8%)	336	(33.8%)
Smoker, tar 21+ mg/cigarette	154	(15.5%)	44	(4.4%)
VEGETABLE CONSUMPTION[a]				
<35 servings/month	284	(28.6%)	238	(23.9%)
35-74 servings/month	532	(53.5%)	510	(51.3%)
75+ servings/month	178	(17.9%)	247	(24.8%)

(contd)

TABLE B2 (contd)
Number of lung cancer cases and controls in original study, by various risk factors and subject characteristics, New Jersey female lung cancer case-control study, 1982-1983

	No. of cases (%)	No. of controls (%)
TOTAL	994	995
HIGH-RISK OCCUPATION [c]		
no high-risk occupation	791 (79.6%)	892 (89.6%)
high-risk occupation	203 (20.4%)	103 (10.4%)
EDUCATION		
<8 years completed	127 (12.8%)	144 (14.5%)
8-12 years completed	648 (65.2%)	605 (60.8%)
13+ years completed	219 (22.0%)	246 (24.7%)
COUNTY AT DIAGNOSIS[d]		
Low radon	300 (30.2%)	265 (26.6%)
Moderately low radon	354 (35.6%)	389 (39.1%)
Moderate radon	190 (19.1%)	195 (19.6%)
High radon	150 (15.1%)	146 (14.7%)
LIFETIME NONSMOKERS ONLY, BY PASSIVE SMOKING		
No exposure to spouse tobacco	43 (37.1%)	196 (39.3%)
Exposure to spouse cigarette smoke	66 (56.9%)	250 (50.1%)
Exposure to spouse pipe/cigar only	7 (6.0%)	53 (10.6%)

[a] Cutpoints based on distribution of controls in original female lung cancer study (1st quartile; 2nd+3rd quartiles; 4th quartile).
[b] Lifetime average number of cigarettes smoked per day. Cutpoints based on bimodal distribution of controls in original female lung cancer study, with peaks at 10 and 20 cigarettes per day, and a long tail starting at 25 cigarettes per day.
[c] Ever employed in any occupational group shown to have a smoking adjusted risk of 1.5 or greater in the original female lung cancer study. This is an a posteriori definition, used only for the purpose of adjusting in the radon analyses for the possible effect of occupational exposure. See text for further clarification of this variable.
[d] County at diagnosis for cases, or county at ascertainment for controls. Low radon counties include include Atlantic, Cape May, Essex, Hudson, and Ocean counties. Moderately low radon counties include Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union counties. Moderate radon counties include Camden, Monmouth, Passaic, and Salem counties. High radon counties include Hunterdon, Mercer, Morris, Somerset, Sussex, and Warren counties. Grouping of counties was determined by the percentage of houses with basement or lower level screening values above 4 pCi/L in the statewide survey conducted for the New Jersey State Department of Environmental Protection (see Appendix G).

TABLE B6
Odds ratios[a] (95% confidence intervals) for association of lung cancer with passive smoke exposure (lifetime nonsmokers only)
New Jersey female lung cancer case-control study, 1982-1983

	Odds ratio	(95% CI)
PASSIVE SMOKE EXPOSURE		
No exposure to spouse tobacco	1.0[b]	---
Exposure to spouse cigarette smoke	1.2	(0.75,1.8)
Exposure to spouse pipe/cigar smoke only	0.52	(0.22,1.3)

[a] Odds ratios (95% confidence interval) from logistic regression analysis for 116 cases and 499 controls in original female lung cancer study, adjusting for age, race, respondent type, vegetable consumption, occupation, and education.

[b] Reference group: odds ratio=1.0; no confidence interval calculated.

APPENDIX C

Comparison of Women Included vs Not Included in Radon Study

The proportion of women from the original study who were included in the radon study was examined in subgroups defined by the variables shown in Table B2, i.e., by age, respondent type, etc. The results of these analyses are shown in Table C1. Only one subgroup, subjects who were smokers for less than 35 years, showed a statistically significant ($p < 0.05$) difference between cases and controls in the proportion of women included in the radon study. However, among both lifetime nonsmokers and moderate smokers (15-24 cigarettes/day), among long-term ex-smokers, among self respondents, and among whites, there also were proportionally fewer controls than cases who were included in the radon study ($0.05 \leq p < 0.10$). Most of these case-control differences were attributable to more refusals by current residents of control index residences. For short-duration smokers and for long-term ex-smokers, more controls did not meet the residence criterion.

Within cases and/or within controls, there were several statistically significant differences in the proportion of subjects who were included in the radon study. For both cases and controls, there were smaller proportions of young subjects (age < 58) who were included in the radon study, because of the higher percentage of these young subjects who did not meet the residence criterion. There were also smaller proportions of subjects with other next of kin (nonspouse) respondents who were included. Among cases, there were proportionally more subjects with other next of kin respondents for whom we could not obtain specific address information; among controls, there was a higher percentage of subjects who did not meet the residence criterion. There were also significantly fewer nonwhite subjects than white subjects from the original study who were included in the radon study. A higher percentage of nonwhite subjects did not meet the residence

criterion; there were also more nonwhites for whom specific address information could not be obtained.

Within cases and controls, there were no statistically significant differences in the proportion of subjects included in the radon study according to cigarettes smoked per day. However, radon study subjects had higher proportions of nonsmokers and light smokers, and lower proportions of moderate and heavy smokers. Controls who had smoked less than 35 years, cases who were current smokers, and cases who smoked high tar cigarettes had significantly lower proportions of subjects included in the radon study, primarily because fewer of these subgroups met the residence criterion.

There were significant increases in the proportion of original subjects included in the radon study with increasing educational level. This was related to differences in the percentage of subjects for whom address specific information could not be obtained, as well as differences in the percentages of subjects whose index residence could not be tested for radon (because of refusal or because the house had been demolished). These differences persisted when the analyses were restricted only to white subjects.

Those counties with lower proportions of cases or controls included in the radon study also had higher proportions who did not meet the residence criterion. There was little difference by county group in the proportion with no radon testing at the index residence, or in the proportion with refusal by the current resident.

Control nonsmokers with no reported spouse tobacco exposure also had a lower proportion included in the radon study, because there were more subjects for whom specific address information could not be obtained.

Although the case-control differences in radon study inclusion appeared to be minimal within most subgroups, the differences in risk factor distributions were frequent. Therefore, we repeated the logistic regression analyses shown in Appendix

B, with a variable representing inclusion in the radon substudy (Table C2). Among all subjects (TOTAL), adjusting for all other risk factors and subject characteristics shown, it was significantly more likely for a case than a control to be included in the radon substudy [OR = 1.3; 95% CI= 1.1, 1.6]. Given the smoking-related differences in the radon-associated risk observed in this study, it was also important to assess this bias potential within subgroups according to smoking. The radon study inclusion term was marginally significant for nonsmokers [OR = 1.5; 95% CI= 0.98, 2.3] and significant for moderate smokers [OR = 1.5; 95% CI= 1.0, 2.1]. Light smokers, who showed the strongest radon-associated risk, showed the least bias potential. Heavy smokers, who showed no radon-associated risk, also showed little bias potential.

Table C2 also shows the odds ratios for other risk factors within the smoking subgroups, after controlling for inclusion in the radon study. These odds ratios suggest that proportionally more cases with next of kin respondents were reported to be nonsmokers, while proportionally fewer cases with next of kin respondents were reported to be moderate or heavy smokers. The differences in smoking-associated risk according to respondent type discussed in Appendix B are consistent with the possibility of misclassification of smoking by next of kin respondents.

Among nonsmokers, the odds ratios for residence in the three higher radon county groups are significantly low, relative to residence in the low radon county group [moderately low: OR=0.57, 95% CI=0.34, 0.94; moderate: OR= 0.43, 95% CI=0.22, 0.85; high: OR = 0.59, 95% CI= 0.30, 1.2]. Among heavy smokers, the odds ratio for residence in the moderately low radon county group, relative to residence in the low radon counties, is marginally significantly high [OR = 2.0 (0.98, 3.9)]. These observations suggest the possibility that other, as yet undetermined, geographically-associated risk factors might be operating to mask any slight radon effect in nonsmokers or heavy smokers.

Another means of examining the possibility of bias in the radon substudy population is to compare odds ratios for other risk factors and subject characteristics in those subjects included vs. those subjects not included. Table C3 shows the results of such analyses, for the total study population, and for subgroups according to smoking. The 95% confidence intervals for the odds ratios are not shown; however, any statistically significant ($p < 0.05$) or marginally signficant ($p < 0.10$) differences in odds ratios between those included and not included are noted. In the total study population, the only marginally significant difference was in the odds ratio for moderate, long-term ex-smokers relative to lifetime nonsmokers. This difference also appeared in the comparisons for the moderate smokers. The only other statistically significant differences in odds ratios were for the heavy smokers. Those heavy smokers included in the radon study showed a pattern of increasing risk with increasing vegetable consumption, which was opposite to that observed for the heavy smokers not included in the radon study, and opposite to that observed for most other subgroups. The heavy smokers included in the radon study also showed a significantly low risk associated with fewer years of school, compared to no association in the heavy smokers who were not included in the radon study.

The absence of a radon-association among heavy smokers (in fact, the suggestion of a negative trend, although non-significant) suggested that the heavy smokers included in the radon study might be unusual in some respects. The differences observed in Table C3 are consistent with this suspicion, and suggest that the heavy smokers should be examined in greater detail. Table C4 replicates Table C1, but exclusively for heavy smokers. Although the numbers are small, several observations are noteworthy. There is a pronounced deficit of subjects with less than eight years of school among the heavy smoker cases included in the radon study. There is also a deficit of subjects with high vegetable consumption among the heavy smoker controls included in the radon study.

Conclusions The original female lung cancer study was population-based in design. However, in establishing the residence criterion for the radon substudy, and in eliciting cooperation from the current residents of the index addresses, the radon study was no longer population-based. Overall, there was significantly greater participation for cases in the original study than for controls. Moreoever, radon study subjects were more often older, whites, either nonsmokers, light smokers, or exsmokers, residents of counties with higher radon levels, and more highly educated. Nonetheless, there were relatively few significant differences in other risk factors and subject characteristics between those included and not included, except among heavy smokers, who showed some highly unusual risk factor distributions. The significant differences in smoking-related risk by respondent type discussed in Appendix B are consistent with the possibility of misclassification of smoking by next of kin respondents. The possibility of a case response bias and further misclassification of smoking, even by subject respondents, is purely speculative, but cannot be ruled out. All of these factors suggest that the results of this study with respect to differences in radon-associated risk according to smoking status need to be evaluated very carefully in other study populations before they are accepted as proven.

TABLE C1
Total number of women in original study,
and number and percentage (%) included in radon study[a],
by various risk factors and subject characteristics,
New Jersey radon-female lung cancer case-control study, 1982-1988

	CASES			CONTROLS			
	TOTAL	RADON		TOTAL	RADON		
	N	N	(%)	N	N	(%)	[p[b]]
TOTAL	994	433	(43.6%)	995	402	(40.4%)	[0.17]
AGE AT DIAGNOSIS							
<58 years	255	98	(38.4%)	249	78	(31.3%)	[0.11]
58-71 years	489	215	(44.0%)	485	216	(44.5%)	[0.91]
72+ years	250	120	(48.0%)	261	108	(41.4%)	[0.16]
[p[c] (d.f.)]	[0.09 (2)]			[0.002 (2)]			
RESPONDENT TYPE							
Self	532	246	(46.2%)	528	212	(40.2%)	[0.05]
Spouse	155	74	(47.8%)	188	89	(47.3%)	[0.99]
Other next of kin	307	113	(36.8%)	279	101	(36.2%)	[0.95]
[p[c] (d.f.)]	[0.02 (2)]			[0.06 (2)]			
RACE							
White	899	418	(46.5%)	910	386	(42.4%)	[0.09]
Nonwhite	95	15	(15.8%)	85	16	(18.8%)	[0.73]
[p[c] (d.f.)]	[<0.001 (1)]			[<0.001 (1)]			
CIGARETTES/DAY[b]							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
<15 cigarettes/day	198	83	(41.9%)	210	90	(42.9%)	[0.93]
15-24 cigarettes/day	414	178	(43.0%)	195	67	(34.4%)	[0.05]
25+ cigarettes/day	266	111	(41.7%)	91	32	(35.2%)	[0.33]
[p[c] (d.f.)]	[0.21 (3)]			[0.13 (3)]			
TOTAL NO. YEARS SMOKED							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
<35 years	219	90	(41.1%)	235	72	(30.6%)	[0.03]
35+ years	659	282	(42.8%)	261	117	(44.8%)	[0.63]
[p[c] (d.f.)]	[0.10 (2)]			[0.002 (2)]			
NO. YEARS QUIT SMOKING							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
Quit 0-1 years	712	289	(40.6%)	289	112	(38.8%)	[0.64]
Quit 2-9 years	103	49	(47.6%)	78	27	(34.6%)	[0.11]
Quit 10+ years	63	34	(54.0%)	129	50	(38.8%)	[0.07]
[p[c] (d.f.)]	[0.02 (3)]			[0.45 (3)]			

(contd)

TABLE C1 (contd)
Total number of women in original study,
and number and percentage (%) included in radon study[a],
by various risk factors and subject characteristics,
New Jersey radon-female lung cancer case-control study, 1982-1988

	CASES			CONTROLS			
	TOTAL N	RADON N	(%)	TOTAL N	RADON N	(%)	[p[b]]
TOTAL	994	433	(43.6%)	995	402	(40.4%)	[0.17]
AVG. CIGARETTE TAR CONTENT, 1973-1982							
Lifetime nonsmoker	116	61	(52.6%)	499	213	(42.7%)	[0.07]
Nonsmoker, 1973-1982	60	31	(51.7%)	116	45	(38.8%)	[0.14]
Tar <21 mg/cigarette	664	284	(42.8%)	336	126	(37.5%)	[0.13]
Tar 21+ mg/cigarette	154	57	(37.0%)	44	18	(40.9%)	[0.77]
[p[c] (d.f.)]	[0.04 (3)]			[0.50 (3)]			
VEGETABLE CONSUMPTION							
<35 servings/month	284	118	(41.6%)	238	83	(34.9%)	[0.14]
35-74 servings/month	532	241	(45.3%)	510	209	(41.0%)	[0.18]
75+ servings/month	178	74	(41.6%)	247	110	(44.5%)	[0.61]
[p[c] (d.f.)]	[0.49 (2)]			[0.09 (2)]			
HIGH-RISK OCCUPATION[c]							
no high-risk occupation	791	350	(44.3%)	892	363	(40.7%)	[0.16]
high-risk occupation	203	83	(40.9%)	103	39	(37.9%)	[0.70]
[p[c] (d.f.)]	[0.43 (1)]			[0.65 (1)]			
EDUCATION							
<8 years	127	35	(27.6%)	144	51	(35.4%)	[0.21]
8-12 years	648	278	(42.9%)	605	232	(38.4%)	[0.11]
>12 years	219	120	(54.8%)	246	119	(48.4%)	[0.20]
[p[c] (d.f.)]	[<0.001 (2)]			[0.01 (2)]			
COUNTY AT DIAGNOSIS[d]							
Low radon	300	112	(37.3%)	265	89	(33.6%)	[0.71]
Moderately low radon	354	163	(46.1%)	389	178	(45.8%)	[0.99]
Moderate radon	190	84	(44.2%)	195	76	(39.0%)	[0.35]
High radon	150	74	(49.3%)	146	59	(40.4%)	[0.15]
[p[c] (d.f.)]	[0.05 (3)]			[0.02 (3)]			
LIFETIME NONSMOKERS ONLY, BY PASSIVE SMOKING							
No spouse tobacco exp.	43	18	(41.9%)	196	70	(35.7%)	[0.56]
Spouse cigarette exp.	66	38	(57.6%)	250	116	(46.4%)	[0.14]
Spouse pipe/cigar only	7	5	(71.4%)	53	27	(50.9%)	[0.54]
[p[c] (d.f.)]	[0.16 (2)]			[0.03 (2)]			

(contd)

TABLE C1 (contd)

[a] Number of women included in radon study includes those whose index address was tested for radon and those whose index address was an apartment higher than the second floor (see Table 2). Percentage represents number of women included in radon study divided by total number of women in original study.

[b] p value for Chi-square test (1 degree of freedom, with continuity correction) comparing the percentage of all cases vs. all controls included in the radon study.

[c] p value for Chi-square test (with degrees of freedom noted in parentheses) comparing the percentage of cases (or controls) included in the radon study, by subgroups of the various risk factors and subject characteristics.

[d] Lifetime average number of cigarettes smoked per day.

[e] Ever employed in any occupational group shown to have a smoking adjusted risk of 1.5 or greater in the original female lung cancer study. This is an a posteriori definition, used only for the purpose of adjusting in the radon analyses for the possible effect of occupational exposure. See text, Appendix B, for further clarification of this variable.

[f] County at diagnosis for cases, or county at ascertainment for controls. Low radon counties include include Atlantic, Cape May, Essex, Hudson, and Ocean counties. Moderately low radon counties include Bergen, Burlington, Cumberland, Gloucester, Middlesex, and Union counties. Moderate radon counties include Camden, Monmouth, Passaic, and Salem counties. High radon counties include Hunterdon, Mercer, Morris, Somerset, Sussex, and Warren counties. Grouping of counties was determined by the percentage of houses with basement or lower level screening values above 4 pCi/L in the statewide survey conducted for the New Jersey State Department of Environmental Protection (see Appendix G).