



REVIEWS AND COMMENTARY

When Genius Errs: R. A. Fisher and the Lung Cancer Controversy

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R. A. Fisher's work on lung cancer and smoking is critically reviewed. The controversy is placed in the context of his career and personality. Although Fisher made invaluable contributions to the field of statistics, his analysis of the causal association between lung cancer and smoking was flawed by an unwillingness to examine the entire body of data available and prematurely drawn conclusions. His views may also have been influenced by personal and professional conflicts, by his work as a consultant to the tobacco industry, and by the fact that he was himself a smoker. *Am J Epidemiol* 1991; 133:416-25.

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Editor's note: For a discussion of this paper and for the author's response, see pages 426 and 428, respectively, and page 429.

All men are liable to error; and most men are, in many points, by passion or interest, under temptation to it. . .

—John Locke, *An Essay Concerning Human Understanding*, Chap 20, Sec 17

In an article published in 1989, Jan Vandembroucke (1) briefly reviewed the lung cancer controversy three decades ago when two prominent statisticians, J. Berkson and R. A. Fisher, doubted that cigarettes caused lung cancer. Vandembroucke described the "...well-written and cogent papers that might have become textbook classics for

their impeccable logic and clear exposition of data and argument if only the authors had been on the right side. . ." (1, p. 3).

My reading of everything Fisher ever published on the subject—five letters, two lectures, and an article—revealed almost precisely the opposite of Vandembroucke's evaluation: incomplete and highly selected data (or no data but much speculation), with scant attempts to weigh the evidence or reveal the obvious deficiencies in his data. Fisher sounds like a man with "an axe to grind." I have tried to piece together the story of his involvement in this controversy and to place it in the context of his career and personality. Fisher was not just on the "wrong side," to use Vandembroucke's terms; he was unwilling to seriously examine the data and to review all the evidence before him to try to reach a judicious conclusion.

In this article, I briefly review Fisher's life and times and then present his own data and arguments. Readers can then judge for themselves if these are "textbook classics for their impeccable logic" (Vandembroucke's phrase), or if they are much less.

Who was Fisher? What was his back-

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ground? What were his contributions to statistics? How did he get interested in smoking and cancer? What were his arguments? Why did he refuse to perceive the causal nature of the association?

FISHER'S BACKGROUND

The problem that confronts the historian is that most of what we know of Fisher comes from the lengthy biography written by his adoring daughter, Joan Fisher Box (2). She also wrote the biography of Fisher that appears in the *Encyclopedia of Statistical Sciences*. Married to George Box, a famous statistician at the University of Wisconsin, she sides with Fisher in his many controversies with Karl and Egon Pearson, with Neyman, and with Gossett. Her account of his involvement with the lung cancer controversy is typically uncritical and one-sided. This historical resource is valuable but must be taken for what it is, a daughter's loving account of her famous and domineering father.

R. A. Fisher was born in a suburb of London in 1890 and died in Adelaide, Australia, in 1962 of colon cancer. His father was a fine arts auctioneer who worked in the West End of London, but his business failed when Fisher was 16, so economic circumstances were modest. In 1904, when he was 14, his mother died of peritonitis; that same year he won a scholarship in mathematics to Harrow, the famous private academy. There he demonstrated his talent early on by winning a math medal.

At age 19, Fisher was awarded a mathematics scholarship to Cambridge University and in 1912 at 22 was made a "Wrangler," a high academic achievement. He studied statistics and physics under Jeans. In 1912 he published his first paper wherein the concept of maximum likelihood was introduced. He corresponded with Gossett (who published under the pseudonym of "Student") about some of his pioneering work. He was very interested in evolutionary theory and genetics and combined this interest in the eugenics movement, now discredited,

but very avant-garde before World War I. In 1911, Fisher helped form the Cambridge Eugenics Society and gave a speech at the second annual meeting. He was concerned that the birthrate fell by social class, with those of the highest class having the fewest children. To the eugenicists, this implied selection against "qualities" thought to bring success and achievement in life. They believed this would lead to "degradation of the genetic stock" and, thus, the decline of civilization. The connection between eugenics and racist theories has been pointed out by many authors, most recently by Kevles (3). To encourage those with excellent "qualities" to produce offspring, Fisher urged the passage of a family allowance. The argument went as follows. The most intelligent figure out that they can improve their opportunities in life and their economic conditions by having fewer children; the less intelligent don't understand this and have more children. Fisher proposed as a solution a state subsidy of the intelligent to encourage them to procreate.

Fisher was active in the London Eugenics Society for 20 years and had many children himself.

FISHER'S CAREER

In 1913 he took a job as a statistician with a London bank and trained with the Territorial Army. At the outbreak of war in August 1914, he volunteered immediately to fight for Great Britain. Rejected for bad eyesight, he served his country during the next 5 years by teaching high school physics and mathematics, a job he disliked but continued, according to his daughter, as a public service. One wonders what would have happened (or rather, not happened) to the field of statistics if he had entered the infantry and been killed, as were nearly a million young British men.

In 1917 he married Ruth Guinness and immediately started a family. His income was limited, and he farmed to provide for the family.

About the time of World War I, the "dean" of all English statisticians was Karl

Pearson. He took notice of Fisher's work and genius and, as editor of *Biometrika*, arranged to publish some of Fisher's articles. Pearson published Fisher's paper describing the general sampling distribution of the correlation coefficient. When Pearson published another article by Fisher about maximum likelihood and editorially criticized it without first informing Fisher he would do this, Fisher developed a strong antipathy for Pearson, the first of Fisher's several feuds.

In September 1917, Fisher started work as a statistician at Rothamsted experimental agricultural station which, under his leadership, was to become a world center for the theoretical development of experimental design. There he developed the analysis of variance, the principle and contribution of randomization, and the idea and importance of replication. He made great contributions to the understanding of confounding and created designs to handle problems created by confounding. In 1925 he published *Statistical Methods for Research Workers* (4), and 10 years later *The Design of Experiments* was published (5). In 1938 he and Frank Yates brought out *Statistical Tables for Biological, Agricultural, and Medical Research*, still used today (6). (See figure 1.)

Following up his work on the distribution of the correlation coefficient, Fisher derived the sampling distributions of other statistics in common use, including the *F* distribution and the multiple correlation coefficient. He developed the theory of estimation in 1922. In later years he made many other contributions to genetic and evolutionary theory that are considered central to the understanding of the theory of natural selection (7).

Fisher was offered the chair as the Galton Professor of Eugenics at University College in London. Actually, a new Department of Eugenics was created in order to attract him to the University. Fisher would never have agreed to work in the statistics department under Karl Pearson because of the antipathy between them which had originated with Pearson's critical editorial in *Biometrika*. Consequently, two departments doing the same kind of work coexisted at the Univer-



FIGURE 1. Passport photograph of Ronald Aylmer Fisher at age 34. Reprinted from Box JF. RA Fisher: the life of a scientist. New York: John Wiley & Sons, Inc., 1978.

sity College—Statistics under E. G. Pearson, who headed the department after his father, and statistics (misnamed Eugenics) under Fisher. An intense rivalry and bad feeling existed between Pearson and Fisher which was reflected in their departmental activities.

Jerzy Neyman joined Egon Pearson in Statistics in 1934 and immediately challenged some of Fisher's ideas on hypothesis testing, introducing the ideas of power and decision theory which he developed further in the United States with Abraham Wald. Fisher was unaccustomed to being contradicted and confronted Neyman as follows (related to Constance Reid by Neyman when he was an old man in working retirement at the University of California, Berkeley):

And he said to me that he and I are at the same building. . . he had published a book and that's *Statistical Methods for Re-*

search Workers—and he is upstairs from me so he knows something about my lectures—that from time to time I mention his ideas. . . this is not acceptable to him. And then I said, “Do you mean that if I am here, I should just lecture using your book?” And then he gave an affirmative answer. “Yes,” that’s what he expected. And I said, “Sorry, no, I cannot promise that.” And then he said, “Well, if so, then from now on I shall oppose you in all my capacities.” And then he enumerated them: member of the Royal Society and so forth. There were quite a few. Then he left. Banged the door (8, p. 126).

Fisher continued to do ground-breaking work in his new post and even became interested in a seroepidemiology of sorts when the blood group discoveries were announced. In 1943 he assumed the chair in Genetics at Cambridge University. During this period, he was estranged from wife and family and depressed by the death of his son George in World War II. Still, he continued to publish important statistical papers, consult, and do original genetic research. He began to receive many honors, was knighted by the Queen for his work, and finally retired, at age 67, in 1957 (perhaps not coincidentally, the same year he became embroiled in the lung cancer/smoking controversy).

FISHER’S ARGUMENTS CONCERNING LUNG CANCER

Fisher developed four lines of argument in questioning the causal relation of lung cancer to smoking. I will first list these and then briefly describe the evidence he produced in support of these arguments.

1) If **A** is associated with **B**, then not only is it possible that **A** causes **B**, but it is also possible that **B** is the cause of **A**. In other words, smoking may cause lung cancer, but it is a logical possibility that lung cancer causes smoking.

2) There may be a genetic predisposition to smoke (and that genetic predisposition is presumably also linked to lung cancer).

3) Smoking is unlikely to cause lung cancer because secular trend and other ecologic data do not support this relation.

4) Smoking does not cause lung cancer because inhalers are less likely to develop lung cancer than are noninhalers (9).

Fisher sees the argument that lung cancer causes smoking as an essentially unsupported speculation. His view is best described in his own words:

The subject is complicated, and I mentioned at an early stage that the logical distinction was between **A** and **B**, **B** causing **A**, or something else causing both. Is it possible, then, that lung cancer—that is to say, the pre-cancerous condition which must exist and is known to exist for years in those who are going to show overt lung cancer—is one of the causes of smoking cigarettes? I don’t think it can be excluded. I don’t think we know enough to say that it is such a cause. But the pre-cancerous condition is one involving a certain amount of slight chronic inflammation. The causes of smoking cigarettes may be studied among your friends, to some extent, and I think you will agree that a slight cause of irritation—a slight disappointment, an unexpected delay, some sort of a mild rebuff, a frustration—are [*sic*] commonly accompanied by pulling out a cigarette and getting a little compensation for life’s minor ills in that way. And so, anyone suffering from a chronic inflammation in part of the body (something that does not give rise to conscious pain) is not unlikely to be associated with smoking more frequently, or smoking rather than not smoking. It is the kind of comfort that might be a real solace to anyone in the fifteen years of approaching lung cancer. And to take the poor chap’s cigarettes away from him would be rather like taking away his white stick from a blind man. It would make an already unhappy person a little more unhappy than he need be (10, pp. 21–2).

Fisher never produced any data or organized any study to follow up on this implausible hypothesis. It is also noteworthy that Fisher was a smoker himself. Part of his resistance to seeing the association may have been rooted in his own fondness for smoking and in his dislike of criticism of any part of his life.

Fisher’s data concerning the genetics of smoking are sparse indeed. In two letters to *Nature*, he presents some tables based on information he received from a Professor

Verschuer of Germany and from Dr. Eliot Slater of London. There are no further details about these twin studies nor do we know how the smoking histories are obtained, categorized, or analyzed. He presents tables that the reader is expected to take at face value with almost no information about the study protocol or methods of investigation. In a 1979 review article, Cook has summarized the Fisher data in a single and brief table (table 1) (11).

Fisher concludes from these data that smoking is genetically determined, at least in part, but his discussion is surprisingly brief.

...There can therefore be little doubt that the genotype exercises a considerable influence on smoking, and on the particular habit of smoking adopted... Such genotypically different groups would be expected to differ in cancer incidence... (12, p. 108).

In a second letter to *Nature* he has more data, again sparsely described but purporting to give the smoking habits of female twins

raised either together or apart (tables 2 and 3). The 53 monozygotic twin pairs appear in both tables 2 and 3, but it is surprising and unexplained that over one-half (51 percent) of the pairs were raised separately. No definition of "raised separately" is given in his letter to *Nature* (13).

As Vandenbroucke mentions in his article, Floderus et al. (14) using the Swedish twin registry fail to support these data. Given the lack of detail Fisher provides, it is impossible to account for the discrepancies.

The secular trend and ecologic data will be dealt with later in the article where I discuss the difficulty Fisher had in synthesizing data and how he wandered out of his area of expertise.

His argument about inhalation is the only one he fully developed, but his use of the data was devious and selective.

In their first case-control study, Doll and Hill showed a slight difference in reported inhalation by cases and controls, with controls reporting slightly more inhalation: 62 percent among cases versus 67 percent

TABLE 1. Fisher's data on smoking habits of monozygotic and dizygotic twin pairs*

	Smoking habits		
	Alike	Somewhat alike	Different
Monozygotic	33 (65)†	6 (12)	12 (23)
Dizygotic	11 (35)	4 (13)	16 (52)

* Source: references 11-13.

† Numbers in parentheses, percentage.

TABLE 2. Fisher's data on smoking habits of monozygotic and dizygotic female twin pairs*

	Smoking habits		
	Alike	Not alike	Total
Monozygotic	44 (83)†	9 (17)	53 (100)
Dizygotic	9 (50)	9 (50)	18 (100)

* Source: references 11-13.

† Numbers in parentheses, percentage.

TABLE 3. Fisher's data on smoking habits of female monozygotic twins raised together or separately*

	Smoking habits		
	Alike	Not alike	Total
Raised separately	23 (85)†	4 (15)	27 (100)
Raised together	21 (81)	5 (19)	26 (100)

* Source: references 11-13.

† Numbers in parentheses, percentage.

among controls. This was surprising, but in their second case-control study this was not confirmed, and when they combined information about inhalation in both studies, the results were 67.5 percent inhaling among cases versus 65.9 percent among controls—essentially no difference.

Fisher seized upon this discrepancy and makes much of it. He uses a device that smacks of sophistry to magnify the difference and its importance by transforming the percentages into observed versus expected figures (using a chi-square analysis). He then suggests that, if the cases had inhaled, 45 lives could have been saved. He does this with attempts at humor and a mischievous tone that seemed to have pleased the audiences that attended his lectures on this subject. The data are presented in the tables below, in which I have summarized his data (tables 4 and 5).

Fisher interprets the data as follows:

...Should not these workers have let the world know, not only that they have discovered the cause of lung cancer (cigarettes), but also that they had discovered the means of its prevention (inhaling cigarette smoke)? How had the Medical Research Council the heart to withhold this

information from the thousands who would otherwise die of lung cancer?

Those who refuse the jump from the association to causation in the case of cigarette smoking will not be tempted to take it in the case of inhaling; but the Medical Research Council and its Statistical Research Unit think the argument is valid in the first case. Can they refuse to admit it in the second (10, p. 47)?

He used the data from only the first case-control study even though the data from the second study and the combined data were available at the time; he probably did this because the inhalation differences became less after the data were combined. Using this technique, he has metamorphosized a trivial difference between inhalers and noninhalers into 45 lives saved if only the noninhalers had inhaled!

Other studies were available at the time that showed no differences or showed more inhaling by cases, but he never mentions this. Subsequent studies, of course, have shown that the cancer risks are associated with inhalation.

Throughout his writings on the topic of smoking and cancer, he strives to be provocative rather than responsible. Fisher fails to deal with the entire body of evidence on the

TABLE 4. Doll and Hill data from first case-control study of smoking and lung cancer, smoking prevalence in cases versus controls (men only)*

	Maximum no. of daily cigarettes							
	1-14		15-24		25-49		>49	
	I†	N†	I	N	I	N	I	N
Cases	148	84	133	63	96	78	21	24
Controls	179	101	157	44	74	44	16	7

* Source: adapted from reference 11.

† I, inhaling history reported; N, noninhaling reported

TABLE 5. Fisher's estimate* of lives saved if the cases not inhaling had been inhalers†

Cigarettes/day	Expected cancers	Observed cancers	No. of avoidable cancers
1-14	149	149	0
15-24	153	133	20
25-49	109	96	13
>49	33	21	12
Total	444	399	45

* For details of the computation, see reference 11.

† Source: data adapted from reference 11.

subject and makes too much of small differences. While he claimed to be interested in the dispassionate search for truth, his arguments have a polemic tone.

THE FAILURE OF HIS GENIUS

Why would Fisher persist in pressing these arguments when the main body of data, even by 1957–1958, was so large and firm in the indictment of cigarettes and his own counterarguments so tenuous and precious? Why didn't he try to integrate or even weigh his arguments against the rest of the data? In none of his writings on this subject do we have any attempt to evaluate the importance of the inhalation exception and put it in some sort of proper relation next to the large body of available epidemiologic data. In sum, he had trouble distinguishing the possible from the probable and was unable to synthesize all the data to reach a reasonable conclusion.

I believe, from trying to reconstruct his involvement, by accounts of his contemporaries of his personality, from his own writings, from testimony of his daughter, and by some guessing (which he would disdain), that there are several explanations for the failure of his genius in this matter. Fisher's zest for confrontation and polemic was legend; all who knew him comment on this, even his usually uncritical biographer/daughter. Domestic fits of temper paralleled the intolerance and aggressiveness he demonstrated toward his colleagues. He feuded with the two Pearsons, Karl and Egon; with Gossett; with Neyman; and with others. He hated to admit that he was wrong on any subject. He preached the scientific method and was eloquent on the subject, but had great difficulty following his own strictures. I have already quoted Neyman on Fisher's attempt to force him to teach only Fisher's theories and to censor him. There are also other accounts, even by those who revered him, as did Mather and Mahalanobis (7). Fisher described his own behavior relatively well in 1947 when, reflecting on the way he felt he was treated by Karl Pearson, he said:

A scientific career is peculiar in some ways. Its *raison d'être* is the increase of natural knowledge. Occasionally, therefore, an increase in natural knowledge occurs. But this is tactless and feelings are hurt. For some small degree it is inevitable that views previously expounded are shown to be either obsolete or false. Most people, I think, can recognize this and take it in good part if what they have been teaching for ten years or so comes to need a little revision; but some undoubtedly take it hard, as a blow to their amour propre, or even as an invasion of the territory they had come to think of as exclusively their own, and they must react with the same ferocity as we can see in the robins and chaffinches these spring days when they resent an intrusion into their little territories. I do not think anything can be done about it. It is inherent in the nature of our profession; but a young scientist may be warned and advised that when he has a jewel to offer for the enrichment of mankind some certainly will wish to turn and rend him (2, p. 131).

This was written by the same man who tried to bully Neyman! When his first letter to the *British Medical Journal* was attacked and he was impugned for taking a fee from the tobacco industry, it probably fixed his views. His daughter mentions how offended he was by the rebuttal letters that pointed out he was a paid consultant.

Secondly, Fisher was a political conservative and an elitist (as were most eugenicists) and was disturbed by the British Medical Association's appeal to censure cigarette advertising and launch a public health campaign against smoking (Fisher was a smoker of pipes and cigarettes). He compares this proposed public education campaign to totalitarian propaganda and complains it is premature in a letter to the *British Medical Journal*:

Your annotation on "Dangers of Cigarette-smoking" [*sic*] leads up to the demand that these hazards "must be brought home to the public by all the modern devices of publicity." That is just what some of us with research interests are afraid of. In recent wars, for example, we have seen how unscrupulously the "modern devices of publicity" are liable to be used under the impulsion of fear; and surely the "yellow peril" of modern times is not the mild and

soothing weed but the organized creation of states of frantic alarm.

A common "device" is to point to a real cause for anxiety, such as the increased incidence of lung cancer, and to ascribe it in urgent tones to what is possibly an entirely imaginary cause. Another, also illustrated in your annotation, is to ignore the extent to which the claims in question have aroused rational skepticism. The phrase, "in the presence of the painstaking investigations of statisticians that seem to have closed every loophole of escape for tobacco as the villain in the piece," seems to be pure political rhetoric, even to the curious practice of escaping through loopholes. I believe I have seen the source of all the evidence cited. I do see a good deal of other statisticians. Many would still feel, as I did about five years ago, that a good *prima facie* case had been made for further investigation. None think that the matter is already settled. The further investigation seems, however, to have degenerated into the making of more confident exclamations with the studied avoidance of the discussion of those alternative explanations of the facts which still await exclusion (15, p. 1518).

Fisher was upset by the public health response to the dangers of smoking not only because he felt that the supporting data were weak, but also due to his holding certain ideologic objections to mass public health campaigns.

Third, it was recognized by those who worked most closely with him that he was good with data while working on one small set but was not easily able to integrate multiple or large data sets. Here is a quotation from Yates and Mather:

In his own work Fisher was at his best when confronted with small self-contained sets of data, and many of his solutions of such problems showed great elegance and originality. He was never much interested in the assembly and analysis of large amounts of data from varied sources bearing on a given issue. The analysis of a single experiment and the conclusions that could be drawn from it, for example, interested him greatly, the assembly and analysis of the results of a varied collection of experiments scarcely at all. This would not have mattered—it could well be left to others—had he not tended to brush aside these more laborious and pedestrian labours, while re-

membering and continuing to maintain his own first conclusions based on an examination of part of the data, conclusions which inevitably required reexamination in the light of subsequent work.

The smoking-lung cancer controversy is a case in point. To those who mistrust the alarms and excursions of the medical world Fisher's skepticism of the evidence that cigarette smoking had been established as the causative agent of lung cancer was refreshing. His deduction from Doll and Hill's published results that inhalers were less subject to cancer than non-inhalers, and his subsequent confirmation of this from a more detailed breakdown of the results supplied to him by Doll and Hill, was a striking demonstration of the pitfalls attendant on the interpretation of survey data; and his demonstration, from data on monozygotic and dizygotic twins, that smoking habits are strongly genetically conditioned was delightful. Yet although for some years passionately interested in the controversy, he never attempted any review of the later evidence (16, p. 106).

Perhaps another reason he persisted in the cigarette controversy may have had to do with the circumstances of his life when he became engaged with the issue. He had just retired and was at loose ends. He received a lot of public attention for his views on lung cancer and fashioned a talk on the subject which he gave all over the world and which he arranged to have reprinted. It was well received, even by medical audiences. His daughter describes the lectures he gave on this subject:

In Australia his lectures on the subject were frankly intended to raise questions. He spoke of "teasing" the British Medical Association and of being "deliberately provocative." He obviously enjoyed giving these talks, which so clearly illustrated points he wanted to make about inference: the consequences of not randomizing, the need to check on the assumptions implicit in any hypothesis, and the need to admit all of the data, particularly those which seemed anomalous. Audiences were quick to laugh when he pointed out the negative correlation between lung cancer and inhaling; and there were other anomalies; the positive correlation between smoking habit and genotype, the lack of success at that time in isolating any carcinogenic sub-

stance from tobacco, the difference in incidence of cancer between smokers of cigarettes and smokers of tobacco in other forms, between people in different environments, between the sexes. It became obvious that more and better data were needed before any hypothesis could be considered to have been convincingly demonstrated. If he seemed to some the devil's advocate, his reasonableness, good-humor and wit were hard to resist. His talks were generally well-received (2, p. 475).

Such a positive reception, as well as the opportunity to laud his personal achievements and opinions, must have made these talks gratifying to a man nearing the end of his career.

Fisher's belief in the importance of genetics and his feeling that it is often neglected in medical research also influenced his views; indeed, he says this in his discussion of the genetic predisposition to smoke. His advancing the hypothesis as to the genetic predisposition to smoke in an effort to supplant the cigarette hypothesis reflects his intense interest in genetics.

FISHER OVEREXTENDED

Perhaps the greatest contributor to his erroneous analysis is that the great man had wandered too far out of his field. He knew very little of the case-control method but was entirely suspicious of it because of the absence of his beloved randomization. Nor was he well informed about the ecologic data concerning smoking and cancer and, when he comments on it, he gets it quite wrong.

The change over recent decades gives not the least evidence of being due to increasing consumption of tobacco. We can't tell much about the absolute magnitude of this secular change. It is certain that radiology has facilitated the detection of lung cancer enormously, that radiological apparatus and radiologists are much more abundantly available for our populations than they formerly were.

... Again, the attention of the medical profession has been forcibly drawn to lung cancer, and it invariably happens that when the attention of the medical profession is drawn to any disease, that disease begins to take up more space in the official reports—

it is more often seen and more often diagnosed with confidence; death certificates more often include the particular disease. Consequently it is not easy to say how much of the increase is real. I think part of it must be real, because there's no doubt that the populations concerned have been enduring or enjoying a very considerable increase in urbanization.

... You might say that the whole population during the last twenty, thirty, forty years has been becoming steadily urbanized, and as the urban rate for lung cancer is considerably greater than the rural rate, in my country as in yours, we must recognize here the possibility of our real cause of the increase in lung cancer. There may be others.

But the only good comparison we can make in respect of the time-change is that between men and women. The same apparatus, the same radiologists, the same physicians diagnose both men and women. Whatever effects improved apparatus may have, whatever effects an increased attention to the disease may have, will be the same in the two sexes. Whatever effects urbanization may have you would think might be the same in the two sexes. Consequently, we can, at least, inquire whether the rate of increase of lung cancer in men is the same, or greater, or less, than the rate of increase of lung cancer in women. For it is certainly true, I think in both our countries, that whereas the smoking habits of men have not changed very dramatically over the last fifty years, yet the smoking habits of women have changed a very great deal. And on making that comparison, it appears that lung cancer is increasing considerably more rapidly—absolutely and relatively—in men than it is in women, whereas the habit of smoking has certainly increased much more extensively in women than in men. There is, in fact, no reasonable ground at all to associate the secular increase in lung cancer with the increase in smoking as has been done with dramatic eloquence, I suppose as part of the campaign of bringing home the terrible danger, just as though it was impossible that statistical methods of inquiry should supply a means of checking that very rash assumption (10, p. 24-5).

He apparently made no attempt to put all the evidence together, to read all the older papers, to wonder if the disease was uncommon among populations that didn't smoke. He wasn't an epidemiologist and seemed not

