Chapter 2

AND THE NUMBERS WERE FRUITFUL AND MULTIPLIED

> *Epidemiology is a crude and inexact science. Eighty percent of cases are almost all hypotheses. We tend to overstate findings either because we want attention or more grant money.*
>
> — Charles Hennekens[1]

> *One might say that giving computers to analysts was akin to giving aerosol paints to graffiti artists.*
>
> — John H. Fennick[2]

> *Eat less fat, don't smoke, exercise regularly, drink alcohol only in small quantities, practice safe sex, use seatbelts, avoid lawyers and doctors unless absolutely necessary, and choose your parents carefully . . . In spite of the most prudent behavior, however, our epitaphs may still be: "Stopped smoking 1968. Stopped drinking 1973. Stopped eating meat 1981. Died anyway 1993."*
>
> — Peter H. Gott, M.D.[3]

IN MIDSUMMER 1997, the USA cable channel ran back-to-back versions of the same movie, "Twelve Angry Men"—the original film that was released in 1957, followed by a modern remake for television.

I'd seen the first one and remembered that it had starred Henry Fonda as a lone juror in a murder trial stubbornly holding out against a snap verdict of guilty that every other juror favored. I was curious to compare it with the remake to see what difference 40 years might have made, not in the story but in the environment of the jury room.

The 1997 version was virtually a scene-for-scene and word-for-word replay of the original. What differences there were were minor. For instance, the jury was still all male but no longer all white. There was a nonworking electric fan in the 1957 jury room; in 1997 it was a

nonworking air-conditioner. This time Jack Lemmon played the Fonda character. In 1957, the juror most vociferously demanding that the accused be found guilty was played by Lee J. Cobb; in 1997, George C. Scott was the "bad guy."

But here is where 40 years of intervening history come in: in 1957 there were no fewer than five ashtrays on the long table the 12 men sat around and several of them smoked during deliberations, mostly cigarettes but also one pipe. In keeping with what has been true throughout history, the majority did not smoke.

(If it means anything, one member of the 1957 cast was Jack Klugman, who later attributed severe damage to his vocal cords to his cigarette smoking. Yet, curiously, he was one of the jurors who didn't smoke in the film.)

I don't remember if there was a "No Smoking" sign in the 1997 jury room; there wasn't, of course, in 1957. In any case, Scott was the *only* juror who smoked in 1997, and he did it in the washroom.

One point of this exercise is that in 1957, those jurors who smoked did so freely and naturally, as Americans did most everywhere back then, and none of the nonsmokers objected, or even thought about objecting. But one can be sure that if Scott had lit up in the 1997 jury room, there would have been all kinds of exclamations of distress and moralistic lectures from his nonsmoking fellows.

There was, however, one incident having to do with smoking in the 1957 movie. The foreman, Martin Balsam, is standing at the head of the table trying to say something and smoke from the cigarette of a nearby member of the jury keeps drifting into his face. He waves at it irritatedly and says to the man, "Would you mind?" To which the other replies, "Oh . . . sorry."

But this was hardly some kind of early antismoking statement; Balsam himself smoked in the movie. It was simply a bit of naturalistic byplay. Scott's apparently voluntary banishment to the lavatory was likewise quite naturalistic in 1997. Only a nut like me would even notice it. (It was a little strange, though, that none of the men complained about his fouling the air in that small room.)

Half of all present-day Americans weren't alive 40 years ago and many others were too young to have memories of what it was like in 1957, when smoking was simply something some people indulged in and others didn't, when it was not the divisive issue it is today and when

certainly nobody regarded it as a social offense on a galactic federation scale. Yet even many who were in adulthood four decades ago, and who smoked, have undergone a fundamental "attitude adjustment" toward smoking since then.

The example closest to home for me is that of my wife, who was a heavy smoker of cigarettes from age 16 to 63 (but is nevertheless still alive and healthy in her 80s and looks to be in her 50s) and who is an otherwise tolerant and nonjudgmental person. Every time we watch an old film on American Movie Classics, she remarks, in a disapproving tone, something on the order of, "They're doing an awful lot of smoking in this movie"—having completely forgotten that the smoking was a reflection of the times and not, as antismokers allege about smoking in current movies, an insidious scheme by the cigarette companies to seduce impressionable youth.

Some years ago, in Venice, Florida, I went to a restaurant with my wife's sister's husband and five or six of his friends. They were all several years older than me and all of them, I believe, were veterans of World War II. When the host asked, "Smoking or nonsmoking?", they all laughed and chuckled, as if to say: What a silly question. Do we look like we're stupid enough to smoke? No one asked me and I didn't say anything.

I don't know how many of those guys were former smokers, but would bet most of them were. Their old comrade the cigarette was now their enemy. My brother-in-law, however, who had been a tail gunner on a B-24 in the Pacific theater, never smoked or drank a day in his life, yet his last 10 years were spent in and out of hospital, either in his hometown of Framingham, Massachusetts, or in Sarasota, Florida, as he went through one health crisis after another. He always recovered, so strong was his constitution. We used to say he would outlive all of us because the doctors monitored him so closely. It was only after his kidneys failed and he went on dialysis and after two heart bypass operations that a massive stroke felled him at the age of 75.

My wife and I visited him in the hospital in Sarasota after the second operation. Three doctors came into the room to talk to him. They had given him a 20 percent chance to survive the operation and I heard them tell him that if he had been a smoker, he wouldn't have had any chance at all.

Thus smoking was adjudged guilty even though it wasn't anywhere near the scene of the crime in this case.

The change in the way the overwhelming majority of Americans view smoking today is the major accomplishment of the surgeon general's 1964 report on *Smoking and Health*. Which brings me to a second reason for dwelling on "Twelve Angry Men." In both the 1957 and 1997 versions, the Fonda/Lemmon character gradually persuades the 11 other jurors, who are chafing to discharge their duty and get back to their lives, to overcome their prejudices against the defendant and take a close, analytical look at the evidence. They do so and eventually find him not guilty.

That was what I attempted to do in Chapter 1 regarding the surgeon general's indictment of smoking—to take a look at the original evidence and try to arrive at some verdict about its validity. It was, when I first started out, an endeavor wholly out of range of my competence as a lay person untrained in medicine, epidemiology or statistics. But the jurors in both versions of "Twelve Angry Men" are also ordinary people, unskilled in forensics. It is their collective, native common sense and the use of logic that eventually leads them to agree that the evidence against the defendant simply does not hold water.

That is how our justice system should work and does work (one hopes) more often than not. To paraphrase Winston Churchill's definition of democracy, it's probably the worst system we could use for arriving at the guilt or innocence of an accused person—except for every other system. Or to paraphrase another saying about war and generals, the administration of the law is too important to be left to the lawyers. But when it comes to the medical establishment and its unanimous indictment of smoking, anyone without medical expertise who attempts to judge the evidence armed only with common sense and personal experience and observation is undertaking a hopeless and, many would say, a foolish if not downright suspicious task. People's health is too important to be left to any but the health experts.

Maybe so, but when medical findings (or beliefs or assumptions) are translated into laws or ordinances affecting people's personal lives and behavior; when they divide the citizenry into the favored and disfavored, with the taking away of certain rights from the latter (even such a basic one as the right to earn a living) and the giving of more rights to the former; when they promote and justify all manner of discrimination against and disparagement of the minority in however "good" a cause—when this happens, as it has in the case of smoking,

even the medically untutored citizen retains the right to say: *"Wait a minute. Is all this evidence really true? Even if it is true, is what we are doing on the basis of it wise and necessary?"*

So just for the sake of curiosity, let's delve a little more into epidemiology and statistics and in this and following chapters look at some of the important evidence against smoking that has been presented since the surgeon general's 1964 report.

Above all, let's exercise our common sense.

MY ATTEMPTS TO become at least minimally conversant with statistics and its uses have come a long way since I undertook this book, but it is a monumentally daunting field to the layman, especially one not especially skilled in mathematics. Even before I wrote Chapter 1, I'd already encountered "one-tailed" and "two-tailed" distributions and "standard deviations" and "logistic regression analyses" in epidemiological studies, but that was merely the tip of the iceberg. A catalog I received from John Wiley & Sons, a publisher of books on statistics, listed volumes on such topics as:

"Discrete Multivariate Distributions," which include such "families of distributions" as "multinominal, binomial, negative binomial, Poisson, power series, hypergeometric, Pólya-Eggenberger, Ewens and order *s*."

If you wanted to get into "Continuous Univariate Distributions," you'd be dealing with "extreme value, logistic, Laplace, beta, uniform, slash, *F*, *T*, noncentral *t*" and still other distributions such as "Pearson family, Johnson family, lognormal, gamma, loggamma, generalized gamma, Cauchy, exponential, Pareto, Rayleigh, chi-squared, inverse Gaussian and Weibull."

Then there are your "Univariate Discrete Distributions" which, according to the catalog, would introduce you to "extensive new work on mixtures, including generalized hypergeometric families; the increasing relevance of Bayesian inference to discrete distribution theory; the rapidly growing field of computer-generated, discrete random pseudo-random variates, and much more."

"Bayesian theory" is a whole field in itself, involving "single equation nonlinear models," "time series models" and "multivariate regression models." Not to be overlooked is "discrete stochastic dynamic programming," which has something to do with "discrete-time Markov decision processes."

But we're interested in how statistics is applied to the study of human health and things like smoking that affect it, and that gets us into the field of biometrics and such areas as "empirical Bayes and hierarchical Bayes procedures in simultaneous estimation of parameters and computer-intensive statistical methods" as well as "multivariate and multidimensional analysis" and "Bayesian analysis of sequential trials," without which we cannot hope to make meaningful "risk assessments."

Fortunately for today's medical researchers, it isn't necessary to have much more knowledge of statistics than I have. The early statisticians did the drudge work of solving equations and devising tables and formulas and so on, either by hand or with primitive mechanical calculators. Today it's all contained in computer software programs, and just as anybody with a can of spray paint can be a graffiti artist, anybody with a computer and one of these programs can come up with the "latest study." By tweaking a "parameter" here or fine-tuning a "parameter" there or "adjusting" for this or that "variable," you can arrive at just about any "finding" you want to, especially when dealing with "relative risks" that are, in most studies having to do with smoking and health, so close to pure chance that they could easily go one way or the other.

Statistics is a valuable means of telling us about probabilities—if, like any tool, it is properly used. In his 1964 report, the surgeon general illustrated a typical use of the "Poisson distribution" (see pages 40-42 of this book). Invented by French mathematician Siméon Denis Poisson in the 19th century, one of this formula's earliest applications was to calculate the probable number of annual deaths from horse kicks in the 14 cavalry corps of the German army between 1875 and 1894.[4]

More modern examples include using the Poisson distribution to help a grocery store manager determine the probability that he will sell X number of cans of, say, artichoke hearts in one week or half a week or two weeks or any period of time, based on the past average of cans sold per week. Or the formula could tell the manager of a busy airport the probability that any particular plane will be on the runway at any particular moment.

In these kinds of cases, the variables and confounding factors associated with them are few, if any, and can be pretty well defined and controlled for. Not so with living things like laboratory mice or rats or other animals, however, and even less so with human beings, when we are dealing with diseases and their causes.

Say that researchers want to estimate the probability that a new artificial sweetener might cause cancer in humans. It would be wholly impractical, as well as unethical, to test it directly on people. You'd have to enroll one group—perhaps consisting of hundreds if not thousands of volunteers—who would consume the chemical, say in a soft drink, and another "control group," similar in number and every other way to the first group, who would consume the same beverage sans the chemical under investigation. Both groups would have to be young, because you would have to follow the subjects for at least 20 years since it would take that long for cancer to develop in people.

Much, much easier and quicker to do a "bioassay" with laboratory mice. Here you can feed massive quantities of the chemical to a couple hundred of the unfortunate animals—quantities equivalent to a person's drinking thousands of cans of soda pop a day—and simply count how many of the mice had developed tumors at the end of a couple years, which is the average lifespan of the animals. Then you could report that yes, indeed, this chemical can cause cancer in people. One scientist calls this the "stuff and snuff" method.[5]

Unfortunately, some of the problems with this approach—ignoring the big one that a human would die of sheer bloat before he could drink anything remotely approaching thousands of cans of anything a day—are that different species or strains of laboratory animals are prone to different kinds of cancers, not all of which occur in humans. In some strains of mice "genetic drift" has increased the rate at which they develop spontaneous tumors. There may be factors the researchers are not even aware of. An example of this was the discovery by Italian scientists that the element manganese, which is present in most standard rodent diet formulations, was sometimes at levels up to nine times higher in the food the rodents were fed than the amount of manganese that was found to cause cancer in bioassays testing the element.[6] Obviously, overlooking a confounding factor like this could seriously "skew" (translation: screw up) the results of a study.

Sometimes just plain human fallibility can produce a misleading result. In September 1996, researchers at the University of North Carolina reported a study in *The New England Journal of Medicine** showing

*Or as one statistician heard a colleague say, "*The New England Journal of Medicine*? Oh, the *National Enquirer* of the medical world."[7]

that poor blacks 30 years ago ate healthier food than well-off whites. A year later they discovered that—oops—"an error in the computer program used to analyze their data" had given them a result 180 degrees wrong. In reality there was little difference in the healthfulness of whites' and blacks' diets in the 1960s.[8]

As Emily Litella, the comedic parody character on the television program "Saturday Night Live," would have said, "Never mind."

As fraught with complications as animal studies are, consider the statistical gymnastics a team of researchers went through after following a total of 20,551 U.S. male physicians aged 40 to 84 for up to11 years in an attempt to find out if the eating of fish helped prevent heart attacks:

> Relative risks were computed using Cox proportional hazards models, controlling for age and randomized aspirin and beta carotene assignment. A multivariate Cox proportional hazards model was used to control for potential confounders, including prior cardiovascular disease, body mass index, smoking status, history of diabetes, history of hypertension, history of hypercholesterolemia, alcohol consumption, vigorous exercise, and use of vitamin E, vitamin C, and multivitamins. Other dietary factors (red meat, vegetables, fruits, dairy, chicken or turkey, and fried foods) were tested individually for associations with sudden cardiac death in separate Cox models, and each was entered into the multivariate model to test for confounding. Tests for trend were performed by assigning an ordinal variable for each level of consumption and modeling this as a continuous variable in separate Cox proportional hazards models. For each RR [relative risk], 2-sided *P* values and 95% confidence intervals (CIs) were calculated. The relationships between both fish intake and n-3 fatty acid intake and risk of sudden death were explored further using spline regression modeling. Restricted cubic spline models with 4 or 5 knots were used to flexibly model these relationships using measured values of fish or n-3 fatty acid intake and avoiding the need for prior specification of the RR function or the location of a threshold exposure value.[9]

After all that, their conclusion:

"The existing evidence *suggests* that consumption of fish once a week will help prevent coronary heart disease and therefore should be a component of a healthy diet." [Emphasis added.]

No one ever knew that eating fish was good for you before.

(Funny thing: Americans are awed by epidemiological statistics, especially regarding smoking, yet don't trust the use of statistical sampling in the 2000 census—the Republicans in Congress don't, that is.)

Even if a study is well-structured and the controls and adjustments for variables and confounding factors and the resulting statistics are impeccable, what a researcher *doesn't* tell us because of the way in which he presents his findings can be as revealing as what he *does* tell us.

Consider that pioneer 1956 study by famed researchers Richard Doll* and Austin Bradford Hill[10] that was ranked first among the seven studies upon which the surgeon general based his landmark 1964 report (see page 55 of this book). By extrapolating from the actual smoker and nonsmoker deaths they recorded among British doctors, Doll and Hill calculated that the death rates for lung cancer in the general population were 166 per 100,000 for smokers of 25 or more grams of tobacco a day, or a pack or more, compared with only seven deaths per 100,000 for nonsmokers. Thus smoking was associated with an almost 24-fold increase in the risk of death from lung cancer (166 divided by seven=23.7).

But look at the figures from another angle. What Doll and Hill reported was that 99,993 nonsmokers out of every 100,000 people escape death from lung cancer as opposed to 99,834 smokers per 100,000 who are so lucky (subtracting seven nonsmoker deaths and 166 smoker deaths from 100,000 respectively). When we divide the smaller number by the larger number, we get a smoking vs. nonsmoking "survival factor" of appoximately 0.998.

What this means is that, while a person who smokes may indeed incur a 24 times greater risk of dying from lung cancer than a person who doesn't smoke, the smoker also has 99.8 percent of the nonsmoker's chance of *not* dying from lung cancer. But only the "24 times as likely" risk was considered by the surgeon general. Why?

There would seem to be but two explanations: either Luther L. Terry and his advisory committee were unaware that epidemiological findings can be presented in more than one way, which would not speak well of their scientific competence, or they opted for the version they

*Subsequently knighted Sir Richard for his work in alerting the world to the scourge of tobacco. Maybe I am hopelessly prejudiced, but I have trouble placing a great deal of confidence in a physician who deliberately misuses, as Doll does, the term "epidemic" in regard to smoking.

knew would resonate most deeply among Americans, which would speak better of their public relations acumen than their scientific integrity and impartiality.

If the calculation above seems like some sort of statistical sleight of hand, it is easily verified with a hand calculator by multiplying 100,000 by 0.998. The result is 98,800, which is reasonably close to the extrapolated number of smokers per 100,000 the Doll and Hill study calculated do not die of lung cancer. (Multiplying the full factor of 0.99841 against 100,000 gives an even closer result: 99,841.)

To put it the other way around, the nonsmoker's chance of not dying from lung cancer is a mere one two-thousandth of a percent better than the smoker's (subtracting 0.998 from 1.000, which is "unity" or equal chance). Not exactly an impressive margin.

The first two calculations are from an article by Peter Finch, a professor of mathematical statistics in Australia, published on the Internet by the British organization FOREST (Freedom Organisation for the Right to Enjoy Smoking Tobacco).[11] The third bit of elementary arithmetic is my own.

Finch applied the same method to another study,[12] this one of ischemic heart disease, or IHD (a term synonymous with coronary heart disease, or CHD). Among other things, this study reported the estimate that, for males below age 45 who smoke 25 or more cigarettes a day, the death rate from IHD is 104 per 100,000 and for like-aged nonsmokers is only seven per 100,000, the same as Doll and Hill's figure for lung cancer deaths among nonsmokers. Since 104 divided by seven is 14.8, that meant that male smokers, in this age group and in this study, ran nearly 15 times the risk of nonsmokers of dying from coronary heart disease, and the researchers duly informed the scientific community of that, and no doubt the newspapers as well.

"This way of telling it is a very effective way of highlighting the message that 'smoking kills,'" comments Finch.

("Smoking Kills" is one of six rotating warnings on cigarette packages in Australia. In Ireland, I'm told, it's "SMOKING KILLS!" As actress Brooke Shields is reported to have said, "Smoking kills. If you're killed you've lost a very important part of your life.")

But what is the smoker's chance of *not* dying from coronary heart disease? When we answer that question, things once again appear in a different light.

To answer it, Finch says, again note that 99,993 per 100,000 nonsmokers escape death from IHD whereas 99,896 smokers do (seven and 104 subtracted from 100,000 respectively). Dividing 99,896 by 99,993, this time we get a smoker vs. nonsmoker "survival factor" of 0.999. (To prove it again, multiplying 0.999 against 100,000 gives the figure of 99,900—almost exactly the same as the 99,896 smokers that this study estimated do not die of IHD.)

In other words, while the data tell us that smoking is associated with a 15-fold increase in risk of death from IHD, it also tells us that the smoker nevertheless has 99.9 percent of the chance of a nonsmoker of escaping death from IHD—*or virtually the identical chance.*

And this time the nonsmoker's chance of not dying from IHD is only about a thousandth of percent better than the smoker's.

(Incidentally, it is a curious fact, if it is a fact, that while heart disease is the leading cause of death among the general population, neither it nor lung cancer is the leading cause of death among smokers. That is, according to one source, "smoking results in about as many deaths from heart disease among smokers as it does lung cancer deaths."[13] If it is true, as we are told, that lung cancer takes one of every 10 smokers, then heart disease must take another tenth, which means that eight out of 10 smokers die from something other than lung cancer or heart disease. Furthermore, according to the Centers for Disease Control and Prevention, smokers who stop smoking cut their risk of heart disease in half after one year.[14] Maybe nonsmokers worried about their hearts ought to take up the habit for a while and then quit!)

I tested Finch's method of looking at statistics with a claim I found in a book called *Cigarettes: What the Warning Label Doesn't Tell You* (about which more below). The authors cite one study which found that the death rate for people with asthma who had never smoked was 3.7 per 100,000, whereas among current and former smokers it was 8.3 per 100,000. Smokers were thus more than twice as likely to die from asthma than never-smokers.[15]

So as not to give smoking even the slightest statistical edge, let's say that nonsmokers' death rate from asthma is only three per 100,000 and that of smokers is nine—a full three times greater. Even minimizing the one death rate and maximizing the other, the chance a smoker has of not dying from asthma is 99.9 percent of that of the never-smoker (99,991 divided by 99,997). Again almost the identical chance.

How can all this be? If smoking is, allegedly, a proven health risk for an array of diseases, and especially for lung cancer and heart disease, how can the smoker's chances of not dying from those diseases be so close to the nonsmoker's chances?

The explanation simply is that in studies of these diseases we are dealing with very small numbers of deaths (of some of the subjects in the studies themselves) projected to a very large population. It is by multiplying the small X-per-100,000 death rate by the number of 100,000s in a total population that we come up with impressive numbers.

Here the unconvinced reader (is there any other kind?) may interpose an objection: If smokers' and nonsmokers' chances of not dying from lung cancer or heart disease are practically the same, why is it that in *every* study of these diseases smoker deaths *always* outnumber nonsmoker deaths?

That's a very good question, even if I raised it myself—although it should be clarified to say: every smoking study that we are *told* about. Studies that fail to come up with the results the researchers are looking for are seldom reported. Anyway, the question actually states that in every epidemiological study, a certain number of people turn up dead. This is because in any random population observed over a period of years, some people are inevitably going to die.

The fact that, in studies of smoking, dead smokers outnumber dead nonsmokers is certainly presumptive evidence that smoking may have been a causative factor in the smokers' deaths. But we are not talking about how many smokers vs. nonsmokers die in a particular study but about the comparative chances that *everybody* in the population at large has of not dying from the disease in question.

As one commentator wrote in *Fortune* magazine a decade ago:

> Although the Public Health Service has been reticent about publishing the fact, every study cited in support of the statement that "cigarette smoking causes cancer" reveals that a smoker is unlikely to get cancer—only that he is statistically more likely to get it than a non-smoker. No one can say how much more likely. This is true of all supposed carcinogens.[16]

No epidemiological study, even one that involved a truly random population in which subjects and controls were perfectly matched—which is impossible and is why researchers must "control" and "adjust"

for all kinds of variables—can predict what will happen to any individual. Epidemiological studies only tell us what did happen to a certain number of people in the study itself. The big numbers we get when we extrapolate that number to the general population do not represent any actual dead people who have been counted, or even could be counted. They are an abstraction. No matter how many studies come up with more dead smokers than nonsmokers, it is the *chance* that any actual, real person has of dying or not dying from smoking that is the important thing.

Of course, whether or not anyone should decide to continue to smoke or to take up smoking based on that chance is also the important thing. I repeat Prof. Finch's discussion of that below.

FORGETTING THE "comparative chances" aspect for a minute, is Doll and Hill's smoker lung cancer death rate estimate even remotely valid today, assuming it ever was valid?

According to the Census Bureau's Statistical Abstract of the United States, the death rate from all causes was 880 per 100,000 in 1995 when the U.S. population was approximately 263 million.[17] (This was the latest year listed on the bureau's Website at the time of this writing.) Multiplying 880 times the number of hundred thousands (2,630) gives us a figure of 2,314,400 total deaths from all causes in 1995.*

Lauren Colby tells me that it is estimated that about six percent of all deaths in any given year are due to lung cancer (which is another way of saying that 94 percent of all deaths are *not* due to lung cancer). If this estimate is correct, it means that nearly 139,000 Americans must have died from that disease in 1995. Medical authorities also generally agree that at least 80 per cent of lung cancer deaths are "caused by smoking," which means that slightly over 111,000 smokers paid the ultimate price for their habit and perished from lung cancer in 1995. This works out to about 4.8 percent of all deaths.

Yet according to figures fed to Ann Landers on the occasion of "The Great American Smokeout" in 1995 and reported in her column, the American Cancer Society predicted that more than 157,000 smokers would die from lung cancer that year (see Chapter 5). That's 46,000 more deaths than the 111,000 estimate.

*Of course, the bureau had to do it the other way around first. That is, count the total number of deaths, then calculate the rate per 100,000.

In its 1995 "Cancer Risk Report" which I found on the Internet, however, the society estimated that there would be a *total* of 157,400 lung cancer deaths that year and further stated that "About 30 percent of all cancer deaths can be attributed to smoking."[18] Thirty percent of 157,400 is only 42,200. Thus on the one hand the society was telling Ann that *all* lung cancer deaths are attributable to smoking and on the other hand was suggesting that 70 percent of lung cancer deaths occur among nonsmokers!

The ACS wasn't suggesting any such thing, of course, for elsewhere it has claimed that not 80 but 90 percent of lung cancer deaths are "caused by smoking." But even applying 90 percent to the 139,000 figure from the previous page, that comes to some 125,000 smoker lung cancer deaths in 1995, which is still 32,400 fewer deaths than the society's prediction of 157,400.

I am not deliberately trying to confuse anyone; it's only that I myself am confused. Unfortunately, things become even more confusing when we consider that, according to the Census Bureau, the death rate from *all* cancers was 200 per 100,000 in 1995, or a total of 526,000.* Six percent of that (the percentage of lung cancer deaths among all deaths) is 31,560, and 80 percent of that (the conservative percentage of lung cancer deaths "caused by smoking") is 25,248—which is nearly 132,000 fewer smoker lung cancer deaths than the Cancer Society's prediction. And even using the society's 90 percent figure gives a difference of more than 128,000.

It's also nearly 86,000 fewer lung cancer deaths "caused by smoking" than the figure we get when we multiply 4.8 percent times total deaths in 1995.

It's also some 302,000 fewer lung cancer deaths "caused by smoking" than we get when we apply Doll and Hill's rate of 166 per 100,000 to the 75 percent of the U.S. population that was over the age of 18 in 1995.

What if the estimate that 4.8 percent of all deaths in a given year are smoker lung cancer deaths is way off base? What if it were higher?

*Could this be where the Cancer Society arrived at its figure of 157,000-plus smoker lung cancer deaths? Thirty percent (the percentage of all cancer deaths it says are attributable to smoking) of 526,000 is 157,800. But in this case then, the society would be saying that *all* cancer deaths among smokers are lung cancer deaths!

It's certainly possible, but to get the cancer society's number of 157,400 smoker lung cancer deaths it would have to be quite a bit higher—not 4.8 percent but 6.8 percent of deaths from *all* causes in 1995.

How many people really die from lung cancer every year because they smoke? Doll and Hill didn't know. The Census Bureau doesn't know. The American Cancer Society doesn't know. I sure as heck don't know. Nobody knows.

WELL, I'VE OFTEN been asked (more usually, challenged), what about all those famous actors and actresses and other prominent people who smoked and died of lung cancer—Edward R. Murrow, Gary Cooper, John Wayne, Yul Brynner, Audrey Meadows? The list is endless.

This is what is called anecdotal evidence, and all I can say is, my anecdotal evidence is as good as your anecdotal evidence. For instance, Hal Roach, the producer of the original "Our Gang" movies, is said to have smoked four packs of cigarettes a day, and he lived to 102. We all remember that George Burns was never without a cigar in his hand, and he just missed the 100 mark. (Of course, he may not have inhaled cigar smoke directly but he breathed the fumes for many, many years, and we "know" how deadly secondhand smoke is, not to mention the danger of oral cancer from cigars.) And top this—the amazing Jeanne Calment of France, who lived till 122 and only stopped smoking cigarettes at 120 because she could not longer see to light the things.

When my nonsmoking son read the above paragraph in an early draft of this book, he remarked that it was really a pretty weak argument. After all, he said, how many Hal Roachs or Jeanne Calments are there? And true enough, if we were to get into a numbers game that pitted smoking anecdote against smoking anecdote, skeptics like me would lose. But would that prove anything, or would it only be a reflection of the fact that we keep a count of those smokers who die of lung cancer or some other "smoking-related" disease but not of those who don't die. Anyway, as another person reminded me, "The plural of anecdote is not fact"—and that should work both ways, shouldn't it?.

If we were to put the existence of UFOs up to a popular vote, the nonbelievers would again be overwhelmed. But what would be proved? If the government were ever to admit that it had been covering up the truth about visitations by UFOs for 50 years and had even recovered dead aliens from UFO crashes, we would immediately witness an ex-

plosion of UFO sightings and people would quite sincerely and truthfully swear to their factuality.

In a similar way, the fact that the government told us in 1964 that "smoking kills" goes a long way toward explaining many of the subsequent "sightings" of smoking-caused deaths.

It's in the genes, I'm increasingly coming to believe. Maybe John Wayne et al. were constitutionally susceptible to lung cancer. Maybe there was also something in their genes that predisposed them to go into show business. Or maybe the simple fact is that smoking, and smoking alone, did indeed kill them. But again, it's only those smokers who die of lung cancer that we hear about, not those who don't die.

THIS IS PROBABLY as good a place as any to insert an unfortunately necessary disclaimer: This book is written from the viewpoint of an ordinary layperson who decided to try to pierce the statistical smoke screen (forgive me) generated by the antismoking movement in hopes of finding out what really are the health dangers of smoking and whether they are of sufficient magnitude to justify the extreme "remedial actions" that have been imposed, and continue to be imposed, on American society.

I have never received any money from any cigarette company, and I would decline it were it offered. I must confess that I have gotten a number of free cartons of Doral from R. J. Reynolds for sending in tabs from cigarette packages, and my wife and I were both given a souvenir pen and complimentary pack of cigarettes one time when we toured the Philip Morris plant in Richmond, Virginia. (The company used to conduct daily tours, open to anyone, but for some reason discontinued them several years ago.) I also occasionally receive coupons in the mail for a few dollars off a carton from some of the companies. But that's the extent of it.

Furthermore, I have never knowingly owned stock in any cigarette company, or even had any communication whatsoever with any cigarette company before or during the writing of this book. Maybe I'll receive a letter from one of them saying, "Good job trashing the antismokers, Mr. Oakley. Please accept this check as a token of our appreciation." I won't, but I hereby give advance notice anyway that I will return it.

Not only do I not now have, nor have I ever had, anything to do

with any cigarette company or anyone associated with the tobacco industry, I have no trust in, no respect for and vanishing sympathy for the whole bunch of them. They have shown themselves to be quite willing to sell their only friends—the consumers of their products—down the river. But that's something I get into in Chapter 12.

Incidentally, speaking of the tobacco industry's "friends," in his book, *Thank You For Smoking,* Christopher Buckley ridicules the kind of people who constitute the membership of the "prosmoking" or "smokers' rights" groups I mention in this book. He portrays them as a rather pathetic crew, both in they way they are hooked on smoking and how they are used as pawns by an industry that itself views them with amused contempt.

The only thing I disagree with is the characterization of smokers' advocates as pathetic. Betrayed, yes. Pathetic, no. The pathetic smokers are those who have not rallied to support the few and small smokers' advocates groups but instead stand by silently and submissively as the antismokers push them farther and farther to the margins of decent society—even while picking their pockets with higher and higher taxes.

Finally, the statistics in the examples above are those reported by respected researchers and institutions. If following the numbers to their logical conclusions leads us to a different picture of the risks of smoking than is officially reported, shouldn't people know about it?

Not that most people will believe anything I say in this book. But it's possible that a few readers will be encouraged to reexamine what they know, or think they know, about smoking and, more important, what they think this nation should do about smoking.

It may be a vain hope. I recently came across a book titled *You* MAY *Smoke* by a Briton named H. Harcourt Kitchin, a layman like myself, who attempted to stem the rising antismoking tide. This slim volume, published in 1966, examined the flaws in both the 1962 report of the Royal Society of Physicians and Surgeons and the 1964 report by the U.S. surgeon general. Although Kitchin's arguments remain valid today, it goes without saying that his effort went for naught.

Maybe it was because the book was premature; the antismoking crusade was barely getting under way in 1966. In any case, I'd never heard of the book or seen any reference to it, even on a "prosmoking" Internet site, but my local library was able to borrow a frayed and yellowed copy of it from, of all places, Jerry Falwell's Liberty Baptist

College in Lynchburg, Virginia, and I quoted it from it in Chapter 1.

I'm indebted to Stanton Glantz's *The Cigarette Papers* (also cited in Chapter 1) for alerting me to *You* MAY *Smoke*. That was hardly the intention of this prominent antismoking activist, of course. He'd come across references to it in one of those stolen Brown & Williamson Company documents and discovered—oh, the perfidy!—that a British cigarette company, Carreras Tobacco, had purchased 7,000 copies of the book and that an American public relations firm working for B&W had approached and assisted Kitchin in publishing a U.S. edition.

Just one more pebble to be added to the mountain of evidence showing the utter lack of conscience on the part of the tobacco industry and just how low it will stoop to defend itself.

TO GET BACK TO Prof. Finch, his calculations don't mean that smokers can continue on their merry way without a worry that they might be inviting a health problem sometime in the future. As he writes:

> These relatively high percentage chances of escaping the disease in question do not by themselves establish that smoking is harmless. For one thing, they ignore death from other causes and refer only to one disease at a time rather than to the spectrum of diseases with which smoking has been associated . . .
>
> The point here is not that the second calculation [comparing the numbers of smokers and nonsmokers who don't die from the disease] exonerates smoking but that presenting only the result of the first calculation [the "times as likely" figure for smokers] suppresses an aspect of the data that could lessen the force of the message that "smoking kills." For telling a 40-year-old male heavy smoker that his chance of not dying from IHD is about 99.9 percent of that of a comparable non-smoker is much less likely to persuade him to abandon smoking than telling him that his chance of dying from IHD is almost 15 times that of a comparable non-smoker.

This of course is why studies never mention the more favorable chance. Doctors don't want to be perceived as encouraging anybody to smoke, and as Finch himself acknowledges, there is some degree of risk associated with smoking.

"Nevertheless," he says, "even if one accepts the edited versions of the facts presented by health promoters, then their message should perhaps more accurately be: *smoking kills relatively infrequently*." [Emphasis mine.] He goes on:

> The fact that a smoker has almost as much chance as a comparable non-smoker of escaping a disease does not itself make that a chance worth taking. One can best see this in an unemotional context that has nothing to do with smoking and the diseases that have been associated with it. Early work on the vaccination of children against polio showed that polio was contracted by 57 in 100,000 unvaccinated children, but only 16 in every 100,000 vaccinated children. This meant that an unvaccinated child was about 3.6 times more likely to contract polio than a vaccinated child. On the other hand, an unvaccinated child had 99.96 per cent of the chance of a vaccinated child of escaping polio. Nevertheless most people did not see this as a chance worth taking.
>
> This does not mean that vaccination is pointless. It is mentioned here only because it has been argued that the logic that sees polio vaccination as worthwhile should also suggest that smoking be abandoned. However, this argument ignores what the smoker sees as the benefits of smoking. Thus, in the case of smoking, as opposed to refusing to be vaccinated, some people do see a benefit in it and may value that benefit so highly that they see 99.9 per cent of the chance that a non-smoker has of escaping death from IHD as worth taking. A person's decisions about what risk factors to avoid involve balancing what he or she perceives as their benefits against the chance of falling to what some say might be their consequences.

And there in a nutshell is what bugs antismokers—the idea that any sensible person would weigh some perceived benefits of smoking against the possible—if not probable, if not inevitable—consequences and deliberately and freely c*hoose* to smoke.

This attitude was reflected in a letter sent to *Reason* magazine by Elizabeth M. Whelan, Sc.D., president of the American Council on Science and Health, in response to an article by senior editor Jacob Sullum. Sullum had written about how, with the traditional infectious diseases largely conquered, the public health field had switched to combating "metaphorical epidemics" like smoking and other health-threatening habits or lifestyles, and speculated about how this new emphasis could affect individual freedom.

Whelan observed:

"People tend to succumb to social pressure and impulse in adopting health-compromising lifestyles and then justify their actions by rationalizing about them . . . Choices made by neglecting likely negative

consequences may give some people a sense of 'freedom,' but how free can someone be who chooses chronic self-destruction?"[19]

To which Sullum responded that this notion of freedom "gives me the willies."

He wrote:

> Personally, I do not perceive enough benefit in smoking to justify the risk. But I'm sure a lot of smokers would have a hard time understanding why I enjoy bungee jumping. By saying that individuals who defend their "health-compromising" lifestyles are simply "rationalizing". . . Ms. Whelan impl[ies] that such choices are inherently irrational and that people who make them must be ignorant, stupid, or crazy. . . If people are free only to make careful, reasonable, fully considered choices that they will never regret, they are not free at all.

Here, however, I would disagree slightly with both Prof. Finch and Mr. Sullum. I really don't think that many smokers have made a decision, freely or otherwise, rational or otherwise, to smoke. Most smokers I know fully believe all the warnings about the health risks of smoking, yet they smoke anyway—not because of "addiction" or a weighing of perceived benefits vs. the risks or because of a what-the-hell-you're-gonna-die-eventually-whatever-you-do attitude but because, unconsciously, they sense that the risks to them personally are remote, even though, consciously, they may accept that for the entire class of smokers the risks are real and substantial.

To this it can be countered that the damage done by smoking accumulates by such slow and imperceptible degrees over many years that the smoker may not become aware of the damage until too late. I can only speak for myself, and it's been more than half a century since I started smoking. I'm now in my 70s and normal aging alone ensures that someday I will develop some kind of health problem or problems, one of them eventually terminal. Will I have croaked because I smoked, or because we all wear out sooner or later?

It should also be remembered that the main case the antis make against smoking is that it causes *premature* death, and that is a flexible term. As average expected lifespan has advanced over the past 100 years, from the 40s or 50s at the turn of the century to the 70s today, so has the age advanced at which we consider a death to be premature. But to the antis, no matter at what age a smoker dies, it is always "premature."

In all the years between 1964 and 1994, between Surgeon General Terry's report and my starting research for this book, I had no reason to suspect that this landmark document was not all it was cracked up to be. If anyone had asked me, I would have said I believed it, more or less, although I continued to smoke. When I was with Scripps Howard News Service from the late '70s to the mid-'80s, where I was one of three editorial writers, I somehow always got stuck with writing an editorial on Surgeon General C. Everett Koop's latest annual blast against smoking. But I never did actually editorialize; I merely, dutifully and reluctantly, wrote a column repeating his words without comment. I realize now that I was not only getting sick and tired of the endless assaults on smoking issuing from the surgeon general's office and everywhere else but that, intuitively, I had known all along that they were so much hokum.

DR. WHELAN'S LETTER to *Reason* brings me to the book I mentioned above, *Cigarettes: What the Warning Label Doesn't Tell You*. The book was compiled under the auspices of her American Council on Science and Health (ACSH) and is a compendium (in fewer than 200 pages) of everything that is known, suspected, alleged or assumed about the role of smoking in just about every human disease, affliction, ailment and condition under the sun.

The Preface gives a roster of 20 "selected established and suspected health effects of cigarette smoking" that are discussed in the book's 20 chapters. They are:

Lung Disease: Lung cancer. Chronic obstructive lung disease. Increased severity of asthma. Increased risk of developing various respiratory infections.

Cancer: Esophageal, laryngeal, oral, bladder, kidney, stomach, pancreatic, vulvular, cervical and colorectal cancers.

Heart Disease: Coronary heart disease. Angina pectoris. Heart attack. Arrhythmia. Aortic aneurysm. Cardiomyopathy.

Peripheral Vascular Disease: Peripheral vascular disease. Thromboangiitis obliterans.

The Skin: Wrinkling. Fingernail discoloration. Psoriasis. Palmoplantar pustulosis.

Surgery: Need for more anesthesia during surgery. Increased risk

of postsurgical respiratory infection. Increased need for supplemental oxygen following surgery. Delayed wound healing.

Orthopedic Problems: Spinal disc degeneration. Less successful back surgery. Musculoskeletal injury. Delayed fracture healing.

Rheumatological Conditions: Osteoporosis and osteoarthritis.

Environmental Tobacco Smoke and Pediatric Illnesses: Infections of the lower respiratory tract. More severe asthma. Middle ear infections. Crohn's Disease and ulcerative colitis. Sudden Infant Death Syndrome. Impaired delivery of oxygen to body tissues.

Complications in Obstetrics and Gynecology: Infertility. Miscarriages. Fetal growth retardation. Prematurity. Stillbirth. Premature "water breaking." Transmission of HIV to the fetus. Birth defects. Intellectual impairment of offspring. Sudden Infant Death Syndrome. Earlier menopause.

Male Infertility and Sexuality Dysfunctions: Decreased sperm motility. Decreased sperm density. Impotence.

Neurological Disorders: Transient ischemic attack. Stroke. Worsened multiple sclerosis.

The Brain and Behavior: Depression.

Abnormalities of the Ears, Nose and Throat: Snoring and hearing loss.

The Eyes: Cataracts. Complications from Grave's disease. Macular degeneration. Optic neuropathy.

Oral Health: Periodontal disease.

The Endocrine System: Increased metabolic rate. Blood sugar abnormalities. Increased waist-to-hip ratio (redistribution of body fat).

Gastrointestinal Disease: Stomach and duodenal ulcers. Crohn's Disease.

The Immune System: Impaired humoral and cell-mediated immunity.

Emergency Medicine: Injuries from fires. Occupational injuries.

Holy, uh, smoke! What a list! If we could only eliminate smoking, doctors could play golf all day long.

I look at the most important of these diseases or disorders in this book and the evidence that smoking is a causative factor of or is "associated" with them. (One I don't look at is palmoplantar pustulosis, which I never heard of but believe is a form of psoriasis.) But I must say

something here about Crohn's Disease, which is actually a group of inflammatory diseases that can occur anywhere in the gastrointestinal tract, and ulcerative colitis, an inflammatory disease of the colon.

According to the highly respected *Merck Manual of Diagnosis and Therapy*, the cause or causes of both these diseases is unknown. Although numerous hypotheses have been put forward, ranging from bacteria and viruses to environmental chemicals to poor diet, none has been proven. They may strike both sexes at any age, although Crohn's Disease is more common among Jewish people and seems to run in families.[20]

Nowhere does the manual suggest smoking as a possible factor, let alone "environmental" tobacco smoke. Ditto for Grave's Disease (hyperthyroidism/goiter). For the ACSH to implicate smoking in these diseases is more than a wild stretch; it is an irresponsible one.

Of all the maladies listed above, the only one I have personal experience with is periodontal disease.

The first time I had my teeth deep-cleaned (at age 68), the periodontist asked me if I smoked. To avoid a possible lecture, I told a half-lie and said I used to. My gums were fibrous, he said, and that was caused by smoking. I asked him how he knew. "How do I know? Why, 15 years of practice and hundreds of papers." I didn't ask him if his personal observation had come first and was later confirmed by articles in dental journals, or whether he first learned about this cause of fibrous gums from his reading and then began observing it in his patients who smoked.

Was smoking the cause of my periodontal disease? Don't nonsmokers ever get it? Or was it because I inherited bad teeth and the water wasn't fluoridated when I was growing up in Pittsburgh and my mother let me indulge in sweets and didn't make me brush immediately afterward and we never heard of flossing and all that? I still smoke, but my gums are improving and my teeth (those I have left) are more sound than they have ever been, thanks to conscientious brushing and flossing and regular cleanings by the periodontist.

More personal experience:

Twice, when I have had a tooth extracted by other dentists, I have been advised not to smoke because that would cause "dry pockets." I smoked anyway, and my "pockets" stayed wet.

A few years ago in Georgia I cracked a rib when I tripped and fell

against a low retaining wall in my backyard (which figures since according to *Cigarettes* smokers are more likely than nonsmokers to have accidents). The doctor at the emergency clinic advised me not to smoke for a couple weeks because that could invite pneumonia. I smoked anyway and didn't get pneumonia and, despite the "delayed fracture healing" the American Council on Science and Health says smoking causes, the fracture healed in the same amount of time the medic said these things normally took to heal.

I'd cracked a rib once before, when after my retirement from Scripps Howard I became a school bus driver in Fairfax County, Virginia. I was standing on the bus's bumper one morning checking under the hood and slipped off the bumper. My chest hit hard against the edge of the radiator. At the clinic they gave me a big elastic bandage to wear around my chest. When I asked the doctor in my second rib-cracking episode about that, he told me that was the worst thing you could do.

I don't pay a whole lot of attention to doctors.

As if anticipating my immediate knee-jerk argument against *Cigarettes*, in the Introduction, one Kristine Napier, who is not identified in the Acknowledgments, writes that "[E]veryone knows at least one smoker 'who has smoked for 40 years and is healthy as a horse.'"

But consider an analogy of two soldiers, she suggests, each of whom must traverse a field. Soldier A has to cross a field laden with land mines, and soldier B a field relatively free of such deadly traps. Even though each soldier may cross his field safely, the odds definitely favor soldier B. Soldier A may avoid many, perhaps even most, of the land mines, but he is highly unlikely to avoid all of them.

According to the American Council on Science and Health (and everybody else), the odds are heavily against the smoker. If he doesn't trip the lung cancer land mine, he may fall afoul of the esophageal cancer land mine. Or if he misses that one, there are the peripheral vascular or the osteoporosis or the psoriasis, etc., etc., land mines. So pay no attention to all those healthy smokers "everybody knows." Above all, don't ask how realistic the odds are.*

*I remember as a boy hearing that the expected lifespan of a Royal Air Force pilot in the Battle of Britain in 1940 was something like two weeks. But I later read in a book I have unfortunately lost that, despite the odds, the overwhelming number of pilots who survived those two weeks went on to survive the entire six-months-long battle.

This is a vivid analogy, but a little misleading. In real life, both soldiers (representing all of us) cross the same minefield and face the same deadly traps of disease and accident. Leaving aside the role genetics may play* in endowing some of us with greater or lesser protection from one or another of those traps and others of us with more or less vulnerability to them, the difference between soldier A and soldier B is the former's lack of caution. He just plows on ahead, ignoring warnings from such as Whelan et al. that his recklessness—his smoking—is "highly likely" to get him blown up or seriously injured.

But smoking is not the only so-called risk factor in life, although in the view of ACSH it apparently outweighs all others. Many people have unhealthy diets. Many work in high-risk occupations. Many engage in dangerous sports. Nor is there only one single risk factor associated with the major diseases. Consider heart disease, or the family of diseases we lump under that term, which kills more Americans every year than any other disease. Heart attacks from coronary artery disease (CAD) alone took away some 487,000 of us in 1994.[22] The term CAD embraces arteriosclerosis (degenerative changes in the arteries) and atherosclerosis (fatty deposits, or plaque, on the arterial walls).

According to my source for this figure, the famous Framingham Heart Study, now in its 50th year, has to date identified over 100 potential risk factors for atherosclerosis.

This isn't a typo. The researchers believe there may be *100* potential risk factors for atherosclerosis. The major "modifiable" ones that individuals can do something about by way of helping them tread more safely through life's minefield include smoking, obesity, high blood pressure, physical inactivity, diabetes and cholesterol. The "unmodifiable" risks, those you can't change, include age (45 percent of the deaths in 1994 were of people over 65), gender and family history (genetics again).[23]

But an earlier source reported that the dedicated researchers in

*Which is leaving aside a heck of a lot. Some hereditary diseases which have been traced to defects in specific genes include: Alzheimer's, neurofibromatosis, cystic fibrosis, muscular dystrophy, retinoblastoma (tumor of the retina), Wilm's tumor (childhood kidney cancer), hemophilia, Hodgkin's disease, amyotrophic lateral sclerosis (Lou Gehrig's disease), colon cancer and, yes, lung cancer.[21]

Framingham had identified some *200*(!) risk factors for heart disease, which I guess must include CAD and IHD and CHD and everything else that can affect the heart.[24]

(Although my brother-in-law lived in Framingham he wasn't a participant in the study, which is a good thing for them because his health history would have seriously confounded their statistics. Incidentally, while he never smoked or drank, he refused to wear a seat belt in a car. That was a modifiable risk factor for personal injury that he voluntarily assumed.)

It really doesn't matter whether it's 100 or 200 risk factors or some other ridiculously outlandish number, for how could people possibly avoid all of them, even if they knew what they were? How many more will the Framingham study "identify" in its second half-century, and what value will its findings be? You might as well say that being alive is a major risk factor for death from a heart attack. Indeed, simply having been born is the one constant factor in deaths from every cause.

According to cardiologist William Castelli, director of the Framingham study, while smokers have a 70 percent chance of dying of a heart attack, and while high blood pressure increases the risk of heart attack and stroke by up to five times, high total cholesterol and low HDL (high-density lipoprotein cholesterol, the "good" kind) are among the strongest predictors of heart disease. And for him personally, high cholesterol is the Number One risk factor.

"I'm living proof that you can beat the odds of heart disease—even if it runs in your family," he says. He is the first male among his kin ever to reach the age of 50 without having a heart attack.[25]

(The first male? That suggests that gender might be a greater heart attack risk factor for him than high cholesterol.)

Dr. Castelli did it by bringing his total cholesterol down from somewhere in the 270s to 190 and raising his HDL to 63 from a former 49, and he accomplished that by starting, in his 30s, to jog every day and to cut down on dietary fat.

"When you see the Golden Arches," Castelli told an audience of elderly residents of Framingham and next-door Natick at a health fair in 1997, "you are probably on the road to the Pearly Gates."[26]

Probably? For everyone? I've eaten hamburgers and french fries all my life and smoked for most of it, and if I'd never taken a bite out of one of those fatty things or sucked on a single "cancer stick," I

couldn't be any healthier than I am (knock on my wooden head). Even if smokers have a 70 percent chance of dying from a heart attack (70 percent of what—a 100 percent chance?), I humbly submit that there is probably just as much, if not more, of a chance that other important factors are also involved, chief of which is a natural proneness toward high cholesterol, a condition my wife has but I don't, thanks to my wise choice of forebears.

According to the National Center for Health Statistics, a branch of the Centers for Disease Control and Prevention, 738,731 Americans died of diseases of the heart in 1995.[27] The American Council on Science and Health says that "Each year, cigarette smoking accounts for nearly 200,000, or one fifth, of all deaths from heart disease in the United States."[28] That would mean there are nearly a *million* heart disease deaths each year. Again never mind; the ACHS is grinding its ax.

Let's assume that a fifth of annual heart disease deaths are caused by smoking. That means that four-fifths of annual heart disease deaths are *not* caused by smoking but by something else, and the most likely something else is a high-fat diet.

Or so I believed until recently. A common airborne bug called *Chlamydia pneumoniae*, which is responsible for 10 percent of the respiratory illnesses people usually call flu, is being increasingly scrutinized as a cause—possibly even the most important cause—of heart disease. Or if not exactly a cause per se, certainly an important trigger for a heart attack. A cousin of *Chlamydia trachomatis,* which according to the Merck Manual is now the most common cause of sexually transmitted diseases in the United States, *Chlamydia pneumoniae* was discovered in the 1960s by Dr. Thomas Grayston of the University of Washington, but only in the past few years did researchers begin detecting telltale signs of the bacterium in plaque taken from the clogged arteries of heart patients.

According to Grayston, the "scenario" may go something like this:

> An unfortunate soul inhales *C. pneumoniae.* To fight off the bacteria, the immune system sends macrophages—cells that are supposed to destroy invaders—to the lungs. Lab tests have shown, though, that the microbe can survive being gobbled up by the immune cells. Now the macrophages carry the wily bugs throughout the body. Everywhere they go, they assail the arterial walls, entering cells to reproduce.

> Because macrophages seek out trouble spots, *C. pneumoniae* are often carried to places where there's already an arterial injury—say, from a deposit of bad cholesterol. As the bacteria damage or kill cells in the coronary arteries, more macrophages arrive to repair the injury, but they only bring more of the intruder. A pile of macrophages, reproducing bacteria, and other detritus forms in the arterial wall. As it grows it bulges into the bloodstream and narrows the artery, setting the stage for a clot to choke off blood flow.[29]

Confirmation of this scenario could help solve two medical mysteries: 1) why half of all heart attacks happen to people with normal levels of cholesterol, and 2) why the incidence of heart disease, which began rising in the 1920s (as did lung cancer), has been falling (as has cancer of all kinds, including lung cancer), even though Americans are living as high on the fatty dietary hog as ever. It would also raise the possibility that heart attacks could be treated, or even prevented, with ordinary antibiotics. In fact, the decline in heart disease could be due to the introduction of antibiotics, which have inadvertently been killing *C. pneumoniae* while being administered for other purposes.

If so, the *Chlamydia pneumoniae* story would repeat that of another common bacterium, *Helicobacter pylori*, which doctors now know causes most stomach ulcers and can be eradicated with antibiotics. These discoveries have inspired scientists to start pursuing other bacteria and viruses as possible culprits in a number of debilitating diseases, including cancer. Maybe even lung cancer?

In the meantime, universal medical opinion holds that a diet high in fats is the best way to set oneself up for heart disease. So how come the states aren't suing the fast-food industry to the tune of a few hundred billion dollars as reimbursement for the medical costs it has inflicted on society by enticing unsuspecting people through seductive advertising—*much of it aimed at hooking little children!*—to scarf down those fatally fatty hamburgers, french fries and chocolate shakes? Why hasn't the victim of a heart attack caused by clogged arteries sued one of the hamburger chains?* Surely hamburger lovers are no more re-

*It's already happened to the dairy industry. In Washington State, one Norman Mayo filed suit against Safeway stores and the Dairy Farmers of Washington, claiming that a lifetime of drinking milk contributed to his clogged arteries and a minor stroke he suffered. In addition to monetary balm, he

sponsible for their personal choices than those hopelessly "addicted" smokers who have sued the cigarette companies. What kind of secret research is this industry hiding from us? How come employers aren't refusing to hire people who are "addicted" to Big Macs, or aren't firing people caught chewing on one during lunch break, as many companies have done to smokers (see Chapter 8)?

Never fear. Once they've eradicated smoking, the healthists, the lifestyle monitors, those who know what's best for everyone, will get around to hamburgers. Indeed, some of them have already demanded that the federal government "do something" about our reckless eating habits (see Chapter 5).

All we need is a book called *Fast Food, Slow Death: What Ronald McDonald Doesn't Tell You*.

THE LONG LIST OF hazards the American Council on Science and Health says the smoker flirts with reminds me of the comic strip "Shoe." In one strip the character Cosmo takes his antique and ever-malfunctioning automobile into the shop. After the mechanic stares at the engine for a while, Cosmo asks, "What do you think it is?" "It could be anything," opines the mechanic. "That's what *I* thought," says Cosmo.

Imagine going to a doctor to find out why you're feeling so poorly lately, and even before examining you his saying, "It could be some respiratory infection. It could be an ulcer. It could be heart disease. It could be your blood sugar is up. It could be your metabolism. It could be anything."

Indeed, so many are the health hazards the smoker courts that, writes Napier in *Cigarettes*, "the full spectrum of diseases causally linked with smoking can be described collectively as 'tobaccosis.'" The term is again defined in an Afterword as "denot[ing], collectively, all those diseases resulting from the smoking, chewing and snuffing of tobacco and the breathing of tobacco smoke."

Medieval alchemists searched in vain for a "universal solvent" and an elixir that would both transmute base metals into gold and prolong

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also wanted a warning label on milk. "It's my opinion that the dairy industry is to blame," he said. "They push their dairy products without warning you of the hazards."[30]

life. We moderns have succeeded in discovering the "universal agent" of disease—"tobaccosis"—and the elixir of life is simply to avoid tobacco.

Wonderful. Now all the doctor has to do is ask you if you smoke and he can then proclaim, "Why, it's obvious what your problem is. You're suffering from tobaccosis."

Doctors, thank goodness, delve a little more deeply than that. A shoot-from-the-hip diagnosis of "tobaccosis" would be of no more value to them or to their patients than was the diagnosis of "neurasthenia" that 19th-century physicians often fell back on when they could find no obvious cause of an ailment.

Is the American Council on Science and Health seriously suggesting that medical science adopt the term "tobaccosis"? Apparently it is. It's needed, says the Afterword, because "few [people] are yet aware of the bodywide range and nature of the total spectrum of diseases caused by tobacco . . . because such diseases are treated disparately by clinical specialists and assigned numerous anatomic diagnoses devoid of the unifying term tobaccosis. Hence the basic causative role of tobacco is often missed and disregarded."[31]

(Here the attentive reader may point out that Prof. Finch also spoke of "the spectrum of diseases with which smoking has been associated." But I doubt very much that he would agree that such a varied group of diseases warranted the all-embracing suffix "osis.")

In the next paragraph the ACSH further justifies the term by noting that in the case of tuberculosis, "the records of all patients with any manifestation of tubercle bacillus infection are ordinarily labeled tuberculosis. The same is true for histoplasmosis, asbestosis, silicosis, syphilis and AIDS."

There is one problem with this. Tuberculosis is not a "spectrum" of a score of different and unrelated diseases. It is a single disease caused by *Mycobacterium tuberculosis* and related organisms, and by those organisms alone. The same with the other "osises." With the possible exception of AIDS, they also are single diseases, each caused by an identifiable germ, virus or environmental agent uniquely associated with that disease. This misuse of the suffix "osis" suggests to me that the authors of *Cigarettes* are infected with a bad case of "antitobaccosis," which in their case is indeed a spectrum—of biases, prejudices and selective interpretations of clinical evidence.

As for the book's subtitle, *What the Warning Label Doesn't Tell You*,

I trust that the council isn't advocating that the tobacco companies be required to list all the diseases, etc., allegedly caused by tobacco and smoking on their cigarette packages. The type would have to be so small you'd need a magnifying glass to read it. Anyway, smokers don't read the warning labels that are already there and nonsmokers don't need to be warned. So who would be served? Teenagers, maybe? But there's good reason to believe that the more the antismokers have tried to scare teenagers away from smoking, the more attractive they have made it to them (see Chapter 10).

Yet while doctors don't jump to a diagnosis before actually examining a patient, some of the first questions they do ask of every patient today are, "Do you smoke?" "How long have you smoked?" "How many cigarettes a day do you smoke?" "Have you ever smoked?" "How long and how much did you smoke?" Questions like that have been on every questionnaire I've filled out during a first-time visit to a doctor in recent years.

Luckily so far, in my own crossing of life's minefield, those visits have been infrequent and, except for my two rib accidents, have been for general checkups—just to make sure; after all, you know what they say about smoking.

The first such examination I'd had in many years was in 1992. I confessed my sin of smoking to the M.D., but try as he might he couldn't find anything wrong with me. Lung X-ray—clear. Sphirometer test—I blew it off the dial. Blood pressure and everything they test for in the blood—normal. Prostate gland—ditto. Cholesterol—only about 140. Muscle tone and joint articulation and reflexes—fine.

He pronounced me in good health—"at this time"—but advised me to stop smoking. Certainly I was aware, wasn't I, of all the studies showing the risks of smoking. What did I think about them?

I mumbled something to the effect that I thought a lot of studies proved what the people doing them wanted to prove. He kind of scoffed gently at that.

My heart was also okay, as far as he could tell. But he recommended I see a cardiologist for a stress test and he arranged an appointment. Once again I failed miserably to exhibit any problems, from smoking or anything else.

The first doctor also recommended I have a sigmoidoscopic examination and here, at last, they found something warranting "remedial

action." Like President Reagan, I had polyps, which can turn cancerous, growing on the walls of my colon. So I had a polypectomy. It's a fairly routine, outpatient operation which doesn't require general anesthesia, only something to put you in a kind of twilight zone. Thus I didn't need the extra anesthesia and postsurgical oxygen Whelan et al. say smokers have to have. Since then I've had a second polypectomy because the polyps started to grow again, as they did with President Reagan, but as far as I know smoking has never been blamed for this condition, even by Whelan et al.

(A sad side note is that the surgeon who performed the first operation was forced to retire in the prime of his career because of unusually severe tinnitus, a ringing, or in his case a roaring, in his ears that rendered him unable to practice any longer. I myself have mild tinnitus, but this is quite common, especially among the elderly, and also isn't blamed—yet—on smoking.)

Four years later, I visited an osteopath because of "jumpy legs" which bothered me periodically. He gave me the same complete examination as the first doctor, and the only problem he could find was a buildup of wax in my ears. But I had also confessed my irrational, self-destructive lifestyle choice of smoking and he was of course aware of all those studies in scientific journals. He thought he saw a suspicious spot on my lung X-ray and had me undergo a CAT scan at the clinic next door. When the result came back negative, he expressed amazement that I could have smoked for so many years and be so healthy. In fact, he said it twice: "I'm amazed." Doctors have been snow-jobbed along with the rest of us by the antismokers.

There's a disease of the blood vessels of the lower limbs called Buerger's disease (the "thromboangiitis obliterans" in the list above from *Cigarettes*). Seventy years ago doctors, including Dr. Leo Buerger himself, believed it was almost exclusively a "Jewish disease."[32] Today doctors believe it is almost exclusively a smoker's disease. But they can't find anything wrong with my circulation, and although Buerger's disease does apparently occur most commonly in smokers and the symptoms often disappear if they stop smoking, it sometimes afflicts nonsmokers too.

Could be that other notorious drug, caffeine, is the culprit. I drink seven or eight big cups of coffee a day. Or it could be the two shots of booze I reward myself with every afternoon after a hard day at the word processor.

Now that I think about it, "it could be anything."

What I really wonder about is whether the $450 (or whatever it was; I forget the exact amount) that Medicare coughed up as its share of the cost of my CAT scan, as well as for my previous stress test, whatever it cost, went on the books as "smoking-related" expenditures. Would that be fair, just because the doctors only ordered them because I was a smoker? Do doctors report it to the government if a patient smokes? Were my brother-in-law's years of kidney dialysis (wholly paid for by Medicare) and his other illnesses recorded as nonsmoking-related? Who records this stuff anyway? Who determines which person's medical costs are "smoking-related" and which person's aren't? How do all those states claiming reimbursement from the cigarette companies for the billions of dollars of excess Medicaid costs they allegedly foist on nonsmoking taxpayers—which really means extorting money from smokers because it will ultimately come from them in the form of higher prices for cigarettes—how do they know down to the decimal point which costs are "smoking-related" and which are not?

Well, they don't know and they don't *have* to know. It's "common knowledge" that if a smoker gets sick, it's because he smokes. (Nonsmokers never get sick, or hardly ever. Even if they do get sick, their treatment doesn't cost the healthcare system anything.) And thanks to the tobacco companies' running away with their tails between their legs, the con artists will never have to prove their claims in a court of law.

Speaking of Medicare and making people pay for other people's medical costs brings me to my most recent physical examination, in 1997, when I joined Kaiser Permanente's "Senior Advantage" program. And what an advantage it is for old geezers like me, who pay nothing for their medical and hospital insurance (except for a small amount deducted from their social security checks that they never miss), because this and similar programs for seniors are subsidized by raiding the paychecks of all those sappy baby boomers and members of the "X" generation.

This time, fed up with feeling apologetic about smoking, I straightout lied and told the doctor I had never smoked. I had debated about it with myself, and decided that omitting this information would not compromise the doctor's ability to establish the state of my health; to the contrary, eliminating the extraneous issue of smoking would aid him. Too late, it afterwards occurred to me that I might also have inadvert-

ently confirmed any belief he might have that only nonsmokers are healthy. Oh, well.

"No history of smoking," he said as he wrote that down. Because of that (I'm guessing) he didn't even have my lungs X-rayed. At least that's one cost to Medicare they couldn't record as "smoking-related." I consider this as both a personal act of revenge against the states' attorneys general and a small way of thanking the boomers and X-ers, who are maintaining me and all seniors in a style of living no previous older generation in history ever enjoyed.

Anyway, once again I was pronounced in fine fettle—except my cholesterol was up to 148.

Finally, in early 1998 I had an operation to remove a hydrocele, a fluid-filled sac, from a personal area of my body. I'd had it for years and every doctor said not to worry about it, hydroceles never turned cancerous. But it had grown quite large and uncomfortable. When the Kaiser urologist said he preferred to do the surgery under general anesthesia (the patient under it, that is, not him), I felt a little trepidation. I hadn't had general anesthesia since my tonsillectomy at age 5. They didn't know I was a smoker. What if they didn't give me enough anesthetic or enough oxygen? What if I choked on phlegm? What if Whelan et al. were right?

But all went well. I was no sooner put under than it seemed that they were bringing me out of it. I asked the anesthesiologist how I'd done. Just fine, she said. How was my oxygen level? Also fine. I then told her that I'd been smoking cigarettes for 52 years. She expressed no astonishment at, or even much interest in, that revelation. Which confirmed my belief that it is the antismoking zealots who manufacture these scares about smoking, not your workaday physicians.

Yet for all my familiarity with the tactics of the zealots, I'd let them sucker me in by planting unnecessary fear in my mind.

All of which personal history is again purely anecdotal and proves absolutely nothing, though I'm sure it has been intensely fascinating to the reader.

WHERE WAS I? Oh, yes, Dr. Whelan. I had lunch with her one day in 1975 when she was in Cleveland promoting her first book, *Panic in the Pantry* (co-authored with Frederick J. Stare, M.D., chairman of the department of nutrition at Harvard School of Public Health, and with

her a founder of the American Council on Science and Health). I was happy to help publicize her book with an editorial mentioning it because it presented a nonalarmist view of and effectively debunked a flurry of scares at that time about the dangers of additives and pesticides and other suspect stuff in the food we ate.

I don't remember if I smoked in the restaurant, but if I did she said nothing about it. I do know that smoking was not discussed during our lunch. But this was before secondhand smoke hysteria started and before smoking became a social offense as bad as urinating on the floor, so I may have smoked. If I did, and if she was as vehemently antismoking then as she is today, she exhibited considerable politeness and self-restraint in not lecturing me. Her favorite terms for the cigarette companies are "merchants of death" and "purveyors of poison" and she has written at least one other antismoking book, which I haven't read, *A Smoking Gun: How the Tobacco Industry Gets Away With Murder*.

It saddens me that a person who demonstrated, and still does demonstrate, so much common sense when it comes to exaggerated environmental dangers can not only so sincerely believe that smoking is either implicated in or is *the* cause of a multitude of health problems but can so earnestly promote that belief—and the fears it engenders—to the general public.

Not that she or the specialists who reviewed each chapter in *Cigarettes* are, as Dave Barry would say, making anything up. They cite study after study. They are also reasonably honest; if there are conflicting studies, with one indicting smoking in this or that disease and another finding no evidence against smoking, they tell the reader. But then they take it back.

For instance, on page 19 they note that there is an abundance of literature on smoking and prostate cancer and that most of it does not support an association. However, this is followed by a sentence, which I quote in full below, to the effect that smoking increases the severity of this cancer.

On page 106 they note that the risk of Alzheimer's disease appears to decrease the more cigarettes are smoked per day and the more years they are smoked, possibly because smoking boosts the function of the neurotransmitter acetycholine. But then they say that other researchers postulate (I think they mean "suggest") that it is actually a difference of genes in smokers and nonsmokers that accounts for this

"paradox" rather than any protective effect of smoking. A difference in genes is never "postulated" in regard to any other disease, however.

On page 147 they note that a bacterium, *Helicobacter pylori*, is associated with an increased risk of developing ulcers, which occur in smokers and nonsmokers alike. (This bacterium is far more than merely "associated" with ulcers, as I mentioned above.) But smoking decreases immunity, they say, thus lowering resistance to infection by this organism.

I also find it significant that throughout the book it is stated that "one study" has estimated this or "one study" has found that.

Some examples from just the first chapter:

"One study has estimated more than a doubling of penile cancer among men who smoke more than 10 cigarettes a day compared with nonsmokers. Another study has estimated more than a tripling of risk for people with a history of more than 45 pack-years." (Page 17)

"One study has estimated that the risk of anal cancer is elevated by almost eight times for heterosexual men who smoke and by more than nine times for women who smoke." (Page 18)

"One study found that smokers had a higher incidence of more invasive and high-grade prostate cancer than did nonsmokers." (Page 19)

Such statements are always followed by a reference number, with the study identified at the end of the chapter. This certainly doesn't mean that the authors could not have cited more than the one study regarding a particular disease or disorder or condition, etc. Frequently they do in fact reference several studies in support of a statement. But the book is aimed at a general audience, not a scientific one, so the multitude of references that typically buttress an article in a scientific journal isn't necessary here.

Yet allowing for that, the fact that *Cigarettes* relies so often on single studies suggests to me that the book ought to carry a warning label of its own, perhaps borrowing the caution of one statistician:

> [M]aking one study is about the same as throwing a pair of dice, once, observing that the result is, say, ten, and proclaiming "When you throw dice, the number ten comes up" . . . Never read anything, or listen to anyone, if the opening statement resembles "According to one study . . . "[33] [Second ellipsis in original.]

To wind up this chapter, let's look at two examples of "one study," each of which was conducted on a different battlefront in the crusade

against smoking and both of which were ballyhooed as having broken new ground in confirming the health dangers of cigarettes.

The first was not an epidemiological study that observed living persons but a laboratory study which, its authors claimed, led them to the holy grail of the antismoking movement—the elusive, long sought-after "smoking gun," the exact biological mechanism by which smoking causes lung cancer, or, as *The Wall Street Journal* put it, "the final link in the chain of evidence."[34]

This study involved a gene called the "p53" gene. In the late 1980s, according to the *Journal*, researchers at Johns Hopkins in Baltimore had discovered that p53 is a tumor suppressor that regulates cell division and keeps cells from going out of control and turning cancerous. Because it performed that function, it was called "the guardian angel" gene. It was "suggested" that damage to that gene by cigarette smoke "could" cause lung cancer.

Here already I get a little confused because in April 1996, cancer researchers at the Kimmel Cancer Center at Thomas Jefferson University in Philadelphia reported in the scientific journal *Cell* that they had found a gene they called FHIT (fragile histidine triad) which functioned as a shield to block the growth of cancerous tumors and that, when damaged by cigarette smoke, played "a critical role in the development of lung cancer."[35]

Whether FHIT is another name for p53 or is a different gene entirely, the big news came a few months later when, in October, a team of cancer researchers at the M.D. Anderson Cancer Center in Houston announced that they had found a "direct link" between cigarette smoke and damaged p53. The discovery was reported in *Science*, the journal of the American Association for the Advancement of Science, in an article titled "Preferential Formation of Benzo(a)pyrene Adducts at Lung Cancer Mutational Hotspots in *P53*,"[36] authored by biochemist Moon-Shon Tang and colleagues.

(The print and television news media were also informed, of course. But while ABC's Peter Jennings and NBC's Tom Brokaw merely told viewers about it, CBS's Dan Rather played it up big as usual by running video footage of Tang making the historic announcement at a press conference in Houston.)

Benzo(a)pyrene, or BAP, had been a leading carcinogenic, or cancer-causing, suspect in cigarette smoke even before the 1964 surgeon

general's report. According to Tang et al., however, it isn't BAP but a metabolite of BAP, benzo(a)pyrene diol epoxide, or BAPDE, that is the nemesis of the p53 gene. (A metabolite is a substance produced by an organism's natural process of metabolism. Metabolites flow into the bloodstream where they are carried throughout the body, including of course the lungs.)

Tang et al. stated that in about 60 percent of lung cancer cases, there is mutational damage to the p53 gene. The Philadelphia researchers had stated that the FHIT gene was mutated in 80 percent of the lung tumors they tested. In either case, it is the same as saying that in 20 to 40 percent of lung cancer cases there is *no* damage to the genes. Thus whichever the gene and whatever the percentage, a logical conclusion is that people can get lung cancer even if their genes are in perfect, unmutated condition.

This is only one obstacle in the way of accepting Tang et al.'s finding. In a critique of their experiments, Lauren Colby, who seems to be one of the few commentators who actually read the *Science* article, points out that the researchers did not study any actual human lung cancers. Rather, they studied cultured human cells, which they exposed to BAPDE and then tested for mutational damage. He writes:

> Not having any humans to work with, the authors of the study compared the mutations which they had induced with specimens of DNA taken from a gene data base, compiled by others. Now, if the goal of the study was to prove that BAP from smoking causes lung cancer (and that was, indeed, the goal), it would seem to be scientifically necessary to compare the genes of smokers who fall victim to lung cancer with those of non-smokers who fall victim to the disease. Such a comparison would show whether lung cancer in smokers has a different etiology (cause) than in non-smokers.
>
> The authors of the study, however, deliberately excluded from the study any DNA samples obtained from non-smokers or from "radon associated cancers." They did not say how they knew whether any particular samples came from non-smokers or were "radon associated"; apparently they took the word of the people who compiled the data base. The point is, however, that while all experiments should always be controlled, these authors deliberately threw out the controls![37]

In my own opinion, the first and biggest obstacle to pinning the blame for lung cancer on the BAP in cigarette smoke is, as I noted in

Chapter 1, that BAP is a ubiquitous substance in the environment. It is produced by the combustion of vegetation and fossil fuels as well as the cooking of food. In fact, better than 90 percent of the BAP consumed by humans, including smokers, comes from food.

Colby continues:

> The authors of the study apparently concede that BAP, in and of itself is not terribly carcinogenic (although, like any irritating substance, it will produce skin cancers in specially bred "nude mice"); it must be converted to BAPDE. There is no evidence that the lungs, themselves, can metabolize BAP into BAPDE. Even if they could, the amount of BAP reaching the lungs from cigarette smoke is dwarfed by the amount reaching the lungs in the blood supply (and already metabolized into BAPDE) from consumption of burnt food. Thus, at the outset, the study appears flawed. However, it gets worse!
>
> The authors make the astonishing statement that "This study provides a direct link between a defined cigarette smoke carcinogen and human cancer mutations." I say "astonishing," because the study dealt with BAPDE, not BAP, and there is no BAPDE in cigarette smoke. Thus, at best, the study could claim only an "indirect link." But, because of the failure to take into account the BAP consumed in food, it isn't possible to claim even an "indirect link." The study could just as well be said to prove an indirect link between the consumption of burnt food and lung cancer. However, it doesn't prove even that, because (a) it does not explain lung cancer in the 40% of victims who have no p53 gene damage and (b) the authors compared their results with DNA samples which they selectively picked and chose, throwing out those which they deemed to be "radon associated" or from non-smokers (free translation: throwing out those that would not have validated their conclusions).

This is a devastating analysis—and why was it left for a nonscientist to make it? To my knowledge, no scientist has pointed out the fatal deficiencies in the Tang study. At the same time no researcher has even bothered to try to replicate it, which is the way things are supposed to be done in science. We haven't heard anything more about this "smoking gun" since October 1996. The p53 gene seems to have gone . . . *fhit*. Yet as far as the general public is aware, science now "knows" exactly how smoking causes lung cancer.

This p53 gene business only deepens the mystery for me. Whether it is BAPDE or some other metabolite in the blood that sets off lung

cancer, or whether it's something in cigarette smoke acting directly on lung tissues, why should it take 20 or 30 or 40 or more years for a tumor to appear?

I can understand how microscopic particles of asbestos or coal dust can reside in the deep recesses of the lungs for many years before they cause mesothelioma or black lung disease. That is, I can visualize some sort of "festering" process. But a pathologist dissecting the lungs of someone who died of lung cancer has no way of knowing whether the victim smoked or did not smoke. There are no residues of anything from cigarette smoke in the lungs and no histological (tissue) differences between a smoker's tumor and a nonsmoker's. In fact, the pathologist cannot tell whether the cancer originated in the lungs or whether it metastasized from some other organ. The only way he can "know" that smoking caused the cancer is if the victim's medical record shows a history of smoking during some period in his life.

Actually, the search for the biological mechanism by which smoking causes (or may cause) lung cancer always was a fruitless and unnecessary quest—and not just because everybody, laymen and doctors alike, everybody with half a brain, already "knows" that it does. There are any number of diseases or conditions whose causative agents have been identified but medical science doesn't fully understand the exact mechanism by which they affect the body, yet nevertheless has ways to treat them. For example, in the 1960s doctors in Europe became aware that women who had taken the new sedative thalidomide in early pregnancy were giving birth to babies with undeveloped limbs. Even though they didn't know exactly how the drug worked its mischief on the fetus, they didn't *need* to know in order to remedy the situation.

However, there is an important difference between knowing from sad experience that thalidomide causes birth defects and that the "cure" is simply to prevent pregnant women from taking the drug, and "knowing" from epidemiological studies that cigarette smoking causes lung cancer and attempting to frighten or force people to give up the habit.

If some madman were to administer thalidomide to a group of women at the right stage of pregnancy, he could predict with certainty that all, or certainly most, of them would deliver defective babies. But not only does a mere one out of 10 smokers ever develop lung cancer (we're told), no one can possibly predict which one. Our knowledge of thalidomide is the result of the direct observation of its effect on real

people; our "knowledge" about cigarette smoke is a statistical abstraction from epidemiological studies. No matter how many people we can cite who smoked and died of lung cancer, this evidence remains at bottom purely anecdotal. It may be highly suggestive, even persuasive, evidence, but so is my anecdotal evidence in the other direction.

We're back again to Prof. Finch and the question of chances. With something like thalidomide, the chance of harm is virtually certain. With smoking, it is, to paraphrase him, relatively infrequent.

IN THE MEANTIME, although we also "know" that secondhand smoke kills, dedicated researchers continue to pile up what they would have the public believe is convincing evidence in that area. The second "one study" is perhaps even more important than the first, both because it is one of the most recent in this field and because of its social ramifications. It was announced by the American Heart Association on May 20, 1997:

> DALLAS, May 20—Constant exposure to second-hand smoke—in the workplace or at home—nearly doubles the risk of having a heart attack, a landmark study of more than 32,000 women suggests. Results of the research appear in today's American Heart Association journal *Circulation*.[38]
>
> Boston scientists say their 10-year investigation involving female nurses found a higher level of risk from passive smoking than has been seen before and provides the strongest evidence yet that exposure to smoke in the workplace is as dangerous as exposure at home.
>
> Healthy, non-smoking nurses who said they were regularly exposed to "passive" smoking by their co-workers or home companions had a 91 percent higher relative risk of a heart attack or death, compared to nurses who were not subjected to smoke, the researchers at Brigham and Women's Hospital and Harvard Medical School report. For nurses who reported only occasional exposure to smoking in work or home environments, heart disease risk was 58 percent greater.
>
> "Those are larger risks than have been previously reported," says Ichiro Kawachi, M.D., the study's lead author . . .[39]

Reaction of the media was predictable. A few examples:

The Washington Post: ". . . the largest study ever conducted on the issue . . . provides strong new evidence . . . " (John Schwartz, staff writer.)

The Los Angeles Times: ". . . the largest study of its kind ever con-

ducted . . . one more powerful scientifically based argument for banning all smoking in the workplace . . . " (Commentary.)

Associated Press: "The findings . . . could help advocates of a nationwide ban on smoking in workplaces . . . " (Melissa Williams, AP writer)

USA Today: " . . . Passive smoking is deadly . . . The U.S. Occupational Safety and Health Administration has dallied since 1994 over a workplace smoking ban, trying to accommodate smoking interests. It's time it saved some lives instead." (Editorial, "Bring on the Ban".)

No newspaper informed its readers that a "91 percent greater risk" meant that the researchers had established a relative risk of a mere 1.91, which it may be remembered from Chapter 1 is barely significant, if it is significant at all. A "58 percent greater risk," or a relative risk of 1.58, is even less significant. Yet that 91 percent risk was represented by the American Heart Association as nearly a doubling of the risk of a heart attack.

Not that there is anything new about this. Researchers and institutions have always reported statistics in ways that are most easily understood (actually, misunderstood) by the general public and that make their "findings" look important—although the antismoking movement has raised this technique to a high state of perfection. As one student of the subject wrote a full decade before the crusade against smoking was launched, "The secret language of statistics, so appealing in a fact-minded culture, is employed to sensationalize, inflate, confuse, and oversimplify."[40]

Some of the other findings of the Brigham and Women's/Harvard study the newspapers did not report:

• "Among women exposed only at work, the multivariate relative risks of total CHD [coronary heart disease] were 1.49 (95% CI [confidence interval], 0.71 to 3.14) among those occasionally exposed and 1.92 (95% CI, 0.88 to 4.18) among those regularly exposed to secondhand smoke." (Page 2376 of *Circulation*.)

(Another reminder: when the lower boundary of a confidence interval range is below one, the result of that calculation is considered to be statistically insignificant by every authority on epidemiology.)

• "The multivariate relative risks of total CHD were 1.81 (95% CI, 1.08 to 3.02) in women exposed at home or work and 1.36 (95% CI, 0.72 to 2.54) in women exposed in both settings. However, the data on

women exposed in both settings was based on a small number of cases (n=24)." (Page 2376.) [Which is rather a long way from 32,000!—D.O.]

• "There was no relation apparent between duration of living with a smoker and risk of CHD." (Page 2374.)

• "Unknown confounding factors may have contributed to the observed excess risk . . ." (Page 2377.)

• "A limitation of the present study was its reliance on self-reported assessments [anecdotal evidence—D.O.] of exposure to passive smoking." (Page 2377.)

• "A further limitation of this study is that exposure to passive smoking was ascertained only at baseline." (Page 2378.)

What that last sentence refers to is that only at the beginning of the study (1982) were the nurses asked how much cigarette smoke they "thought" they were exposed to. They were never asked again in the 10-year duration of the study.

The above passages from the study were published on the World Wide Web by the National Smokers Alliance, a smokers' advocate group partially supported by the tobacco industry.[41] The following critique by Martha Perske was also published by the NSA (but who else would publish it, certainly not the unbiased mainstream media). Ms. Perske is, like me, a private citizen with no medical credentials. She is an award-winning artist and stamp designer. However, she possesses common sense and uses it in a new vocation dedicated to exposing the exaggerated claims, if not outright fraudulent "science," of the antismoking movement. She writes (with underlining and other emphases hers):

> The Harvard study—supported by public funds from the National Institutes of Health—seems to have been presented to the media in a disingenuous way, apparently in an attempt to rouse public resentment and promote smoking bans.
>
> For example, not mentioned in the Harvard press release or the American Heart Association's website release is the fact that <u>this study found no statistically significant increased risk from exposure to secondhand smoke in the workplace.</u> Instead, the American Heart Association's release states, "The finding of high risk associated with workplace smoke is a very important one, Kawachi emphasizes." *What high risk?* The finding wasn't even statistically significant!
>
> In the Harvard press release, Kawachi says that the "strong association" suggests that secondhand smoke is responsible for up

> to 60,000 deaths each year from coronary heart disease, and *"From the standpoint of alleviating risk, the good news is that certainly smoke-free building policies would go a long way to changing this picture."*
>
> By presenting their results according to exposure at home OR work, the Harvard researchers were able to make it appear there was risk from workplace exposure (and thus a need for smoking bans), when in fact there was no statistically significant increased risk at all from workplace exposure alone. All significant findings reported by the Harvard researchers involved exposure in the home, and even those are questionable . . .
>
> [V]irtually all of the relative risks reported in the Harvard study are below 2.00, and according to the National Cancer Institute, relative risks below 2.00 are considered "small" and could be due to statistical bias, confounding factors, or chance. Not exactly the "high risk" claimed by Kawachi.

Perske also calls attention to these shortcomings:

> 1. The study—while erroneously touted in the press as the largest ever done—was based on only 152 coronary heart disease cases, 25 of which were fatal. The largest studies ever done were based on data from the American Cancer Society and reported no increased CHD risk from workplace exposure and no statistically significant increased risk for women married to smokers.
>
> 2. There were no measurements of actual exposure to secondhand smoke. Study subjects were asked if they were "occasionally" or "regularly" exposed to secondhand smoke, and they were asked this question only once, at the beginning of the 10-year study. Follow-up questionnaires were mailed to study subjects every 2 years to update information on cardiovascular risk factors, etc., but no attempt was made to update self-reported information on secondhand smoke exposure.
>
> 3. Study subjects were all female nurses, not representative of the U.S. female population. Nurses often work under stressful conditions, yet the Harvard team did not consider or adjust for "stress."[42]

Alas, she concludes, "This is yet another secondhand smoke study that has been overblown in the media. Unfortunately, after it's been sensationalized in neon lights, it's next to impossible to get the media to take a second look, let alone investigate the possibility that the public (whose money was used to fund the study) was knowingly misinformed in order to advance the anti-smoking agenda."

And once again it was left to a layperson to expose the fallacies in

an antismoking study, while the scientific community remained silent.

I don't know who financed the p53 gene experiment, but would it be altogether too cynical of me to suggest that both it and the nurses' study (not to mention many other such studies) were conducted because there is a great deal of grant money—much of it lifted from the pockets of smokers—available from the government and private health associations for just about anyone with minimal scientific credentials and a half-baked theory who wants to cash in on the antismoking crusade? It's the best way for a researcher to get his name in print and enhance his career these days.

Speaking of nurses and stress and half-baked theories, Dr. Kawachi headed another team of researchers who studied another bunch of nurses—86,626 of them no less—from 1980 to 1990 and "discovered" that coffee drinkers are less likely to commit suicide than those who don't imbibe. (Note carefully the tense: "coffee drinkers *are* . . ," not "coffee drinkers *were* . . ." Researchers try to avoid using the past tense, for that might suggest to the public that the findings of their studies had limited import, applying only to the actual participants in the studies at the time of the studies and not to everyone in the world for all eternity.)

Specifically, out of those 86,626 nurses the study recorded 11 suicides among those who drank two cups of coffee per day, compared with 21 suicides among those who said they almost never drank coffee. To give it a number, drinking two or three cups of coffee a day reduced the risk of suicide by 66 percent. Kawachi cautioned, however, that the results may not be significant because doctors might have told nurses who were depressed (for any number of reasons) not to drink coffee.[43]

In any case, one presumes that the nurses who committed suicide had told the researchers about their rate of coffee drinking *before* they killed themselves. Somehow, even though I'm a coffee drinker, reading about that study left me very depressed.

I don't know if this coffee-drinking group of nurses included any of the 32,000 secondhand-smoke-inhaling nurses in Kawachi's other study that was going on at the about the same time, but obviously the doctor was a busy man. He is also a prime example of a researcher who has found rewarding lifetime employment thanks to the antismoking movement and Americans' general hypochondria.

As for why reputable journals publish studies like these in the first

place, that is another question entirely. Where is the peer review that is supposed to weed out this kind of junk science?

In the next chapter I'll examine some even more egregious examples of of junk science conducted in the cause of a "smoke-free society."

Notes

1. Quoted in *The New York Times*, October 11, 1995**,** and cited in "Secondhand Smoke-and-Mirrors (How the Pros Tamper with Jury Pools)." Posted on Steven Milloy's Junk Science page at www.junkscience.com/news/mirrors.htm. Hennekens was one of seven authors of the notorious secondhand smoke study of nurses published in *Circulation* (see Note 38 infra).

"[T]he whole apparatus of grants-and-research projects . . . discourages candid acknowledgement that one may work for years at what turns out to be a dead end, and constitutes a standing encouragement to exaggeration, half-truth and outright dishonesty about what one has achieved."—Susan Haack, professor of philosophy at the University of Miami. *Skeptical Inquirer*, November/December 1997, p. 38.

2. John H. Fennick, *Studies Show: A Popular Guide to Understanding Scientific Studies* (Amherst, N.Y.: Prometheus Books, 1997), p. 69.

3. Peter H. Gott, M.D., "Health Warnings: Enough Is Enough." Column distributed by Newspaper Enterprise Association, December 5, 1993.

4. Martin Feinstein, Ph.D., *Statistics*, in Barron's EZ-101 Study Keys (Hauppage, N.Y.: Barron's Educational Services, 1994), pp. 49 and 50.

5. Unattributed quote in Laurie McGinley, "Of Mice and Men: How Ex-Lax, Trusted for Nearly a Century Became a Cancer Risk." *The Wall Street Journal*, September 26, 1997. At www.junkscience.com/news.exlax.htm.

6. "Manganese Mangles Mice Experiments." At www.junkscience.com/news/manganes.html.

7. Quoted in Fennick, p. 10.

8. "Study Was Wrong." News & Notes. *The Atlanta Journal-Constitution*, December 18, 1997, p. E3.

9. Christine M. Albert, M.D. et al, "Fish Consumption and Risk of Sudden Cardiac Death." *Journal of the American Medical Association*, 1998;279:23-28 (January 7, 1997). At www.junkscience.com/news/eatfish1.htm.

10. Doll, R. and Bradford Hill, A., "Lung Cancer and Other Causes of Death in Relation to Smoking." *British Medical Journal*, November 10, 1956, pp. 1072-81.

11. Peter D. Finch, "A Closer Look At Statistics on Smoking and Health." From ftp://demon.co/uk/doc/liberty/FOREST/.

12. Breslow, N. and Day, N., "Statistical Methods in Cancer Research." IARC (International Agency for Research on Cancer) Scientific Publications No. 32 (1980), Lyon, Table 2.1, p. 68.

13. Gary T. Schwartz, "Tobacco Liability in the Courts," in Robert L. Rabin and Stephen D. Sugarman, eds., *Smoking Policy: Law, Politics, and Culture* (New York: Oxford University Press, 1993), p. 133.

14. "They can't kick the habit: Smokers' ranks decline little." *The Atlanta Journal-Constitution*, December 26, 1997, p. C5. From the Associated Press.

15. American Council on Science and Health, *Cigarettes: What the Warning Label Doesn't Tell You* (Amherst, N.Y.: Prometheus Books, 1997), p. 10.

16. B. Bruce-Biggs, "The Health Police Are Blowing Smoke." *Fortune*, April 25, 1988. Quoted in David Pietrusza, *Smoking* (San Diego: Lucent Books, 1997), p. 31. Pietrusza didn't identify Bruce-Biggs so I looked up the *Fortune* article and found that he is, or was, an independent policy analyst with no connection with the tobacco industry and a smoker of 30 years' standing. I don't know if he's still standing.

17. "USA Statistics in Brief." From http://ftp.census.gov/stat_abstract/brief.html.

18. American Cancer Society, "Cancer Risk Report, 1995." At www.cancer.org/crrtobac.htm.

19. Letters. *Reason*, April 1996, p. 10.

20. *The Merck Manual of Diagnosis and Therapy*, Sixteenth Edition (Rahway, N.J.: Merck Research Laboratories, 1992), pp. 830, 834.

21. "Inherited genetic disorders." *The Atlanta Journal-Constitution*, March 24, 1993, p. B3.

22. "Coronary Artery Disease, Risk Factors and Detection." North Fulton (Georgia) Regional Hospital *Health Focus*, Winter 1997-1998, p. 7.

23. Ibid.

24. Ilene Springer, "Castelli speaks from the heart." *AARP Bulletin*, May 1992, p. 16.

25. Loc. cit.

26. David Guarino, "The heart of health for seniors; Cardiologist Castelli tells forum goers they must watch diet." *Middlesex News*, November 13, 1997, p. 1A.

27. "Deaths and death rates for the 10 leading causes of death in specified age groups: United States, preliminary 1995." At www.cdc.gov/nchswww/datawh/statab/pubd/453s216h.htm.

28. *Cigarettes*, p. 26.

29. Michael Mason, "Are Heart Attacks Contagious?" *Hippocrates* 1997; 11(12):42-45. At www.medscape.com/time/hippocrates/1997/v11.n.12/h1112.01.maso.html#Beyond.

30. "Brickbats." *Reason*, October 1997, p. 20; *The Atlanta Journal-Constitution*, January 1, 1997, p. A17; R. Cort Kirkwood, "Warning: Idiots may be hazardous to their health." *The Ottawa Sun*, June 11, 1997. At www.canoe.ca/OttawaSun/home.html.

31. *Cigarettes*, p. 169.

32. Bergen Evans, *The Natural History of Nonsense* (New York: Vintage Books, 1946), p. 216 in 1958 paperback reprint.

But even while this renowned rationalist was puncturing many of the fables and myths men live by, he uncritically passed on this piece of nonsense: "Irritability, restlessness, impaired memory, depression of spirits, insomnia, headaches, and fatigue have all been demonstrated to be the physical consequences of excessive smoking. Even two cigarettes can produce measurable dulling of sensitivity and increase of tremor." (Ibid. pp. 160,161.) Evans obviously was a nonsmoker with no firsthand acquaintance either with cigarettes or with people who used them.

33. Fennick, pp. 225-226.

34. "Researchers Show How Smoking Causes Cancer." *The Wall Street Journal*, October 18, 1996, p.1.

35. Associated Press, "Damaged Gene May Open Pathway to Lung Cancer." *The Washington Post*, April 7, 1996, p. A13; Associated Press, "Damaged Gene is Linked to Lung Cancer." *The New York Times*, April 6, 1996, p. A24. Sources courtesy of The Advocacy Institute Also CNN correspondent Andrew Holtz, "Genetic breakdown linked to lung cancer." Cited on the University of Arizona's Internet site at http://hinet.medlib.arizona.edu/~pubhlth/04099601.htm.

36. *Science* 1996;274:430-432.

37. At www.lcolby.com/addendum.

38. *Circulation* 1997;95:2374-2379.

39. "Steady exposure to 'passive' smoke nearly doubles risk of heart attack death for women, study shows." At www.amhrt.org/news/974531.html.

40. Darell Huff, *How to Lie With Statistics* (New York: W. W. Norton, 1954), Norton paperback reissue 1993, p. 8.

41. At www.speakup.org/harvrel.html.

42. Martha Perske, "Alarmist Media Reports Regarding The Harvard Study by Kawachi, et al." At www.speakup.org/harvprsk.html.

43. Ichiro Kawachi et al., *Arch Intern Med* 1996;156:521-525. At www.junkscience.com/news/coffee-suicide.html. Also the Nashville *Tennessean*, April 17, 1996, p. D-1; Bill Hendrick and Amanda Husted, "Women and coffee." *The Atlanta Journal-Constitution*, March 11, 1996. p C2.