UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF COLUMBIA

UNITED STATES OF AMERICA,	:	CA No. 99-2496(GSK)
	:	September 29, 2004
Plaintiff,	:	
	:	9:30 a.m.
	:	
v.	:	Washington, D.C.
	:	
PHILIP MORRIS USA, et al.,	:	
	:	
Defendants.	:	

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MORNING SESSION
VOLUME 6
TRANSCRIPT OF TRIAL RECORD
BEFORE THE HONORABLE GLADYS KESSLER
UNITED STATES DISTRICT JUDGE

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P R O C E E D I N G S

THE COURT: Good morning. This is United States of America versus Philip Morris. CA-99-2496.

And I assume that at this point the government ready with Dr. Samet.

MS. EUBANKS: That's correct, Your Honor. We are ready to call Dr. Samet to the stand.

THE COURT: Would you do that, please?

THE DEPUTY CLERK: Please remain standing and raise your right hand.

JONATHAN M. SAMET, Government's witness, SWORN

DIRECT EXAMINATION

BY MS. EUBANKS:

Q. Good morning, Dr. Samet. One of the things that we're attempting to get for you is a laser pointer which I think will facilitate the testimony today. So if someone has it, could you just pass it up.

MS. EUBANKS: But we can certainly get started right away, Your Honor. The first thing I'd like to do, with Your Honor's permission, is approach the witness and provide him with his written direct testimony.

Q. I've just handed you your written direct testimony that was filed with the court earlier this month on the 20th of September in two separate volumes.

Are you familiar with this document, Dr. Samet?

A. Yes, I am.
Q. Do you need to make any changes or corrections in the testimony that I've given you that was filed earlier in this case?
A. Well, I would note that, as I continued to reread this lengthy testimony, I found some typographical errors. And I did note that in one point I referred to the EPA designation as a Class A carcinogen when I meant Group A.
THE COURT: Do you have -- excuse a minute. I don't know that we need to take any time on typographical errors, but if there's anything substantive, such as what you just mentioned, that you think the record really should reflect, if you happen to have the page citation, it might be good to get that in the record.
THE WITNESS: I'll provide that.
THE COURT: Good. Thank you.
MS. EUBANKS: And with respect to the testimony that's referenced, Your Honor, I'll just note for the court that the testimony references an exhibit that is U.S. 17,206. That should really read at 17,304. So as the testimony references 17,206, if we substitute 17,304, and defendants have been advised of that change as well.
I'd like to hand Dr. Samet the laser pointer
THE COURT: Good. We needed that earlier.
BY MS. EUBANKS:

Q. Do you know how to operate that one? I think you do.

All right. I'd like to spend some time with an exhibit that we prepared that is a demonstrative that will take Dr. Samet through his conclusions, and if we could get that up and the screen.

What I'd like to do, Dr. Samet, is you have it before you here, but also you can see it on the screen. Are you able the see the screen in front of us?

A. Yes, I am.

MS. EUBANKS: All right. Before I do that, Your Honor, I suppose what I should do is make a proffer. We proffer Dr. Samet as an expert in the area of epidemiology and science of smoking and health, pulmonology and internal medicine.

THE COURT: Any objections?

MR. McDERMOTT: No objection.

MS. EUBANKS: Thank you.

THE COURT: He may be accepted as an expert in those areas.

BY MS. EUBANKS:

Q. Dr. Samet, directing your attention to the screen with the first three slides, can you describe for us the significance of the information on those slides to your opinions expressed in U.S. v Philip Morris?

THE COURT: Is that slide just a little fuzzy, everybody, or is it still early in the morning for me?

MS. EUBANKS: It's a little fuzzy, Your Honor, but it's still early in the morning, too. I don't think we can make it any more clear, but hopefully through the testimony of Dr. Samet it will all come into focus.

A. If I could comment. These first three slides address some of the early history of tobacco which was a new world plant used by the Indians for ceremonial purposes brought back by Columbus to Europe at his first voyage and its use soon spread throughout Europe.

I think what's important to note that its early use was largely in the form of snuff and in the form of oral tobacco, chewed tobacco, with some pipe and cigar, and some minor use of tobacco in the form of hand-rolled cigarettes at the time.

Q. All right. I'd like to invite your attention, Dr. Samet, to the next two slides and I would like to take those together.

Mr. Jackson, if you could pull over to the 1880s and 1913.

The 1880 slide indicates the rise of modern cigarette history and then next to that for 1913 we have a pack of Camels. What do these slides demonstrate in relation to your opinions given here?

A. Well, the stage for the epidemic of tobacco-caused disease that began the last century was really set at the end of the 19th Century when the development of the automatic cigarette manufacturing machine, the Bonsack machine, made possible the

manufacture of cigarettes in large numbers and cheaply; and, of course, at the same time there was a rise of a large industry in the United States and elsewhere to manufacture, distribute, and sell the cigarette.

And one difference between the cigarette and the other forms of tobacco use was that the smoke was inhaled deeply into the lung and, as will be discussing, this of course led to the cause of -- causation of lung cancer and other diseases.

Q. Dr. Samet, in terms of leading to the cause of lung cancer and other diseases, would you describe that as a epidemic?

A. Well, the epidemic began, lung cancer and other diseases, across the last century and I'll be addressing its rise over that time.

Q. The next thing I'd like to do is call up an animation and ask you to explain how it is that toxins get into the body from smoking a cigarette.

A. I'm just going to go through a little bit of basics of how the lungs and heart work. I thin this is a foundation for why the cigarette is such a potent cause of disease in the lungs and other organs.

This is simply the anatomy, the respiratory tract and, of course, the mouth, the trachea bringing air into the lungs, the lungs, and the heart shown here.

The lungs have tubes, the bronchi, that bring the smoke or air out to the gas exchanging surface or alveoli. These

tubes branch many times. And, in fact, the lung has a very large surface area, that I'll be describing in a minute, across which gasses exchange.

So here we are now down to the level of the alveoli which is where gas exchange actually takes place. The area of these alveoli, if they were laid out, is roughly the equivalent to that of a tennis court. So it's a remarkable surface for gas exchange.

THE COURT: Dr. Samet, could you go little more slowly?

THE WITNESS: Yes.

THE COURT: Thank you.

A. Of course, as we breathe in, oxygen exchanges across the alveoli moving into the capillaries and carbon dioxide, a product of metabolism, moves out of lung as we exhale.

So we are just seeing this repeated now with oxygen coming in and going out in the capillaries to be circulated through the body and carbon dioxide leaving. So this is the structure of the alveoli.

And now we are going to, in a moment, see this -- here is, of course, the heat beating with the lungs. And now we are going to move on and look at what happens with smoking.

And, of course, as the smoker inhales the smoke follows the same path that the air followed, moving through the bronchi and out to the alveoli, that surface that's there for exchanging gasses.

So in with the smoke will come, of course, the various components of tobacco smoke, which will reach to the alveoli, so here we're back to the level of one of those alveoli.

And now we have, of course, oxygen coming in, but perhaps various toxins and carcinogens, gasses like carbon monoxide. We would have nicotine, which is in the smoke, actually bound to the particles.

And just like the oxygen moving across and going into the blood, some of these components move across and go into the blood and, in fact, some of the tar's retained in the lung.

So we can see then, just shown schematically, that nicotine, various toxins like carbon monoxide and carcinogens like benzene, would move on and enter the bloodstream and then be circulated throughout the body.

Q. Thank you, Dr. Samet.

I'd like to return to the time line that we have up and now I'd like to focus your attention on the slide indicating World War 1. What I'd like to do is have you discuss the significance of the World War 1 slide alongside of the next slide, which is 1935, and can you tell us the significance of the smoking rates that are indicated there?

A. I would just like to talk for a minute about the patterns of smoking across the last century.

Men began to smoke in larger numbers, higher percentages, before women. So, for example, by 1935 a majority

of adult men in the United States were smoking but only 18 percent of women. For men, the time around World War 1 was a time when smoking was -- uptake was more rapid among men.

I think one thing that is important about these patterns is that they're followed by the epidemics of lung cancer and other diseases caused by smoking which were first apparent in men, and with about a 20-year delay reflecting the differences in smoking patterns between men and women, we will see the rise of smoking-caused diseases in women.

Q. I'd like to ask you a question about -- well first, can you tell us what is the first published study to show a relationship between smoking and premature death?

A. Well, to my knowledge, the first published study was that carried out by Raymond Pearl, who is actually the professor of biostatistics at my school, the Johns Hopkins School of Public Health.

Dr. Pearl was following families in east Baltimore. And in a study published in the Journal of Science in 1938, Dr. Pearl showed that what he called heavy smokers -- which is the line shown over here -- had on average about eight years less median life expectancy than nonsmokers.

This paper was important for showing that smoking had such a powerful effect that it could reduce the overall survival rate of people who smoked by eight years compared to those who did not smoke.

Q. Is the Pearl study what you would call a epidemiological study?
A. Yes. This was an epidemiological study, a study that we would today call a cohort study.
Q. Dr. Samet, if you could, the court reporter has to make a verbatim transcript and sometimes I know you and I speak quickly, so I will slow down and perhaps that will help you slow down as well. Okay?
A. We will both try and slow down.
Q. Now, with respect to the next, is there -- who is that?
A. This is Dr. Pearl. I think we can move on.
Q. Now, with respect to 1939, there's been other testimony in the case about Oxner and DeBakey.

Can you tell us the significance of their study in 1939?
A. Well, across the 20s and 30s, as often happens with new epidemics, the physicians caring for patients note that they were seeing, for example, more cases of lung cancer and began to describe their experience in the medical literature.

This is a report by Oxner and DeBakey 1939 describing pneumonectomy, taking out a lung for treatment cancer of the lung. They describe seven cases.

This is the same Dr. Oxner who went on and found the Oxner clinic in Louisiana, and Michael DeBakey, the well known cardiovascular surgeon.

This report is important for the hypothesis offered by Oxner-DeBakey that perhaps smoking was contributing to the rise of lung cancer by causing irritation of the lung.

THE COURT: I'm sorry. Did you say contributing to the demise of lung cancer?

THE WITNESS: The rise.

THE COURT: The rise?

THE WITNESS: The rise.

BY MS. EUBANKS:

Q. Speak slowly, Dr. Samet.

Now, moving on to the next slide, please. We had looked at World War I and smoking rates. What can you tell us of the significance of the chart on the screen and with particular emphasis on the other highlighted portion regarding the increase in smoking?

A. Yes. This slide shows the number of cigarettes smoked per capita by adults in the United States, including smokers and nonsmokers, so all persons 18 years of age and older.

And you can note that the peak here was about 4,000 cigarettes or roughly 200 packets per year per adult.

The yellow simply highlights the time of World War II and you can see the very sharp increase in the number of cigarettes smoked at that point historically. And we know that this is a time when women began to smoke in somewhat larger numbers than before.

Q. Now, what was the first community-based cohort study on chronic disease?

A. Well, we turn now to the Framingham study which was started in the late 1940s by our Public Health Service. The study was started at a time when coronary heart disease and stroke rates were rising. And the Framingham study was started to try and understand what were the causes of this increase.

The Public Health Service went to Framingham, which is a small town near Boston, and enrolled people from the town: obtained medical information about them, obtained information about their smoking and cholesterol, and then followed them over time to look at the rates of heart attack, sudden death, stroke, and other cardiovascular diseases in this population.

And it was this study, along with others, that provided some of the early evidence that smoking was strongly associated with risk for heart attack and stroke.

Q. Does this study continue today?

A. Of course, most of the original participants are now deceased, but in fact their children are under follow-up in further studies of cardiovascular and other diseases.

Q. Can we move to the next slide?

Now, the 1950s was -- was the 1950s an important decade?

A. I think the 1950s in terms of studies on smoking health were a very important decade with many studies carried out.

Q. Why don't you tell us about some of those as we see depicted on the screen?

A. Let me speak first to 1950 and studies of smoking and lung cancer.

I think in 1950 there were five publications of what are called case control studies, studies where the smoking of people with lung cancer was compared to that of people without lung cancer controls.

Three of the best known studies are listed here. That by Doll and Hill in the United Kingdom, a study carried out in London which showed a very strong association between smoking and lung cancer risk.

In the United States, two studies were published in the Journal of the American Medical Association, one by Wynder and Graham. Wynder carried out his study as a medical student and Graham was a thoracic surgeon, a well-known thoracic surgeon, who he worked with. In fact, Graham did the first pneumonectomy for the treatment of lung cancer. This study also showed an association of smoking with lung cancer.

And finally, Levine and colleagues -- Levine was another person who became a member of my department at Johns Hopkins -- published a similar study to that of Wynder and Graham in the same issue of the Journal of the American Medical Association.

Q. Let's look at the next slide, please, on the 50s.

And can you describe the cohort studies that are referenced here?

A. Yes. I was just talking about case control studies, and now I'm going to move on to cohort studies, studies where people were followed over time, smokers and nonsmokers.

And one of the most important and notable of these was the British doctors study that was again started by Doll and Hill who had also done the case control study.

I think importantly they said these retrospective case control studies had provided strong association, now let's move and get prospectively collected evidence.

And in this study they followed 34,000 men and about 6,000 female physicians, and looked at their mortality from lung cancer and other diseases over what is now a 50-year interval.

The veterans' cohort study is another representative study from the United States, this one involving about 250,000 U.S. veterans, and again followed for many years, providing us important information about the risks of smoking and what happens when people stop.

Q. You said it involved 250,000 veterans. In terms of size, what kind of study was this considered at the time?

A. Of course, this was a very large study at the time, and in fact, by the end of that decade the American Cancer Society had started its first cohort study of 1 million Americans.

Q. All right. Let's move to the next slide.

There is a reference in 1955 to smoking rates. Can you tell us the significance of that information?

A. Well, looking at these data we can see that the rate in men remained quite similar to what I had shown earlier, and there's a further increase in the rate of smoking in women above the approximately 18 percent figure I showed earlier to now about 25 percent. And again, part of the importance of these figures is in interpreting the epidemics of disease that were to follow.

Q. All right. Let's see the next slide, please.

Now, in 1957 there's a statement from the Surgeon General. Can you tell us the significance of his statement regarding excessive cigarette smoking to your opinions expressed here?

MR. BERNICK: Your Honor, I have an objection. This is the only time I will rise because this is Mr. McDermott's witness, but this is an issue we didn't foresee. Neither his expert report or the reliance --

THE COURT: Would you come forward so we can hear you?

MR. BERNICK: I'm sorry. Neither the expert report nor the reliance materials for the expert report, nor as I've looked through the direct examination, did I see that this witness was going to be a state-of-the-art witness with respect to the studies that were done in the 1950s.

There's no expression of any view about when consensus arose and there's no reference to this document.

THE COURT: I don't hear him talking about consensus at this point.

MR. BERNICK: I understand that, but that's what I wanted to make sure didn't happen.

But this document in particular is not on his reliance list. I don't even think it's referenced in the direct examination.

And that also applies to the 1959 statement by Mr. Burney, and there's a reference to some G and C document. I believe that that's correct, and that's the best that I can determine here. But I don't believe that these materials -- I know this weren't reference in this expert report in this case.

MS. EUBANKS: Your Honor, first I can say that a copy of this demonstrative has been provided to defendants with all of this information.

We've been instructed not to reiterate everything in the expert report, and this is entirely proper.

He's expressing the significance of certain findings, which I think if we just get through the examination, it will become clear.

As you noted, there is no reference to consensus. We have the two volumes of his expert report and those that are the opinions that he's expressing here. It's not our intent to go outside of that information.

Insofar as the objections that are raised to the

materials, we intend to proffer, and there are a host of objections that defendants have raised, and we've already briefed that. I would urge the court to let us continue with the testimony, and besides which Order 471 does not really permit this teaming up.

As Mr. Bernick says, this is the only time he's going to rise. Your order is clear on this. He's not even examining the witness. It's not appropriate for him to raise an objection.

THE COURT: Mr. McDermott, do you want to second Mr. Bernick's objections, I gather?

MR. McDERMOTT: I not only second it. I will say Mr. Bernick will examine if the court does permit Dr. Samet to go into areas that we think are beyond the scope of his direct and beyond the scope of his expert report because we are not prepared on the basis of what we received from the government to deal with this, at least I am not.

But I would also say, Your Honor, while we are interrupting things, that what the government is doing here doesn't comport with our understanding of the purpose of this one-hour direct which is to explain complicated charts, graphs and exhibits.

This is just a slide show, a talking history. There is nothing complicated about what is going on here. And it seems to us this is a violation of the spirit of what the court

intended.

MS. EUBANKS: Your Honor, if there's nothing complicated about it, with your permission I'd like to proceed with our examination.

Certainly there was nothing in the order that suggested it needed to be complicated. Rather what the order expressly said is that the parties, if they had demonstratives that they wished to use with their witnesses, that they could have one hour to do that. We gave copies of the demonstratives --

THE COURT: The objection is overruled. The government may proceed.

The direct examination certainly references some charts that I am sure the government, or I expect that the government will get to in its demonstrative this morning.

So why don't you proceed, Ms. Eubanks.

BY MS. EUBANKS:

Q. Dr. Samet, I had asked you a question about Surgeon General Burney's statement regarding excessive cigarette smoke. Can you answer that now or do you need it read back?

A. No, I recall the question.

This is a statement prepared by Surgeon General Burney and published in 1957. This is a time when the rising evidence had attracted the attention of the public health authorities, and Surgeon General Burney authored a comment in part based on a review carried out by a group that had looked at this evidence.

One of his statements is here with regard to cigarette smoking and lung cancer, just noting that there is an increasingly consistent body of evidence that excessive cigarette smoking is one of the causative -- I would say he meant causal factors in lung cancer.

Q. All right. Could we see the next slide, please?

What is the significance of the prototype evidence reviewed that occurred in 1959 as depicted here?

A. Well, this is a report --

MR. BERNICK: Excuse me. Your Honor, for the record we have the same objection to this slide and the next slide, just so the record is clear.

THE COURT: The objection is overruled.

Go ahead, please.

A. This is a paper published in the Journal of the National Cancer Institute. I think it's important because it represented a systematic review, a careful look at the evidence on smoking and lung cancer carried out by a notable group of public health researchers.

They carefully looked at the evidence on smoking and lung cancer and considered whether alternatives to causation by smoking were likely to explain the findings of the epidemiological studies.

Their conclusion was that the evidence was consistent with causation of lung cancer by smoking.

Q. Can we see the next slide, please?

This indicates Surgeon General LeRoy Burney's statement regarding smoking and lung cancer. Can you briefly explain the significance of that to your opinions?

A. This was a further statement by Surgeon General Burney, again, based on an evidence review. His conclusions -- several of the conclusions are highlighted here from this report.

The weight of the evidence at present implicates smoking as the principal etiological -- I'll translate that. Etiology means cause -- factor in increased incidence of lung cancer.

There's a second conclusion related to treating tobacco or filtering the smoke that I've shown here.

Q. All right. And that's shown on the screen currently?

A. Correct.

And then this last conclusion relates to the fact simply that higher risks were seen in smokers compared to nonsmokers in many different environments, suggesting that smoking was the explanatory factor for the increase.

Q. All right. Could we see the next slide, please?

Now, in 1962, you have shown here the Royal College of Physicians' report. What is the significance of that report to your opinions expressed today?

A. Well, this report was prepared by the Royal College of Physicians in the United Kingdom.

It was important because it was a health organization stepping back and looking systematically at the evidence on smoking and disease, reaching a conclusion shown here two years before the '64 Surgeon General's report, cigarette smoking is the cause of lung cancer and bronchitis.

That would refer to what we now call chronic obstructive pulmonary disease, or COPD, and probably contributes to the development of coronary heart disease. And, of course, today many medical organizations have issued similar statements.

Q. I'm going to ask you again to speak a little slower. And let's turn to the next slide, please.

Now, there's been a lot of testimony in the case, including in your written direct, on the 1964 Surgeon General's report.

Can you summarize the significance of the conclusions or the statements that we see depicted here to your opinions expressed?

A. Well, the 1964 report often referred to as a landmark represented a systematic review of all of the evidence.

The Surgeon General was asked to convene a multidisciplinary team of experts, people who were known not to have expressed prior views on the smoking health issue.

They went through all of the evidence. They developed criteria for evaluating the evidence. In fact, an approach we still use with the Surgeon General's reports and elsewhere in

public health. They developed criteria for evaluating causality, often referred to as the Surgeon General's criteria, and some of their conclusions are shown here.

Cigarette smoking is causally related to lung cancer in men. The data for women were less extensive because the rise of lung cancer was slower in women and fewer women had been included in studies.

They also offered a conclusion -- causal conclusions on laryngeal cancer and chronic bronchitis. And there was a comment on coronary heart disease noting that there was an association, but they were not so strong in their language with regard to causation.

Q. Could we see the next slide?

What can you tell us about this gathering and why you were interested in depicting that?

A. Well, this is simply the release of the report with Surgeon General Terry and behind him his panel.

I would just say that the Surgeon General continues to release reports to the public with press conferences to disseminate the findings of these reports.

Q. May we see the next slide, please?

Now, this is the 1972 Surgeon General's report. In terms of the conclusions regarding passive smoking, what can you tell us about that report?

A. This is a report from Surgeon General Jessie Steinfeld.

This was the first report in which the exposure of nonsmokers to tobacco smoke was addressed as a topic.

In one of the sections of the report, the information on levels of tobacco smoke components in indoor environments was reviewed, and Surgeon General Steinfeld made some comments about this.

Q. And those comments, are those depicted on the screen now?

A. This is one of the comments from the report.

He simply noted that tar and nicotine levels in sidestream smoke, which is the principal source of what we would now call second-hand smoke, or environmental tobacco smoke, may be higher than those of mainstream smoke, and he said may be harmful to the nonsmoker.

Q. All right. I'd like to go to the next slide, please.

Now, this is the Surgeon General's report in 1979 and it notes on the screen that it expresses an opinion that there is a causal link between smoking and coronary heart disease.

What is the significance of that finding in 1979 to your opinions?

A. Well, the 1979 report was a very comprehensive review of the evidence and in it the Surgeon General stated that smoking causes coronary heart disease, although the conclusion was prefaced by some languages in relationship to purposes of preventive medicine.

Q. Now, I'd like to move to 1981. If we could have the slide

on the 207, please.

What is the Hirayama study?

A. Well, let me comment that in 1981 two epidemiological studies were published on second-hand smoke exposure and lung cancer risks in nonsmockers.

There had been studies earlier on the risks of second-hand smoke particularly related to adverse respiratory effects in children. These two studies: one from Dr. Hirayama in Japan and another from Dr. Trichopoulos and colleagues --

Q. Could you spell Trichopoulos, please? Slowly.

A. T-r-i-c-h-o-p-o-u-l-o-s.

Q. Okay.

A. In Greece, obviously.

Both showed an association between passive smoking and lung cancer risk.

Q. Now, what can you tell us about the chart that we see from the Hirayama study on the screen now?

A. Well, Dr. Hirayama's study was a cohort study, so he was following women over time who were nonsmokers. He measured the rate of lung cancer in women who were nonsmokers themselves and married to a nonsmoker. So that's the number 8.7, the rate per hundred thousand per year.

He also looked at the rated in women who were nonsmokers but who were married to a husband who smoked, and that rate was 15.5, approximately double the 8.7 rate if the

husbands were nonsmokers.

And then finally he provided information on the risks in those women who themselves smoked, and you can see that that rate, 32.8, was the highest, about four times that of the women who were not passively exposed to tobacco smoke.

Q. How did Hirayamas findings compare with those of Trichopoulos?

A. Generally they were similar.

Q. Let's move to the next slide, please.

Now, in 1981 the Surgeon General issued a report called The Changing Cigarette. What did that refer to.

A. Well, by 1981 there had been many changes in cigarettes since the 1950s. Of course, there had been a change from smoking of non-filtered to filtered cigarettes, and there had also been the introduction of ventilation of the cigarettes, and this report reviewed that evidence from a number of perspectives.

Q. All right. The next slide, please.

Now, in 1986 the Surgeon General issued a report focusing on passive smoking. What can you tell us about the Surgeon General's report that's identified here on the screen now?

A. Well, let me say first, in 1986 there were three reports issued that had conclusions on passive smoking and lung cancer.

This report from the Surgeon General, a report from the

International Agency For Research on Cancer of the World Health Organization and also a report from the U.S. National Research Council or the National Academy of Sciences, and all three reached the conclusion that passive smoking was a cause of lung cancer risk.

The Surgeon General's report also provided conclusions on children in addition to its conclusion on passive smoking and lung cancer.

Q. Was there a prior report to 1986 that the Surgeon General had dedicated to second-hand smoke or passive smoke?

A. There had been chapters in selective reports, but there had not been a report that was devoted to this topic.

Q. All right. Let's move on to 1988 and the Surgeon General's report that -- generally speaking, just very briefly, can you tell us about the conclusions of that reported in 1988?

A. Well, this is another report from Surgeon General Koop, and this one again focused on a single topic, nicotine addiction, reaching the conclusion that smoking was addicting and that nicotine was responsible.

Q. All right. Can we move to the next slide, please?

Now, in 1989 there was another Surgeon General's report that I'd like you to tell us basically what the conclusion was and its significance.

A. Well, I think this report, of course, was a 25th anniversary report of the 1964 report. It's labeled 25 Years of Progress

and addresses some of the gains that had been made in tobacco control.

It also reviewed the evidence on health effects, and did offer the conclusion that smoking remained the single most preventable cause of disease in the United States.

Q. Is that the conclusion that's depicted on the screen now?

A. Yes, it is.

Q. All right. Why don't we move now to the next slide, which I believe is 1992.

Can you tell us the significance of the EPA's findings here regarding second-hand smoke to your opinions?

A. Well, the EPA and its 1992 risk assessment covered systematically the evidence on second-hand smoke and lung cancer and also looked at the evidence on exposure of children to second-hand smoke and certain respiratory diseases.

It did reach the conclusion that second-hand smoke -- or, actually, the report called it ETS -- is a human group A carcinogen.

Q. And ETS stand for what?

A. Environmental tobacco smoke.

Q. I noticed that in your report you reference passive smoking instead of ETS for the largest part. Why is that?

A. Well, there are a number of different designations that have been used for this. I prefer using second-hand smoke to ETS for the mixture of tobacco smoke components in the air and, in

general, use the term involuntary smoking or passive smoking for the exposure of people to this mixture.

Q. All right. I'd like to take the next three slides as a group and I'd like you to discuss the significance of these three slides. First, let me ask this question.

Do they all deal with passive smoking or second-hand smoke?

A. Those are among the topics covered. The UK report had some other issues covered as well.

Q. Let's take the first one and, if you would, just take us through 1997, '98, and '99 and the significance of those reports, please.

A. Well, I think what is important about these reports is that they carefully reviewed the now very extensive evidence on passive smoking and child health, and their conclusions were quite similar, stating that passive smoking, particularly smoking by parents, caused more lower respiratory illnesses in children, more chronic symptoms like cough or wheeze in children exposed to tobacco smoke; that if the parents smoked, the lung growth, the development of the lung, was actually slowed compared to children whose parents did not smoke.

If a child had asthma, the asthma was made worse by the exposure to second-hand smoke, and also children whose parents smoked have more problems with their ears, with the middle ear, with chronic ear problems.

There were also conclusions in two of the reports, the California report and this report from the United Kingdom on passive smoking and coronary heart disease.

Again, there had been new evidence, epidemiological and experimental, on the risks of coronary heart disease in nonsmokers exposed to tobacco smoke, and both of these reports reached the conclusion that passive smoking should be considered a cause of coronary heart disease.

Q. Just so that your testimony is clear. When you say "both of these reports," can you identify what you mean by these?

A. I'm referring to the California EPA report and the UK report of the Scientific Committee on Tobacco and Health.

Q. Thank you. Let's move to 2001 and the Institute of Medicine, sometimes referred to in your testimony as IOM, and the publication Clearing the Smoke.

Can you tell us the significance of this, please?

A. Well, the Institute of Medicine is, of course, the medical arm of the National Academy of Scientists.

This report addressed the topic of harm reduction; that is, can the risks of smoking -- tobacco use be reduced, and it covered the evidence on lower machine-measured yield of cigarettes.

Q. Is that what we see depicted on the screen?

A. This is one of the conclusions from the Institute of Medicine reports.

Most current assessments of morbidity and mortality suggests that low yield products are associated with far less health benefit, if any, than would be predicted based on estimates of toxic exposure using Federal Trade Commission machine-measured yields.

Q. You will particularly need to slow yourself down when your reading from something. I know it's a natural tendency to go a little faster. It will be very helpful for our reporter if you could try to speak a slower.

All right. Can we look at the next slide, please? Again it's for 2001, and there's been a previous references to Monograph 13. Can you tell us the significance of Monograph 13?

A. Monograph 13 is obviously the 13th monograph in a series of monographs or books on tobacco control, smoking tobacco control, published by the National Cancer Institute.

And this particular volume had this cumbersome title Risks Associated with Smoking Cigarettes with Low Machine Measured Yields of Tar and Nicotine. So that was the topic of this report.

Q. All right. What is it that we see depicted on the screen for Monograph 13?

A. Well, this is one of the conclusions from Monograph 13. Widespread adoption of lower yield cigarettes by smokers in the United States has not prevented the sustained increase in lung cancer among older smokers.

Q. All right. Next slide. Now, that takes us up to the IARC report of 2002. Could you tell the court what IARC is?
A. IARC, or I-A-R-C, is the International Agency for Research on Cancer. It's the World Health Organization's arm that deals with cancer and it puts out a series of reports, IARC monographs that evaluate carcinogenicity.

This report, Volume 83, covered the topic of both active smoking and involuntary smoking, and I would note that I was the chair of the group that met to develop this volume.
Q. All right. If we could see the next slide with a statement that's found at page 1413 of the report.

Can you tell us the significance of that?
A. Well, this is a one of the conclusions in the report describing the classification by the IARC of second-hand smoke. Involuntary smoking is carcinogenic to humans, group 1, which means there's --

MR. McDERMOTT: Objection, Your Honor.

I may be mistaken, but I believe this was a 2004 report, and it's covered by Your Honor's ruling last night as are the next two reports coming up.

These were not given to us in his expert materials. The government didn't supplement. We did not have a chance to examine. It's covered by Your Honor's ruling last evening, I believe.

MS. EUBANKS: I don't believe that's correct.

For one thing, apparently Mr. McDermott has forgotten that the very statement that we filed alongside of the expert report when we filed those over two years ago included a statement as to this witness and other experts who were continuing research in their field, continuing to keep abreast of materials.

Now, his deposition was taken and he revealed information about what he was doing at the time. So, in terms of this particular report -- and this is all in the briefing that was before the court, particularly with respect to Dr. Samet, whose situation is different from the situation with Dr. Brandt and the reliance materials that were set forth here.

But if I'm missing anything, I'm sure that Mr. Kinner can inform the court more precisely about any other matters with respect to this IARC report and this witness's testimony, because certainly as he's testified, he was involved in the preparation. And our report that our memo that forwarded the report with Dr. Samet, as I said, expressly advised defendants of the fact that these were experts in their field and they would continue to keep up and keep abreast of matters.

And so it's been no secret. This is a document that's been cited in the government's proposed findings and so forth. Dr. Samet's name appears all over it. If you look at the requirements of rule 26, they talk about making sure, and no particular mechanism for doing so, that the other side is

apprised of certain developments. So they certainly had this opportunity, besides which Dr. Samet has testified and I think been deposed by defendants over 10 times. This can be no unfair surprise.

THE COURT: What was the last deposition, if you remember?

MS. EUBANKS: It was in this case and it was in May of 2002.

MR. McDERMOTT: It was more than two years ago, Your Honor. These materials weren't available. They've been supplied to us and we've never had an opportunity to ask Dr. Samet about him or his opinions or his involvement in them.

MR. KINNER: Good morning, Your Honor. My name is Russell Kinner from the Department of Justice representing the United States.

Your Honor will recall that on a Friday we had a discussion in the middle of a deposition about items and whether they were appropriate for supplemental deposition of Dr. Wyant. And in that proceeding the discussion was concerning things that defendants were on notice of and whether or not additional deposition was needed for certain materials.

These materials are things that -- yes, they didn't depose Dr. Samet subsequent to May 2002, but with regard to the Surgeon General's report of 2004, we notified the defendants back in November 2001 of his participation in the Surgeon

General's report of 2004.

And in the summer of 2002, Dr. Samet is also the adviser to Doctors Wyant and Zeger on the epidemiology of the health care cost model. And as soon as the IARC findings were posted on the Web site we provided them with Dr. Wyant -- provided them with Dr. Wyant and Dr. Zeger's report in July of 2002 about four weeks after they were posted on the Web site.

Let's see. So there is notice and they are -- they are cited in the proposed Findings of Fact. And indeed that notice comes before the close of discovery in September 2002.

So in the situation involving Dr. Wyant and Dr. Gruber, there has been numerous -- numerous depositions subsequent to the pursuit of, "Oh, we learned this, now we want this."

And here they were also put on notice, and there's simply been no pursuit of whether or not they wanted additional information with regard to Dr. Samet.

I would also point out that -- I'm sorry. There was another point I was going to make.

I'm sure it will come to me.

MR. McDERMOTT: May I respond briefly, Your Honor?

We are not mind readers and we are not required to be. The solution was to supplement if the government wanted to supplement Dr. Samet's reliance materials and opinions. We didn't get that. They supplemented an awful lot of other experts as this court is well aware.

Of course, we were aware of the report coming out. What we were not aware was that Dr. Samet was going to be used as a witness on this report, that it would -- it would inform his expert opinions, and we did not have the opportunity under the procedures of this court to go back to Dr. Samet and question him about it because we did not have supplemental -- we did not have supplemental notice given to us by the government.

THE COURT: There was constant redepositioning -- if there's such a word, everybody -- and I know that, and that's because between the two sides, you have probably amassed all of the experts in the relevant fields, at least in our country and maybe in a couple of other countries. All of these experts are deeply involved in ongoing research.

That has been one of the problems here, and that's a problem I certainly never anticipated, but then I didn't anticipate a lot of things.

But, in any event, the parties have dealt with that issue by periodically taking additional depositions of these experts who are working day by day on the scientific issues that we're all grappling with in this case.

Certainly Dr. Samet's intensive involvement in the preparation of this report as well as all his other ongoing activities were very well known to the defendants. It's no surprise, and it's certainly not any surprise given his particular role in the 2004 report that he was going to speak to

that issue today.

Joint defendants could have asked for a deposition. If the government had turned them down, that would have been one problem.

The joint defendants came to me -- I don't know how many times -- but a certain number of times objecting when the government did turn them down for additional depositions. And again, I cannot say that every time the defendants won those motions but they won them a lot of times over the government's objection. That was not the case with Dr. Samet.

MR. McDERMOTT: Your Honor, I will plead guilty to two offenses. One, we did tie up an all full lot of experts and we certainly did come back to the court from time to time and seek supplemental --

THE COURT: That was fine.

MR. McDERMOTT: -- but we did so only in response to supplemental disclosures. We didn't just go out willy-nilly and ask to redepose people.

It was always in response to a trigger from the government saying, our opinions are going to change, or here are additional reliance materials. This is stuff that our expert is going to rely on.

We got no such notice in the case of Dr. Samet, Your Honor.

MS. EUBANKS: Your Honor, Dr. Samet's opinions have not

changed and that's what the rule refers to.

THE COURT: We're talking about, I believe -- based on what I read in his direct, at least -- a continuation of his opinions, if anything fortified -- let's use that word -- by additional research that comes in.

Objection is overruled. Let's proceed, everybody, please.

BY MS. EUBANKS:

Q. Regarding the conclusion for the IARC report, what is the significance of that again, Dr. Samet? If you will take us back to where we were.

A. Well, again, the IARC report, the large volume included the findings of two reviews: one related to involuntary smoking and one related to active smoking.

If I could just comment on the 2002. We did hold a press conference in 2002, released the results of the reports and they were posted on the IARC Web site.

Q. Could we see the next slide?

What's the significance of the findings that are shown on the screen to your conclusions?

A. The report on active smoking covered issues related to the exposures and doses of various carcinogens and toxins received by the smoker.

This is one of the conclusions from the IARC volume. Analysis of the ways in which people smoke modern cigarettes

shows that actual doses of nicotine, carcinogens and toxins depend on the intensity and method of smoking and have little relation to stated tar yields.

And there was a further conclusion stating that the tar and nicotine yields, as currently measured -- this would be in reference to machine measurement -- are misleading.

Q. All right. Could we see the next slide, please?

THE COURT: I'd like to go back to that last quote for a minute.

When that quote refers to the current measurement of tar and nicotine yields being misleading, were you, in fact, referring to the FTC measurements and being polite by not mentioning them specifically or were you referring to other measurements?

THE WITNESS: Let me say the -- these reports are global in their perspective.

THE COURT: Yes, I understand.

THE WITNESS: So they would refer to various protocols for machine measurement of yield.

So the FTC method is one. There are other variants, if you will, of that method, but this is not -- this is referring to machine measurement, in general, I would stay interpreting it for you.

BY MS. EUBANKS:

Q. With regard to your opinions here, what is the significance

of the information found at page 1187 of the IARC report?
A. Well, the IARC report is perhaps so thick because there was so much evidence available and up through 2002 on the risks of active and passive smoking.

This is a summary of the evidence on smoking as a cause of various kind of cancers, and I think what is important in this list is that as late as 2002, roughly 50 years into the study of smoking and cancer, we're finding new sites that were causally linked to cancer, to smoking.

Some of those, for example, the uterine cervix, the bone marrow, leukemia, cancer of the liver, were new additions to the list of cancers caused by smoking.
Q. Now, in terms of IARC's findings regarding passive smoking, can you tell us why were IARC's conclusions significant given the fact that in 1992 the EPA had issued its risk assessment?
A. Well, of course, I think there's a difference in the scope and focus of these organizations.

IARC, as an entity of WHO, reviews evidence to present it to the entire world for public health purposes. So I think the U.S. EPA, as an agency based in the United States, has a different focus and emphasis in its reports.
Q. Let's move to the next slide, please.

Now, we are up to the 2004 Surgeon General's report. What was your involvement in that report?
A. I was the senior scientific editor for this report, which

was released earlier this year.
Q. How long did it take to compile the 2004 Surgeon General's report?
A. Approximately three years of work were involved in producing the report.
Q. See the next slide. There's a conclusion that's shown on the screen.
What can you tell us about the significance of that to your opinions?
A. This is one of the many conclusions in the report. This again addresses the question of machine measurement of yields of tar and nicotine.
The conclusion reads: Smoking cigarettes with lower machine-measured yields of tar and nicotine provides no clear benefit to health.
Q. All right. Can we see the next slide? There's another conclusion.
Can you explain the significance of that statement on the screen, please?
A. This report, of course, was an updating of prior Surgeon General's report on the full scope of evidence on active smoking and disease.
Like the IARC report, a number of cancers were added to the list of those cancers caused by smoking; for example, cervical cancer and leukemia. Other additions were cataract and

dental disease.

I think it's also important to note that the report concluded that smoking greatly diminishes the health of smokers. That is, smokers compared to nonsmokers who are simply not as healthy in general.

Q. Could we go to the next slide for 2004, please? The British doctors' study, can you --

THE COURT: Again, I need to go back to the last slide for a minute.

So if I read that correctly, the 2004 Surgeon General's report is saying that now amongst the types of cancer that had not previously been causally associated with smoking were -- and I should say the forms of illness or disease, perhaps is a more accurate way to say it -- the forms of disease that had not previously been causally associated with smoking were cataracts and gum disease; is that correct?

THE WITNESS: Yes, Your Honor. These -- the list includes a number of diseases, cancers and other diseases. So, for example, cataract which had been looked at --

THE COURT: I saw that in your direct.

THE WITNESS: -- 15 years, that's correct.

THE COURT: And I certainly had never -- although I am hardly an expert, but I had not ever read that before. But that is the conclusion in the 2004 Surgeon General's report?

THE WITNESS: That's correct.

THE COURT: And gum disease as well?
THE WITNESS: That's correct.
THE COURT: Okay.
BY MS. EUBANKS:
Q. Can you describe the British doctors' study and its significance? I note it was also in 2004.
A. Well, this is the 50-year report from the study that Richard Doll began in 1951. And I think it's significant because, as this study and others continued, they provided the evidence base that we used in the 2004 report, for example, to better define what the risks of smoking are.
Q. All right. Why don't we go forward.
Now, the last two charts that we're going to look at, the first one that we have up summarizes what?
A. This is a summary of the various diseases and other adverse health effects caused by active smoking as listed in the Surgeon General's report and the report of WHO, IARC.
Q. And if we turn to the final slide, can you tell us what that shows?
A. This is a similar summary for the adverse effects, diseases and other adverse effects. For example, the effects on respiratory health of children of second-hand smoke exposure.
Q. All right. Dr. Samet, when would you say that the evidence made clear that there was a causal conclusion that second-hand smoke caused disease?

A. Well, I think the first conclusions were voiced in 1986 in the three reports that I mentioned on smoking, passive smoking in lung cancer.

Q. Let me ask the question again and see if I can get a direct response to the question that I asked.

I understand that that there are reports out there, but what I'd like to you tell the court is: When would you say that evidence made clear that there was a causal connection between second-hand smoke and disease?

A. 1986.

Q. Thank you.

MS. EUBANKS: Your Honor, I have no further questions at this time.

THE COURT: It's a little earlier than usually. Mr. McDermott, are you ready to start cross or do you want a short break now?

MR. McDERMOTT: The court's pleasure. Either way.

THE COURT: Let's begin the cross then.

CROSS-EXAMINATION

BY MR. McDERMOTT:

Q. Good morning, Dr. Samet. I'm Bob McDermott.

I want to begin by asking you about the subject of environmental tobacco smoke. I tend to use the shorthand ETS, I know you use passive smoking. I trust we will speak a common language, and if there's any confusion, just let me know.

You indicated in your direct testimony a few minutes ago that the first studies, epidemiological studies linking ETS to lung cancer came out in 1981; correct?

A. I said that, and that's correct, except for a very minor earlier report from Hong Kong.

Q. Would it be fair -- well, let me ask you.

You also mentioned a brief passage in the '72 Surgeon General's report where he talked about sidestream smoke, correct, during your slide show?

A. I mentioned a section --

MS. EUBANKS: Objection, Your Honor, to the characterization that what we just showed, what we just demonstrated was any kind of show.

THE COURT: No. Objection is overruled.

Go ahead, please.

BY MR. McDERMOTT:

Q. Do you have the question in mind, Doctor?

A. Well, I referred to a section of the 1972 Surgeon General reported from which a passage had been extracted.

Q. And that referred to sidestream smoke; correct?

A. That is correct.

Q. Sidestream smoke is not the same thing as environmental tobacco smoke, is it?

A. No. Sidestream smoke is one of the principal sources of second-hand smoke.

Q. And it gets greatly diluted and mixed with the air and exhaled main stream smoke and is present in far, far -- sidestream smoke --

THE COURT: I need to interrupt you, Mr. McDermott, for some basic clear definitions all given at the same time. And I know these are all related terms, but I need to be very clear on the differences between them.

Sidestream smoke, mainstream smoke, second-hand smoke, and environmental smoke. To the extent there is a difference between those terms, and I know there is between some of them, would you please spell that out clearly?

THE WITNESS: If I can just talk then, Your Honor.

The sidestream smoke refers to the smoke given off by the cigarette while it smolders, so that is a smoke that comes off the end of the cigarette.

THE COURT: It has not been inhaled by the person smoking?

THE WITNESS: Correct.

So mainstream smoke is the smoke directly inhaled by the smoker through the cigarette as they puff on the cigarette.

THE COURT: And would it be fair to say that's the smoke that is exhaled after having been smoked?

THE WITNESS: So a substantial portion of the smoke, mainstream smoke, is retained in the lung, but of course some is exhaled back into the room.

THE COURT: Okay.

THE WITNESS: And then if I can say the last piece of the story, then, is that the combination of the sidestream smoke, the diluted sidestream smoke, with the exhaled mainstream smoke is what Mr. McDermott has called ETS, or environmental tobacco smoke, and I've been using the term second-hand smoke, and second-hand smoke and ETS might be viewed as equivalent.

And just one last piece of semantics. When I talk about passive smoking or involuntary smoking, I'm referring to the breathing in of this second-hand smoke or ETS by the nonsmoker.

MR. McDERMOTT: May I proceed?

THE COURT: Yes.

BY MR. McDERMOTT:

Q. So we are agreed, Dr. Samet, that nonsmokers don't walk over to cigarette ashtrays, bend over and breathe in sidestream smoke directly as an ordinary course of events; correct?

A. Well, in the ordinary course of events I would not expect that they will do that, no.

Q. The sidestream smoke becomes greatly diluted, and so ETS is much more diluted than sidestream smoke; correct?

A. Well, ETS becomes diluted and the extent of dilution depends on the circumstances.

Q. All right. Notwithstanding the Surgeon General's reference in 1972, would you agree with me that it would be an accurate

statement of the state of the science in 1979 that healthy nonsmokers exposed to cigarette smoke have little or no physiologic response to smoke and what response does occur may be due to psychological factors?

A. I'm sorry. Can you repeat the question?

Q. Certainly.

Would you agree with me that it is an accurate statement of the state of the science in 1979 that healthy nonsmokers exposed to cigarette smoke have little or no physiologic response to the smoke and what response does occur may be due to psychological factors?

A. Well, I would say that by 1979 there had probably been very limited exposure studies of healthy nonsmokers to second-hand smoke. There were certainly studies carried out around the time. I can't demarcate the literature in 1979 versus earlier, looking at responses, for example, of the eyes and upper airway and irritation responses.

Q. So, to answer my question, is it a fair statement of the science or not, or you don't know?

A. Certainly by 1979 there were studies on adverse respiratory effects of second-hand smoke on children showing increased risks for lower respiratory illness and respiratory symptoms.

Q. Doctor, if you would answer my question. Is what I just read to you a fair statement of the state of the science in 1979 or not?

A. Why don't you repeat the question, please?
Q. Healthy nonsmokers exposed to cigarette smoke have little or no physiologic response to the smoke and what response does occur may be due to psychological factors?
A. I think the answer to that really hinges on what is meant by physiologic responses. Clearly, by 1979 there were studies showing adverse effects on the health of children.
Q. So you do not agree with that statement?
A. Not if I consider the data on children. I've not -- that statement may have to do with what is meant by physiological responses, but there was clear evidence of adverse effects on children by 1979.
Q. So you disagree with the Surgeon General's characterization of the statement of science?
A. Again, I would have to see what was meant by physiological response. I suspect this refers to acute exposures of individuals to second-hand smoke.

I don't know whether, in making this statement, the Surgeon General had considered the evidence on children which had been reported at that time. So I can't interpret this without a better context.
Q. Doctor, I thought you were familiar with the Surgeon General's reports. That was part of the expertise you offered in this case.
A. Well, I would certainly like to see the report from which

this has been extracted and the context in which this is made.
Q. We will show it to you right now.
MR. McDERMOTT: For the record, I've handed the witness a copy of Joint Exhibit 064071.
Q. And the page reference, Doctor, is on 11-28. It's the 1979 Surgeon General's report. Have you had a chance to review the report?
A. Well, I've looked at these several pages. I would note that in fact, I think my recall was correct and they did comment on the information on irritation, which I would certainly regard as a physiological response.
I would like to see perhaps the antecedent pages that describe these studies' physiological response. Again, I suspect that these probably in reference to acute exposure studies and whether --
THE COURT: Excuse me, Dr. Samet. If you would slow do you know a little bit.
THE WITNESS: I'll try.
THE COURT: I would like you to repeat what you just said.
A. Well, again, if -- well, the passage has disappeared now.
But my comment earlier was that in fact the -- in summary, comments on the literature that I noted describing irritation and annoyance, which I would certainly count as a physiological response, the comment about healthy nonsmokers

exposed to cigarette smoke I suspect is in reference to human exposure studies and whether there were changes, for example, in things such as respiratory rate or heart rate.

Q. Do you consider irritation a health -- significant health risk?

A. I would consider it a physiological response.

Q. Do you consider annoyance a significant health risk?

A. Again, I would consider it to be an adverse consequence of exposure to smoking.

Q. But not a threat to public health, Doctor? Isn't that correct?

A. Well, it's a threat to well-being, certainly.

Q. Dr. Samet, you spoke of the two 1981 epidemiological studies by Hirayama and Trichopoulos?

A. Yes, I did.

Q. These were not the only epidemiological studies relating to ETS that were published in the early '80s; isn't that correct?

A. Other studies were subsequently published.

Q. In fact, you reviewed some of those studies in the 1984 article which you authored, didn't you, Doctor?

A. I did.

Q. And your research was supported by the National Cancer Institute?

A. My work at the time was supported by the National Cancer Institute.

Q. Let's see Joint Defense 067821.

THE COURT: 0626781?

MR. McDERMOTT: 067821.

BY MR. McDERMOTT:

Q. For the record, that's an article that you wrote entitled: Relationship between passive exposure to cigarette smoke and cancer.

Can you give the doctor a copy?

You're familiar with that article, obviously, Dr. Samet?

A. Yes, I am.

Q. All right. Let's turn to Table 1, please. Table 1.

In this table, Doctor, you list three studies that purport to show significant effects of passive smoking and lung cancer rates; correct?

A. Give me a moment to look again.

Q. Certainly.

A. (Pause) Yes, that's correct.

Q. And then you have a Table 2 in the article; correct?

A. There is a Table 2.

Q. And you list there five studies that do not show a statistically-increased elevated risks of lung cancer among ETS exposed spouses; is that right?

A. They show varying levels of effect and they were a group because they were not statistically significant.

Q. Correct. And you said at the time in your article that there was a paucity of data that was directly relevant to the hypothesis that passive smoking is a risk for lung cancer, didn't you?
A. At that point the evidence was just unfolding.
Q. And you commented on the paucity of data; correct?
A. Well, if I could find -- may have used that word. I would need to see where --
Q. Can you show us 236? Under Conclusions, in summary.
Second sentence. The paucity of data contrasts sharply with the literature crated in the 1964 Surgeon General's report which characterized active smoking as a cause of lung cancer.
You wrote that in 1984, Dr. Samet?
A. That's right. I commented on the nine studies and made the contrast.
Q. And you comment on all the studies that are extant at the time; correct?
A. To my knowledge.
Q. And your scientific judgment, this was -- this reflects your scientific judgment in 1984, correct, this article?
A. Yes.
Q. In fact, Doctor, at the second sentence of your conclusions, if we can go on, you report that this paucity of data highlights the weaknesses of the data for passive smoking; correct?
A. Well, in this article I commented on the methodological

issues involved in studying passive smoking in lung cancer.
Q. And you noted particularly the weaknesses of the data; correct?
A. I commented on, again, the methodological issues involved and the challenges.
Q. And finally, Doctor, you commented then, page 237, first sentence of the first full paragraph.

The association between passive smoking and lung cancer did not yet meet the criteria applied to active smoking in the 1964 Surgeon General's report. Correct?
A. This is a comment that I made, correct.
Q. Doctor, I prepared a chart -- I basically transposed some of the information you have so we can look at it more clearly.

Let me see JDM 060285.

And what this does, Dr. Samet, is to basically extract from your article the studies that you reviewed and put them in a form which is a little bit easier, for me, at least, to deal with.

Would you agree with me that this accurately sets forth the information in your article?
A. I can't agree with that without looking at the table myself.
Q. Please take your time.
A. Well, this -- I'm afraid this is not an easy translation because, for example, what you have for Trichopoulos, 2.113, is not what I have in my Table 1 where I show the dose response.

What you have for Hirayama is not what I have in my Table 1. So I'm reluctant to -- so I don't know where this table came from. It's certainly not Table 1 from my article.

Q. Doctor, let me show you -- excuse me -- let me show you U.S. Exhibit 63708. This is taken from the table -- the table is taken from the NRC report which you rely on in your expert materials, the numbers.

Can we get -- can we zoom in on the top seven studies, please? That's not very....

THE COURT: Did you transpose the columns from this table, which is either 12 or 13-4, to the exhibit you just made up and showed Dr. Samet?

MR. McDERMOTT: That's correct, Your Honor, and this is from information in Dr. Samet's reliance material. This was the cleanest and clearest exposition of what those numbers were.

And it might help the court if I could ask Dr. Samet if we can go back to my exhibit to explain what those columns are. It might help the court.

BY MR. McDERMOTT:

Q. Dr. Samet, can you tell us what "overall relative risk" means?

A. I'm a little reluctant to since I did not prepare this. I will speak to it in general, but I have no idea where this came from at the moment.

Q. I understand. Speak generally.

A. In general, I would assume that relative risk refers, of course, to the comparison of risk and expose. I assume in this case marriage to a smoker compared to the comparison, which would be marriage to a nonsmoker. But since I didn't develop this table, I don't know what you mean by overall.

Q. And how about confidence interval? What does that mean, in general?

A. A confidence interval, in general, describes sort of the width around this relative risk where we think the truth lies, and typically we use 95 percent confidence intervals to describe that window.

So if the number is 0.90 to 1.54, that means that we think with 95 percent certainty essentially that the true increase in risk lies within that bound.

Q. And, Doctor, isn't it true that when the confidence interval goes below 1, that you cannot say that you have statistically significant results?

A. Well, at the stated level of confidence and the confidence interval, you can say that.

Q. Using the 95 percent confidence interval, when the confidence interval dips below 1, the lower end, that is not considered statistically significant; correct?

A. If the criterion for statistical significance is P less than point 05, and the lower confidence interval goes below 1, then the finding is not significant at P less than point 05.

Q. So the answer to my question is, yes, using a 95 percent confidence interval when the lower bound is below 1 you cannot say the study is statistically significant; correct?

MS. EUBANKS: Objection, Your Honor. The witness has answered it.

THE COURT: The objection is overruled. The doctor can answer.

A. Well, I did answer the question. I think I just made a clearer answer, which was not statistically significant at P less than point 05 because you said 95 percent confidence interval.

Q. So the P point 05 is the same thing as the 95 percent confidence interval? Again I want the record to be clear.

A. They are not the same thing. I mean, you've offered me a comment about interpretation of the confidence interval, but a P value and a confidence interval are different.

Q. I understand that. But a P value of point 05 means you are utilizing confidence interval of 95 percent, correct, which is typical for epidemiological studies?

A. Again, your comment about interpretation of the P value and the confidence interval are correct. They are different computationally and they may be used for different purposes.

Q. Doctor, I'm going to ask you to assume, and we will close up the record by the time we come back after lunch and establish it.

I want you to assume that that is an accurate transposition of numbers that come out of a document that you rely on, which is U.S. 63708.

MS. EUBANKS: Objection, Your Honor, as to an assumption. We are here to get the facts and the evidence. To ask the witness --

THE COURT: Well, it's subject to being tied up. And for the moment I'm going to operate on the assumption that counsel would not be so foolish as to give me an assumption and a transposition of statistics that was inaccurate.

MR. McDERMOTT: I can sure you, Your Honor, that if it's inaccurate, heads will roll, including mine. It is not intentional.

THE COURT: Okay.

BY MR. McDERMOTT:

Q. I'm going to ask you to assume, Doctor, that this is an accurate transposition of the numbers.

In that case, the Garfinkel study does not show statistically-significant results; correct?

A. I'm sorry. Can I just have a clarification?

This is data extracted from the 1986 National Research Council report showing studies through 1984?

Q. Correct.

A. Okay.

Q. The Garfinkel study does not show statistically-significant

results; correct?
A. At P less than point 05, correct.
Q. The same is true with the Chan study in 1982?
A. Correct.
Q. The same is true with the Corea study in 1983?
A. Correct.
Q. The same is true with the Kabat study in 1984?
A. Correct.
Q. The same is same true of the Gillis study in 1984?
A. Correct.
Q. The same is true of the Koo study in 1984?
A. Correct, as presented.
Q. You didn't mention any of these studies in your direct examination, did you, Doctor?

You didn't refer to these studies when you were discussing environmental tobacco smoke?

A. I made no attempt to refer to every extant study. They are summarized in the reports that I cited.
Q. Doctor, let me show you a final extract from your paper. Can we have 68 -- excuse me -- 67821 again please, page 237.

In 1984, it was your view that -- this is a first full paragraph.

In conclusion, the association between passive smoking and lung cancer does not yet meet the criteria applied to active smoking in the 1964 Surgeon General's report. While

confirmation of passive smoking as a risk factor for lung cancer --

THE COURT: Not too fast, Mr. McDermott, please.

MR. McDERMOTT: Yes, Your Honor.

Q. While confirmation of passive smoking as a risk factor for lung cancer would offer new ammunition against tobacco, the available evidence does not permit definitive judgments.

That's what you said in 1984; correct?

A. That's correct.

Q. That was an accurate statement of the state of the science in 1984; correct?

A. Well, this is my view of where the evidence stood in 1984.

Q. All right. You also go on to note. In the face of difficult methodological problems, particularly that of accurately quantifying dose, unimpeachable data will be difficult to obtain. Correct?

A. That is read again as written.

Q. Let's talk about some of those methodological problems, Dr. Samet.

You note in page, at 228 of this article under Methods, third sentence, The results of both may be affected by misclassification of exposure and confounding by other risk factors.

You're commenting there, Doctor, on the problems of misclassification of exposure confusion of smokers and

nonsmokers; correct?
A. Well, this is a -- a general comment about methodologic issues that arise in epidemiological studies.
Q. I understand that. And confounding by other risk factors.
What are the other risk factors that might confound an ETS epidemiological study?
A. Well, I think to my knowledge then and now there are very few, perhaps -- because there are few well-documented causes of lung cancer in never smokers, some occupational exposures, in particular, although certainly not well looked at at the time indoor radon might also have been raised as a possible potential compounder.
Q. What about diet, Doctor? Has that been raised as a possible compounder?
A. It's been raised. In my way of thinking, diet has never met the definition --
THE COURT: Just a little slower.
A. We've never shown that diet itself is a risk factor for lung cancer in never smokers.
Q. That view isn't shared by all scientists, however, is it, Doctor?
A. It may well be. There's certainly been work on diet and lung cancer, but it's been certainly generally in reference to smokers. There's been relatively limited work on lung cancer and diet in never smokers.

Q. You are familiar with Dr. Mataneski, a colleague of yours at Johns Hopkins?
A. She's a faculty member in my department.
Q. And you're aware she's published research on dietary confounding and ETS lung cancer epidemiological studies?
A. I'm not aware that Dr. Mataneski has published a single paper showing that diet is linked to risk for lung cancer in never smokers.
Q. She hasn't published a paper, Doctor, but she's certainly raised it as an issue that needs study, hasn't she?
A. I said I thought that there was little evidence available and I know Dr. Mataneski's work. She's in my department. And that paper does not address lung cancer and diet in never smokers.
Q. Let's see what she says. Can I see JD 053942?
THE COURT: This is an unpublished, unpeer-reviewed paper, question mark?
MR. McDERMOTT: This is an excerpt from an abstract called Characteristics of Nonsmoking Women in NHANES 1 and NHANES 2 Epidemiological Follow-up Study With Exposure to Spouses Who Smoke published in the American Journal of Epidemiology in 1995.
BY MR. McDERMOTT:
Q. And she reports that many of the differences that authors observed between women who are exposed and not exposed to

passive smoking could affect the risk of cancer. Therefore, they recommend that future studies of nonsmokers examine the influence of both passive smoking and diet on the risk of disease rather than examine the influence of a single factor.

Correct?

A. Well, it says that, but again the paper does not provide any information directly on diet and risk of lung cancer in never smokers. As I've said, I'm familiar with this paper.

Q. This is a concern, an issue that has been raised by other scientists, correct, Doctor?

A. Well, the issue is raised, but no evidence is supplied directly relevant to whether diet could confound the passive smoking lung cancer association.

Q. It is scientifically legitimate to question whether or not diet has a role in the perceived incidence of ETS-related lung cancer, isn't it, Doctor?

A. Well, again, certainly authors are entitled to offer their speculations, but they offer no direct evidence that diet is associated with lung cancer risk in never smokers here.

Q. Let's talk about measuring exposure, Doctor.

THE COURT: I think now, Mr. McDermott, we will take a break.

Doctor, you will step down.

We will take 15 minutes, everybody.

(Recess began at 11:00 a.m.)

(Recess ended at 11:15 a.m.)

THE COURT: Mr. McDermott, please.

MR. McDERMOTT: Thank you, Your Honor.

BY MR. McDERMOTT:

Q. Let me backtrack for just one second. I should have asked and I think the answer is obvious, but we might at well make the record clear.

Your colleague, Dr. Mataneski, presumably you think she's a competent scientist --

A. Yes.

Q. -- or she wouldn't be on your staff?

A. Yes, I do.

Q. I want to talk to you now a little bit about measuring exposure. That's one of the problems that you identified in your methodological problems that you identified in your 1984 article; correct?

A. Correct.

Q. Would it be fair to say that as of 1986, methodological constraints, particularly accurately quantifying exposure, had produced conflicting results in the epidemiological literature regarding passive smoking?

A. Well, there may have been many sources of conflicting results. I mean, methodological issues would be one source of potentially conflicting results.

Q. Let's see what you wrote. Can I have JD 067843?

For the record, this is a letter that you wrote with Dr. Coultas in 1986 published in the Western Journal of Medicine, the right-hand column, first full sentence.

Because of methodologic constraints, particularly in accurately quantifying exposure, study as a passive smoking and lung cancer, offer conflicting results. Correct?

A. That's correct.

Q. And one of the problems, Doctor, is that an awful lot of the exposure information comes from surveys; correct?

Questionnaires as opposed to actual physiologic measurement.

A. Well, questionnaires were used in the epidemiological studies on passive smoking in lung cancer, that's correct.

Q. And this has been basically the exclusive method in the large epidemiological studies, for quantifying exposure in the large epidemiological studies linking ETS with lung cancer; is that correct?

A. That's generally correct. Some of the more recent studies on other health outcomes have used biomarkers, but questionnaires are the principal source.

Q. In your direct testimony you were asked whether or not epidemiologists had measured exposures to ETS, and you said researchers have also placed monitors on people so the total daily exposures, those received in the full range of everyday living, can be measured.

Do you recall that testimony? I'll be happy to show it to you on the screen if you would like.

A. Yes, I do.

Q. You didn't mean to imply by that, Dr. Samet, that any epidemiologists have actually studied populations over a long period of time with the subjects of the study wearing personal monitors, did you? That hasn't happened, has it?

A. Of course not.

Q. And isn't it true, Dr. Samet, that questionnaires don't accurately quantify exposure?

A. Well, it certainly can't answer that question in a -- in a general sense as you've posed it.

Any questionnaire may have issues related to validity and reliability no matter what the topic is.

Q. Can we see JD 067821? I'd like page 231.

This is your 1984 article, Doctor, which you have, if you would like to take a look at it. I would ask you to focus on the right-hand column, seven lines from the top.

Thus, with regard to domestic exposure, simple descriptions of spouse smoking behavior cannot satisfactorily define gradients of exposure.

That's what you wrote in 1984; correct?

A. I noted that and then commented further, that they are useful to document that exposure has occurred.

Q. Like a light switch. It's either on or off, but you can't

tell how much light, can you, Doctor? It's not a rheostat?
A. I'm not sure about the analogy, but --
Q. I'm not sure about it, either. Let me try again.
You can tell whether or not there's been exposure, yes or no, but you can't quantify the amount of exposure; is that correct?
A. Well, I think what the second sentence refers to the -- the one after the highlight is the fact that, on average, women or men married -- who don't smoke themselves who are married to smokers would be presumed to have more exposure than people not married to smokers.
Q. I understand. Let me see JD 678426, please. Page 250. This is a paper that you co-authored, I believe.
I'm sorry. You were a co-author of the study; correct?
A. That's correct.
Q. For the record, it's called: Variability of Measures of Exposure to Environmental Tobacco Smoke in the Home, and published in Combustion Process and the quality of the indoor air environment 1988. And if you will focus on the last two lines on page 250.
We conclude that, because of the marked variability of these measures, multiple measurements are needed to establish a stable profile of exposure to environmental tobacco smoke in a particular home or environment?
THE COURT: In a particular home or individual.

MR. McDERMOTT: Excuse me. Home or individual. Thank you, Your Honor.

Q. Furthermore, detailed questions to quantitate exposure offer little additional information beyond whether the subject was exposed or not?

You wrote that, Doctor?

A. I think this refers to the fact that it was very difficult using questions to quantify exposure beyond the fact of exposure, correct.

Q. And Hirayama and Trichopoulos in the two 1981 epidemiological studies you referred to did use questionnaires to quantify exposure, did they not?

A. Well, they again used that measure I referred to, was their marriage to a smoker or not, which again would qualitatively signal greater exposure. They used some information on cigarette smoke by their spouse to try and further quantify it, and I think this paper speaks to the limitations of that kind of information.

Q. And in light of those limitations it would be fair to have reservations about the reliability of the dose response findings, would it not, Doctor?

A. Well, I think, in general, the main weakness here is that we tend to misclassify exposure. In epidemiological studies when that happens, it usually detracts from our ability to find a dose response. Air, if you will, introduces additional noise

and it's harder to find the signal.
Q. Let me move on to the 1986 Surgeon General's report which you mentioned in your direct testimony. It's JE 63709. And let me refer to page 71, Table 9.
It's a little bit hard to see on the screen, but let me ask you based on your study, Doctor. Do you recognize that the eight epidemiological studies, which you reviewed in your 1984 article, are also noted in the Surgeon General's report?
A. Well, I won't take the time to make a detailed comparison, but I will assume they are here.
Q. I'm not trying to mislead you.
In addition, there were five newer studies that weren't available at the time you wrote your article and I'll just list them for you. They are in Table 9.
Wu in 1985, Garfinkel in 1985, Lee in 1985. I may not pronounce this correct. Au-KAE-ba in 1986. A-k-i-b-a. Pershagen in 1986.
Does that sound right, Dr. Samet?
A. It sounds right, correct.
Q. To simplify the comparison, I'm going to go back to an expansion of the chart I showed you earlier.
Can you display J-DEM 060286?
What I've done is just, using the same format I showed you earlier -- and we will get to prove at lunch, Your Honor -- simply transposed the results of those five additional studies

considered by the Surgeon General in 1986 into the same format that I showed you earlier.

And it is correct, Dr. Samet, that the Wu study did not show statistically-significant elevated risk, isn't that so?

MS. EUBANKS: Your Honor, I just want to make clear what this is based on because it's a number of assumptions that are here.

THE COURT: You have to speak up.

MS. EUBANKS: There are a number of assumptions that are raised here in the question, and there was a reference to something over the lunch break.

I just want it to be clear what the witness's testimony is about this that he's viewing and which assumption it is he's adopting in his answer.

THE COURT: All right.

BY MR. McDERMOTT:

Q. If you look at the legend on the bottom, Doctor -- and I see I've got a pointer here. I'll try to read it to you.

The relative risks in confidence intervals for all studies, other than Wu, as summarized in the National Research Council environmental tobacco smoke measuring exposure and assessing health effects from Table 12-4 at page 232, that's something on your reliance materials, isn't it?

A. That report is correct.

Q. And the relative risk and confidence intervals for Wu in

1985 are summarized in the health consequences of involuntary smoking at page 83. That's the Surgeon General's report. And the reference there is JE 063709. So that's the source of the data.

Do you understand that?

A. I understand that.

Q. Now, with that explanation, is it not the case that the Wu study did not find statistically-significant elevated risks?

A. As presented here, yes.

Q. And the Garfinkel 1985 study also did not find statistically-significant results; correct?

A. The same answer.

Q. And same for Lee 1985, not statistical elevated risks?

A. That's.

Q. And for Akiba 1986, there is no statistically-significant elevated risk; correct?

A. Akiba, yes.

Q. And for Pershagen, P-e-r-s-h-a-g-e-n, 1986; again, there is no statistically-significant elevated risk; correct?

A. Pershagen. And, yes, I would just note that they are all above 1, indicating some degree of increase but not statistically significant.

Q. But the five additional studies that were available for the Surgeon General's review in 1986, of those five, not a single one showed statistically-significant elevated risks; correct?

A. I would actually note that the reliance document, the National Research Council report summarized those results, and overall when the evidence was put together there was a significantly increased risk at that time in 1986.

MR. BERNICK: Your Honor --

MR. McDERMOTT: Move to strike as nonresponsive.

MR. BERNICK: Mr. McDermott is not aware --

THE COURT: Well, it may have been nonresponsive to the question you asked, but it certainly was a significant answer in terms of the court understanding things. But I want to follow up for a minute.

Dr. Samet, you are indicating that as presented on this particular slide no one of those five more recent studies was statistically significant individually; is that correct?

THE WITNESS: That's correct. As presented, that is correct.

THE COURT: But then were you saying when a further study was done that considered all five together, the conclusion was that they were statistically significant?

THE WITNESS: No. Actually, in the document the National Research Council report they put all the studies together that had been published. That would include these five and the earlier studies.

And when that was done, pulling all of the evidence together there was a statistically significant increased risk of

approximately 34 percent if I recall correctly.

THE COURT: All right. Go ahead, Mr. McDermott.

I will, by the way, treat that as a redirect question and answer from the government.

Go ahead.

BY MR. McDERMOTT:

Q. Dr. Samet, in 1984 you said the association between passive smoking and lung cancer did not yet meet the criteria applied to active smoking in the 1964 Surgeon General's report; correct?

A. That's correct.

Q. The addition of five statistically-insignificant results doesn't offer that picture, does it, Dr. Samet?

A. Well, the '86 Surgeon General's report was based on far more than five additional studies; it was a comprehensive review of all of the evidence, which is what is appropriate for use of the criteria for causality.

So I think that the report in its other chapters goes over the other relevant lines of evidence.

Q. But the human data is as I've laid it out there; correct, Doctor?

A. I am making the assumption that it's so, that as laid out. And again the new five additional studies did all indicate increased risk, not statistically significant, and --

Q. Thank you. Let me turn to the 1992 EPA risk assessment. You testified briefly about that in your direct.

The EPA risk assessment used a meta-analysis, did it not?
A. The EPA risk assessment included a meta-analysis, correct.
Q. And it examined approximately 31 spousal studies to find an overall relative risk of approximately 1.19; is that correct?
A. That, I think, is correct, by my recollection.
Q. And I don't want to jog your memory, but is it also approximately correct -- or is it consistent with your memory that the confidence interval was about 1.04 to 1.35?
A. My memory does not include the confidence interval.
Q. Okay. Let's see if we can throw up -- let me see what's... excuse me, Your Honor.
Doctor, for my benefit, perhaps for the court's but certainly for mine, why don't you explain very briefly what a meta-analysis is?
A. Well, again, without getting too technical, a meta-analysis involves a review of all the evidence, and in a quantitative meta-analysis the findings the individual studies are put together statistically, if you will, and sort of taking account of how variable each one is so that the evidence can be joined together to see what the overall signal is when the individual studies are combined.
Q. So the meta-analysis that you referred to earlier that the NRC did in 1986, it did not involve new studies, it did not involve original research with new populations; correct? It was

just massaging the numbers with the existing reports?
A. Well, the '86 NRC was essentially the same as what the EPA did. It took all the evidence up to that time and pooled it. And then of course by 1990-91 when the EPA was doing its work, there were more studies.
Q. You've done some meta-analyses and presented some meta-analyses in your direct testimony, have you not, Dr. Samet?
A. I have relied on published meta-analyses.
Q. You presented them in your direct testimony?
A. That's correct.
Q. The meta-analyses that you presented using 95 percent confidence interval; correct?
A. That's correct in general, yes.
Q. In 1992 the EPA used a 90 percent confidence interval; isn't that correct, Dr. Samet?
A. That's correct.
THE COURT: What's the significance, if you can describe it, between a 90 percent confidence level and a 95 percent?
I understand, of course, that it's a 5 percent difference, but is there any other meaningful way you can explain that difference? That's one question.
And my second question is, is a 90 percent confidence level generally accepted as adequate in the scientific world?
THE WITNESS: Complicated questions.

The 95 percent confidence interval describes sort of the area of coverage with 90 percent -- 95 percent confidence.

The 90 percent describes it with 90 percent confidence. So it, if you will, it narrows a bit, and analogous to Mr. McDermott's discussion of the P point 05, it's now more analogous to P point 10.

In using 95 percent or 90 percent, there's in a way a prior decision. The 90 percent decision essentially was made because at the time EPA believed that it was appropriate because they would not consider the possibility that passive smoking reduced the risk of lung cancer, so they used the 90 percent confidence intervals.

I know that this was discussed extensively at the time because it was a bit of a departure from the usual use of 95 percent confidence intervals, and it was -- has since been a matter of debate.

But essentially by doing the 90 percent confidence interval there's, if you will, a higher chance that results will be statistically significant because there's been a change from PO 5 to P point 1.

And then there's also the implicit assumption that at that point there's a very strong prior belief that passive smoking could not protect against lung cancer. The 95 percent, if you will, is evenhanded. It says passive smoking could cause lung cancer, passive smoking could prevent.

By convention, we usually stay with 95 percent, EPA went to 90.

BY MR. McDERMOTT:

Q. So just to make sure that I understand your answer, Doctor.

The 90 percent confidence interval was a departure, correct, from the ordinary practice followed in the scientific and regulatory community?

A. I can't comment on the regulatory community.

The scientific community generally used 95 percent confidence intervals, but EPA offered its rationale for the use of the 90 percent.

Q. I understand. You also said, I think, they assumed that the direction would be toward risk or harm; correct?

A. If you will --

Q. Assuming that that was likely to be the answer?

A. Essentially this is equivalent to what we would call one tail, if you will, significant testing.

We're allowing consideration of hypotheses only in the direction that passive smoking causes lung cancer. Certainly a reasonable assumption in 1990.

Q. Isn't it true, Dr. Samet, that at the time the EPA made this assumption there were numerous studies that did show negative risks, negative correlations between exposure to environmental tobacco smoke and lung cancer?

A. There may have been some studies with point estimates below

1, but certainly on a biological basis no one would ever consider the possibility that inhaling carcinogens and second-hand smoke protects against the development of lung cancer.

Q. But the answer to my question is there were studies showing a negative association that were in the scientific record at the time the EPA made this decision; correct?

A. I think we've showed these studies, and again their confidence levels, of course, all went above 1.

THE COURT: Does the use, or did the use of the 90 percent confidence level inevitably skew the final results that EPA would come up with?

THE WITNESS: What is -- it would not have changed what we call the point estimates. So when the evidence is all summarized and they came up with that, I guess 1.19. It would not change the 1.19 at all. What it would change is the bound of uncertainty around that 1.19, making it narrower than with 90 percent, it would have been with 95 percent.

BY MR. McDERMOTT:

Q. Doctor, meta-analysis is a technique for summarizing disparate studies is controversial, isn't it?

A. I'm not so sure that -- I would certainly say under discussion, but certainly widely used throughout by medical research to summarize evidence and increasingly so.

Q. Let me show you Joint Defense Exhibit 067847. This is an

article authored by you entitled: Epidemiology and policy, the Pump Handle Meets the New Millennium published in the Epidemiologic Review, volume 22, at page 45, year 2000.

Let's focus on page 147 at the end of the first column. However -- down to the last sentence of the paragraph.

However, the application of meta-analysis to observational data remains controversial. And in the example the EPA's use of meta-analysis for the effects of environmental tobacco smoke, the methods application was set aside by the North Carolina Federal District Court.

You wrote that in 2000?

A. That's correct.

Q. It is a controversial technique, is it not, Dr. Samet, as you have yourself written?

A. Well, again, very even-handed, and I cited several of the individuals who have said there were problems with it. That said, while controversial, it's in widespread use for summarizing evidence.

Q. Can I see Joint Defense Exhibit 012190?

Dr. Samet, these are --

MS. EUBANKS: If we could wait a moment to get the exhibit.

MR. McDERMOTT: I'm sorry.

BY MR. McDERMOTT:

Q. For the record, these are notes of a meeting. It's

entitled: First Meeting of Authors, Involuntary Smoking and Health: A Report of the Surgeon General. It's dated April 9, 2001. Do you recognize this document, Dr. Samet?

A. I'm familiar with this document.

Q. This is a meeting of the authors of the next Surgeon General's report that's planned, right, the 2005 report?

A. That's correct.

Q. You are slated to be -- or you are designated as a senior scientific editor for this next report; is that correct?

A. That's correct.

Q. And this memo reflects one of the early meetings you had of the authors to organize for that report?

A. That is correct. Possibly the first.

Q. And you, in fact, chaired the meeting; correct?

A. That's correct.

Q. Let's focus on page 23, please.

According to this document, Dr. Samet, you told the meeting participants that the probability value for statistical significance associated with meta-analysis should not be a criteria for causality, correct?

Could you highlight the language there? It's on page 23. I'm sorry, page 26. My mistake.

Do you see that in the middle of the screen? The probability value for statistical significance associated with the meta-analysis should not be the criteria for causality?

Meta-analysis can be used to derive a risk estimate or an estimate of public health impact, but should not be central in decisionmaking.

That's what you told the participants; correct?

A. That's apparently a representation of what I said, yes.

Q. Thank you. You know who Dr. Ernst Wynder and Dr. Dietrich Hoffman are, don't you?

A. Yes, I'm familiar with them.

Q. Dr. Wynder is a very well known and -- he's actually a pioneer in the field of smoking health research, isn't he?

A. He was.

Q. He was. Rest his soul.

And Dr. Hoffman, he is also a well known widely-recognized expert in the field of smoking and health?

A. Correct.

Q. He's a chemist; correct?

A. Yes.

Q. He and his wife have written an awful lot about the chemistry of mainstream smoke, sidestream smoke and environmental tobacco smoke; correct?

A. They've certainly written extensively on mainstream and sidestream smoke, yes.

Q. Would you agree that Dr. Hoffman knows more about the composition of mainstream smoke than you do?

MS. EUBANKS: Objection, Your Honor. We've just --

we've not established proper foundation to put that question before this expert witness.

THE COURT: Objection is overruled. If the doctor can answer, he may.

A. Well, this is Dr. Hoffman's area of expertise. I'm not a tobacco smoke chemist.

Q. You would defer to him in matters of tobacco smoke chemistry; right?

A. In fact, the Hoffmans I believe are contributors to the 1986 Surgeon General's report.

Q. And he is an expert both in mainstream smoke and sidestream smoke and environmental tobacco smoke; is that fair?

A. Well, I think certainly his work focused largely on mainstream and sidestream smoke.

Q. Let me show you JD 000434. This is an article written by Dr. Ernst Wynder and Dietrich Hoffman. It's entitled: Smoking and Lung Cancer, Scientific Challenges and Opportunities. It was published in Cancer Research in 1994.

Can I see page 5291?

The limited reliability of data obtained by questionnaire -- we spoke of that earlier -- and the relatively limited number of nonsmokers with lung cancer who were not exposed to carcinogens in occupational settings point to the need for a prospective epidemiological study with exposure assessment by biomarkers -- and he describes them -- to bring

about a conclusive evaluation of the question on causality between involuntary smoking and lung cancer.

Do you see that?

A. Yes, I do.

Q. Was there a legitimate scientific basis in 1994 for the views expressed by Doctors Wynder and Hoffman? That there was limited reliability of data obtained by questionnaire?

A. Well, I'm not sure what I'm -- I know what they mean by reliability. That means repeatability. I think we've already commented on the issue of the misclassification that may occur with questionnaire use.

Q. Was there a legitimate scientific basis for the view expressed by these two gentlemen -- Dr. Wynder and Dr. Hoffman, in 1994 -- that there was limited reliability to the data obtained by questionnaire?

A. I'm sorry. Which pages? Maybe it would help if I could see the context in which this was -- this was page.

Q. Page 5291.

A. Well, this is helpful to look. The preceding paragraph covers issues related to smoking questionnaires and comparison to -- so I don't think they meant reliability. I think -- which is repeatability.

I think they meant validity which is the questionnaire measuring the truth, so they perhaps misspoke here. I've already addressed the issues of questionnaire

validity and the fact it's impossible if you will to perhaps --

THE COURT: Doctor, just a minute.

A. So I've already addressed the issue of validity of these questionnaires, that is their classification of smoke, and I think that's what they are speaking to here, and they provide in the preceding paragraphs some specific data.

And then they make a reference to nonsmokers who are not exposed to carcinogens and occupational settings. I'm not sure what that reference means.

Q. Was it legitimate in 1994 to express, as they did, did they have a legitimate scientific basis for expressing the view that there was a need for a prospective epidemiologic study with exposure assessment by biomarkers to bring about a conclusive evaluation, the question of causality between involuntary smoking and lung cancer?

A. Well, apparently, in their mines, but I think certainly by 1994 there had been a number of evidence, reviews that we've already talked about, that had reached the conclusion that passive smoking causes lung cancer.

Q. They were voicing a legitimate scientific view and perspective, were they not, Dr. Samet?

MS. EUBANKS: Objection, Your Honor. The question has been asked several times and the witness has responded.

THE COURT: I think it has. But can you give a yes or no to the answer?

THE WITNESS: Certainly Wynder and Hoffman were entitled to express their viewpoints.

Q. And it was scientifically legitimate, wasn't it? It was supported by a scientific basis?

MS. EUBANKS: Same objection.

A. Well, I mean, again, this is a, you know, less than one page review of a very complicated topic, so I'm not sure what leads them to their opinion. But again, they are certainly respected scientists and they are entitled to their opinion.

Q. And this was eight years after the Surgeon General's report in 1986; correct?

A. 1994 was eight years after '86.

Q. And eight years after the IARC report in '86?

A. Yes.

Q. And eight years after the NRC report in 1986?

A. That's correct.

Q. And two years after the EPA's risk assessment; correct?

A. That's correct.

Q. I want to ask you a few questions now about an article that was published in the British medical journal in 2003 by Doctors Enstron and Kabat.

Can I see JD 044908. It's entitled: Environmental Tobacco Smoke and Tobacco-related Mortality in a Prospective Study.

Dr. Samet, you testified in your written direct that

this study, quote, had a number of biases, close quote. Do you remember that?

A. Yes, I do.

Q. The biases you were talking about were statistical biases, were they not?

A. I'm not sure I know what you mean by a statistical bias.

Q. As opposed to a personal bias.

I'm talking about a scientific issue versus an integrity issue.

A. My discussions in my report were certainly scientific.

Q. That's all I wanted to establish.

And these statistical biases, these scientific issues that you raise, these concerns basically inhere in virtually all epidemiological studies relating to ETS, don't they, in one form or another?

A. Well --

MS. EUBANKS: Objection, Your Honor. The pronoun in what he means by "this" in all of the studies. We really don't have that testimony out here for the record to be clear.

THE COURT: Why don't you clarify it, please?

BY MR. McDERMOTT:

Q. The statistical biases that you were referring to your direct testimony are not unique to the Enstron and Kabat study; right?

A. I don't use the word statistic bias. There's

epidemiological bias or problems, if you will, just to use a real word.

And these effect studies to a greater or lesser extent, and in this study I think there's some particular problems and in other studies there are different sets of problems.

Q. And we talked about some of those potential problems during the course of this morning, haven't we?

A. Yes, we have.

Q. And some of those studies relate to questionnaires and the possible inaccuracy of exposure data; correct?

A. That's correct.

Q. Some of them relate to misclassification of smokers, the confusion of smokers and nonsmokers; correct?

A. That's another potential problem.

Q. Another potential problem is the misdiagnosis of cancer at another site as a primary lung cancer; correct?

A. That could be a problem, yes.

Q. Recall bias. That may be another aspect of the questionnaire, but recall bias is a potential problem, correct, or another way of saying it?

A. Depending on the context, yes.

Q. Now, the Enstron and Kabat study utilized the American Cancer Society 1 or so-called CPS-I data set; correct?

A. That's not fully correct. It utilized a segment of this study from California residents that Dr. Enstron had access to.

Q. But it drew a part on that data set; correct?
A. It drew on one component of the CPS-I data set. The cancer prevention study one of the --
Q. Maybe for the record, it would be helpful to explain what CPS-I and CPS-II are, and what these data sets are very briefly, Doctor?
A. If I can mention -- I mentioned before that cohort studies were started in the '50s. One that started in 1959 was by the American Cancer Society. They sent their volunteers door to door, asked people to complete questionnaires, and enrolled roughly a million Americans, and then followed them and looked at smoking and actually some other factors and risk for death from lung cancer and other diseases.

And then just to complete, later in 1980 they did the same thing with a whole brand-new group of a million Americans. So the first one is sometimes called CPS Cancer Prevention Study 1 and the second CPS-II.
Q. But my point here is that they were using existing data that's available broadly to the scientific community; right?

This was not original work they were doing, original field research; correct?
A. Well, they did some -- they used as a starting point the California component of the CPS-I. And then again, I'm certain that they did their own follow-up for the mortality of this group, because the American Cancer Society did not follow their

CPS-I beyond 1972, and this paper goes, as you can see in the title, through 1998.

Q. Let me just -- I want to move on in the interest of time, Doctor.

You say in your direct testimony, let me show you -- let me see page 184 of Dr. Samet's direct testimony, please, at the top of the page. Let's see the question from the previous page. Can you scroll down to the bottom?

Are you aware of any studies that have not found an association between passive smoking and lung cancer? And your answer appears on the following page.

Yes. From time to time, studies are published that do not find a statistically-significant association between second-hand smoke and exposure to lung cancer. We expect such studies to be reported as some are likely not to find significant results just by chance.

Can you pull up, Jamie, U.S. Exhibit 20599?

What you see on the screen, Dr. Samet, is a meta-analysis performed by Hackshaw, et al,. That was relied on in your direct testimony. Are you familiar with that study?

A. Yes, I am.

Q. And it is -- it's entitled: The accumulated evidence on lung concern and environmental tobacco smoke, and I'm having a real hard time seeing where it was published. I guess it's the British Medical Journal.

A. That's correct.
Q. You're familiar with that.
Let's look at --what's the page reference?
Can you blow that up, please?
Dr. Samet, this is Table 1 of that study. It's real hard for me to see. It's real hard for the court to see. It's probably pretty hard for you to see with a copy in your hand.
So what I have done is to literally transpose these results -- at least, I think I literally transposed these results -- into another exhibit which I'll show you now that I think will be easier for us all to read. J-DEM 060303.
I'm sorry, I want -- let's go ahead. I'm sorry, that's just the page out of the article that's bolded. And then let's go to J-DEM 060304. I hope you can see that a little bit better.
I'm going to break this down, Doctor, if I can, but you can see, according to the last row, there are 37 studies. 37 studies of women, eight studies of men; correct?
A. I think so.
Q. And that obviously contains all of the studies.
Let me focus first on the studies extracted that deal with men. Let's see 060305.
These are the studies with men, and I miscounted. I think that's a nine, not an eight. Again, that's hard to deal with.

So let me go ahead to 060306. This highlights these studies.

Is it fair to say -- and take a look at those, Dr. Samet -- that the only study reporting a statistically-significant elevated risk is the Hirayama study in 1984 and that the others do not show statistically-significant elevated risks?

A. That appears to be the case.

Q. Eight out of nine is a little bit more than from time to time, isn't it, Doctor?

From time to time we expect to see statistically-insignificant results.

A. I guess it's all the word. I used the phrase from time to time in my testimony.

Q. Five of the studies that the -- statistically significant results were done in the United States; correct?

A. I would need time to sort out exactly which study is which, which might be difficult considering how difficult it is to read this. That may be correct.

Q. All right. And three of the studies show either a negative overall relative risk or a relative risk of 1; correct?

The negative risk is 1984, no increased relative risk, Kabat 84 and Car-DAE-nas 1984. Did I interpret that correctly, Doctor?

A. I think the issue here is, if you look at the small numbers, the numbers of cases. I mean, for example, the Butler study

had, if I read right, 11 men cases and 10 -- controls -- of course, this is the problem with the studies of men. They were very small numbers in these studies, which is why they were, quote, not statistically significant.

Q. The Cardenas study isn't small, is it, Doctor?

A. Again, I think -- I can't quite -- I can't read the number of cases, actually.

Q. Is it 96,000, looks like. It looks like it's at least four times as large as the Hirayama study; isn't that right?

A. What's more in terms of significance is not only the number of people but the number of cases. The number of cases is still relatively limited. I think I read 97 or 87. I'm not sure.

Q. Let's look at the women now, Doctor. Let's go to J-DEM 060308. Again, this is the table from Hackshaw.

Can we see J-DEM 060309? These are the 39 studies that deal with women.

And you can check the math, but by my count, 30 of these 37 studies do not report statistically-significant excess risk overall. Is that fair?

A. That's what's presented here. Again I'm not going to go through each one of these studies to comment.

Q. It's just extracted from a table of an authority that you rely on, Doctor.

MS. EUBANKS: Objection, Your Honor. If we are going to ask this witness questions and to obtain his opinion, it's

really not appropriate for Mr. McDermott to make representations, unless he gives the witness an opportunity to answer that. The witness has answered the question about what's represented on the chart. They had full opportunity at discovery. This is not appropriate cross-examination.

THE COURT: Objection is overruled.

Go ahead, please.

BY MR. McDERMOTT:

Q. Thirty out of 37, that's just over 80 percent; correct?

A. That's possibly mathematically true.

Again, I think the point here is that most of the point estimates are above one.

Q. But over 80 percent, it's little bit more than from time to time, isn't it, Doctor?

A. I'm not going to get into the definition of time to time.

Q. Can you bring up 060310?

The six studies highlighted there showed negative overall relative risks; correct?

A. That's correct.

Q. I want to turn now to coronary heart disease for a few minutes. In your direct study -- direct testimony you discussed the Kawachi study. Do you recall that?

A. Yes, I do.

Q. The study -- that study was published in 1997, and is a study, an epidemiological study, of nurses in the United States;

correct?
A. That's correct.
Q. All right. Let's just see that for the record. U.S. 28521.
It's a study entitled: A Prospective Study of Passive Smoking and Coronary Heart Disease. And flip down to the bottom. Can you go to the bottom of the page, please?
I can't read where it's published. Good heavens. Doctor, maybe you can help me out there.
A. Circulation.
Q. Thank you very much. My eyes are getting older than I am or getting older faster.
K-a-w-a-c-h-i. Is that correct, Doctor?
A. Yes, it is.
Q. Now, you submitted a demonstrative to the court relating to -- let's just see that. It's a demonstrative to the court that related to the duration of nonsmokers adult wives spent living with adult smokers.
Let me see J-DEM 060221.
I think what we need is U.S -- I'm sorry. We need U.S. 17200.
Do you recognize this as one of the demonstratives attached to your direct testimony?
A. Yes, I do.
Q. Let me see the next demonstrative, 06292.
Let me explain what we've done here, Doctor. What

we've done is to try to -- what I'm going to do in the series of graphics is to take that information and turn it a little bit so we can see more clearly what the numbers mean and gauge the proportionality. All right. So this is just your demonstrative with some of the background information extracted.

Do you want to go back and take a look at a previous exhibit just to verify that this is what we're doing? All we've done is to take the graphic and move it down and isolate it for the moment. All right?

A. I'm sorry. Can I go back again?

Q. Certainly.

Can you put those side by side?

I'm sorry, we can't do that quite yet.

A. Okay.

Q. Let's flip to the next one, JD 060293.

All we've done there is expanded proportionally so that you can see with the 1.0 relative risk so that you, again in proportion, can see a little bit better the extent of the increased risk we're discussing. Do you follow me?

A. I do.

Q. This chart shows, does it not, that living with a smoker for 10 to 19 years produces a higher relative risk than living with a smoker 20 to 29 years; correct?

A. Well, the numbers higher. Again, there would be confidence intervals around this.

Q. Okay. And do you recall, Doctor, that the underlying study, the Kawachi study conducted a multivaried analysis to adjust for or control for, among other things, hypertension, diabetes, high cholesterol, vigorous exercise and saturated fat intake?
A. Yes, I do.
Q. And those factors that I just mentioned, high cholesterol, exercise, fat intake and the like, those are all risk factors that can contribute to the risks or the onset of coronary heart disease; correct?
A. That's correct.
Q. And so the purpose for making those adjustments in the multivaried analysis is to try to strip out the possible effect that those factors would have and to try to isolate on the impact of environmental tobacco smoke, is that fair?
A. Well, the general purpose is what you described, yes.
Q. Let's go to J-DEM 060295. What you see are the red bars we just saw. Those are the unadjusted relative risks. And the yellow bars or the gold bars are the adjusted risk. Correct?
A. Well, I haven't verified that from the tables. I assume that you've just used information in the tables in the paper.
Q. Okay. Again we see that the risk -- this isolates on ETS -- the risk of living with a smoker for 20 to 29 years is lower than the risk of living with a smoker for 10 to 19 years, and lower than living with a smoker for 1 to 9 years; correct?
MS. EUBANKS: Your Honor, my objection here probably

can be clarified by the witness.

When asked the question, he commented about an assumption that he was making, but I still don't know what assumption that this table is based upon so that the witness continues, and we make the record clear, whether the assumption that the witness has about what it is that he's answering is indeed what's intended.

MR. McDERMOTT: Your Honor, this chart depicts the information, the data from Table 4 of the aforementioned Kawachi study, U.S. 17, 2,000. It just represents that expansion of the information that Dr. Samet presented in his demonstrative.

THE COURT: How did you make the adjustment for risk factors? Where did that come from?

MR. McDERMOTT: These are the numbers out of Table 4 in the Kawachi study. The adjustment was made by Dr. Kawachi himself. Can we see Table 4 of that? Let's look at JD 003126.

MS. EUBANKS: Perhaps this could be cleared up if we had the Kawachi articles so that the witness can make that determination.

MR. McDERMOTT: We're going to do that right now.

BY MR. McDERMOTT:

Q. Can we see JD -- here is the Kawachi article.

Have you already given the witness a copy of it?

Would you like to turn to Table 4, Doctor?

A. I can't read a single number in the Table 4 in the copy I've

been given.
Q. Apparently it's the copy supplied by the government, so I can't answer for you. We will try to do something better at the break.
A. This is better.
Q. Do you see that? Okay. Do you see the numbers 1.19, 1.54, 1.11, 1.50?
A. Yes, I do.
Q. Those are the numbers that are transposed onto the graph that I showed you earlier. If you want to... 1.19, 1.54, 1.11, 1.50. Do you see that?
A. Yes, I do.
Q. So after adjustment for all of these potential risk factors or confounders, the Kawachi study shows that living with a smoker for 20 to 29 years produces a lower relative risk than living with a smoker for 10 to 19 or 1 to 9 years. Isn't that what the study shows?
A. The point estimate is lower for that particular stratum, yes.
THE COURT: I'm sorry. Would you repeat that?
THE WITNESS: Yeah. What I'm trying to say is that for that particular stratum technical work group, 20 to 29 years, 1.11 is the lowest value, which I think is the point that's trying to be made.
BY MR. McDERMOTT:

Q. And just so the record is clear. And perhaps to aid the understanding of some in the courtroom, perhaps myself included, why don't you explain briefly what a multivaried analysis is?
A. Okay. Again, epidemiologists use statistical tools which we call multivariable models or multivariable statistical models to try to take several things into account at once so that when we, let's say look at the effect of second-hand smoke or living with a smoker, it's not contaminated by the effects of any other factors.

The technical word we use of contamination is confounding. And so here what the authors have done and I think the comparison that's being made is simply to the unadjusted, where these potential confounding factors have not been taken into account, and that's the red bars, and then the orange bars are where they've used this statistical tool and tried to make certain that there is no residual effect of any of these other factors when they say, what is the risk of living with a smoker?
Q. Let's go back to U.S. 28521 to Table 4 at 2377 and let's try to blow that up.

It is correct, is it not, Doctor, that after adjusting for those potential confounders, none of the results were statistically significant?
A. You may be correct but I can't say that from this table.

MR. McDERMOTT: Can you get a clearer shot of that, Jamie?

THE COURT: It was clearer a few minutes ago.

MR. McDERMOTT: It sure was.

BY MR. McDERMOTT:

Q. After adjustment for the confounding, none of these results was statistically significant; correct?

A. That's correct within any particular group. And again what's happened now is that the full sample size has been broken down into these smaller groups.

Q. Another coronary heart disease study that you discuss in your direct testimony is a meta-analysis by H-e. Is it He or Hay?

A. Ha.

Q. Spelled H-e. And that was published in the New England Journal of Medicine in 1999; correct?

A. That's correct.

Q. Can we see U.S. 64090? Let's look at the first page, first sentence, background. The effect of -- can you just blow it up, please?

The effect of passive smoking on the risk of coronary heart disease is controversial.

And let's go down to the bottom.

Conclusion. Passive smoking is associated with a small increase in the risk of coronary heart disease. Given the high prevalence of cigarette smoking the public health consequences of passive smoking with regard to coronary heart disease may be

important.

The authors used the word "associated" not "caused"; correct?

A. That's correct.

Q. And they say "may," not "is" or "does" or "should be"; correct?

A. That's in regard to interpretation of their findings, correct.

Q. Correct. You also talk about a study published by LeVois or LeVois. Do you know how he pronounces his name?

A. LeVois.

Q. LeVois. L-e capital V-o-i-s. And Layard, L-a-y-a-r-d.

Can we see JD 047060? For the record, this is an article entitled: Publication Bias in the Environmental Tobacco Smoke slash Coronary Heart Disease Epidemiologic Literature, and it is published in Regulatory Toxicology and Pharmacology in 1999.

Now, you noted in your direct testimony that these gentlemen were funded by the tobacco industry; correct?

A. I think it was a 1995 paper. It states that it was supported in part by funding from Philip Morris USA.

Q. Let's look at the bottom. That's expressly acknowledged in the study; correct?

A. That's correct.

Q. And you testified in your direct -- and I can show it to you

if you like -- except for analysis of CPS-I and CPS-II presented by LeVois and Layard in 1995, all other studies have demonstrated at least a modest increase in risk for fatal and nonfatal CHD due to second-hand smoke exposure; correct? You testified about that?

A. It sounds familiar.

Q. Would you like to see your testimony? We can throw it up on screen if you would like.

THE COURT: What page of the testimony?

MR. McDERMOTT: Page 192, lines 4 through 23, and actually that, what I just read, is line 21 to 23. If you can highlight that.

Q. Do you see that testimony?

A. Yes, I do.

Q. You go on to compare their analysis of the data from CPS-I and CPS-II with Dr. Steenland's analysis of CPS-II; correct?

A. That's correct.

Q. Can you give the witness a copy of Dr. Steenland's article? That's JD 003037. And it's an article entitled: Environmental Tobacco Smoke and Coronary Heart Disease in the American Cancer Society CPS-II Cohort, and that's published in circulation in 1996; correct?

A. That's correct.

Q. This is the article you were referring to?

A. Correct.

Q. Now, LeVois and Layard were analyzing both CPS-I and CPS-II data, correct, and while Steenland was only analyzing CPS-II data.

A. Could I just have a moment to look at the --

Q. Absolutely.

A. Okay.

Q. Let's go back to JD 047060 at page 89.

In the LeVois and Layard article the authors break out their CPS-I and CPS-II data and report the combined data; correct? But they have a separate analysis for CPS-II reported; is that correct?

A. Give me a moment, please.

Q. Certainly.

A. There is a table -- I'm sorry, you're referring to Table 4?

Q. I'm referring to Table 4 in the middle where they break out separately CPS-II data.

In addition to dealing with the earlier study, they deal separately and report separately on just the analysis in CPS-II; correct?

A. Correct.

Q. And that's the body of information that Dr. Steenland deals with in his article, correct, just CPS 12?

A. He used CPS-II data, correct.

Q. Let me show you Joint Defense Exhibit 060297, demonstrative, rather.

What we've done here, Doctor -- and please feel free to compare the data with the original articles -- is we have simply extracted from the LeVois and Layard article their reported results on CPS-II and compared them for -- compared them with the report or results in the Steenland study analyzing -- and this is, I believe it's women. Do you see that?

Do you need to check the numbers or can we proceed?

A. Well, I possibly need to check the numbers. I'm just trying to understand -- the comparison in both instances is spouse never smoked. I'm looking -- it's 1.00 for Steenland. I'm trying to see whether ex-smokers -- it's a little confusing.

Q. I don't believe this analysis -- ex-smokers separately on the top line as you can see. I don't believe ex-smokers are mixed into the population group, the study population.

A. Could you refer me to a table in Steenland where I should look for this?

Q. Table 2. Doctor, I don't want to hurry you, but I don't want to make it too complicated either.

My point is really quite simple. I merely want to point out that the relative risks reported by LeVois and Layard are very close to those reported in Steenland; correct? Relative risks?

MS. EUBANKS: Your Honor, the witness has stated that he would like to be able to check. If we're going to get an answer that that question I'd rather have the testimony --

THE COURT: If he needs more time he may have it. It may be that the question is simple enough that he can answer it. So I'm going to leave that up to the doctor.

A. Well, I see where the numbers came from for the Steenland. This is in reference to women and it shows some of the data for some of the categories, all the categories of smoking.

The LeVois, Layard, which table did that come from?

Q. It's Table 4 and it's the segment in kind of the middle where CPS-II was broken out separately. Do you see that?

A. Yes, I do.

Q. And again, if we can go back to the -- my point is simple, Doctor. The reported relative risks are very close, correct, for ex-smokers and a confidence intervals very close?

A. They are similar. They were analysis of similar data.

Q. And the same is for smokers less than 20, or cigarette smoked by a spouse less than 20, confidence intervals?

A. Correct.

Q. There is a slight difference with respect to 20 to 39 because they reported a little bit differently. And then with respect to 40 plus smoking, LeVois and Layard report a higher relative risk; correct?

A. In that stratum, yes.

Q. You didn't mean to suggest in your direct testimony, did you, that the analysis of the data performed by LeVois and Layard was somehow wrong or corrupted simply because they

received funding from cigarette manufacturer, did you?
A. I don't think I made any statement in that regard.
Q. That is not your opinion, correct, that they were corrupted?
A. I have no basis for that opinion.
Q. Thank you. Let me ask you to turn to -- I want to talk now about the what I've sometimes heard referred to as the MRFITs study, the multiple risk factor intervention study by Svendsen. You talked about in your direct testimony, didn't you?
A. It's one of the references I relied on.
Q. Why don't we give you JD 003035. I think that's a copy of the study. Can we pull that up? 003035.
The summary of the study on the first page. That study reports the relative risk of coronary heart disease death for never smokers, married to smokers, is 2.11; correct?
A. That's correct.
Q. And the relative risk for never smokers married to smokers for fatal or nonfatal coronary heart disease for nonfatal coronary heart disease was 1.48, correct?
A. Correct.
Q. And neither one of those results is statistically significant; correct?
A. That P less than point 05, that's correct.
Q. Now, you note in your direct testimony that the study also combined former smokers along with never smokers as part of their analysis; correct?

A. They had the study originally included substantial numbers of smokers.

Q. If you look down at the bottom of the page, 790 -- let's see, footnote, where is that?

You see right here, includes both never smokers and ex-smokers who quit prior to entry into the trial; correct?

A. Correct.

Q. Now, former smokers as a group have a higher relative risk of CHD coronary heart disease than never smokers; correct?

A. Depends on how long it has been since they stopped smoking.

Q. Well, let's look at U.S. Exhibit 17141. This is a demonstrative attached to your direct testimony in which you show a decline in -- excuse me. We got the wrong -- we needed heart disease. We have the wrong exhibit number there.

MR. McDERMOTT: Excuse me, Your Honor. I'm sorry.

(Pause)

MR. McDERMOTT: It should be 17160.

Q. As you show there, Doctor, the risk falls off for years of cessation, but it remains elevated for a substantial period of time; correct?

A. This is a representative group of studies. Roughly the risk falls by about 50 percent of the first year after quitting, and I think, in general -- and I think we covered this in the 1990 report -- it's thought that roughly by 10 years after cessation it's followed back in every smoker, clearly some studies have

shown continued excess risk.
Q. Nevertheless, Doctor, when you add ex-smokers to the group of never smokers and measure the risk of heart disease, you would expect that the reported risk or the apparent risk for the combined groups would be higher than for never smokers only, wouldn't you?
A. Well, I guess within the MRFITs study that would again depend on who this particular group of former smokers were and how long it had been since they quit. Possibly yes.
Q. As a general proposition, when you add former smokers in, you would expect it to increase the apparent relative risk, wouldn't you?
A. I think this slide shows, it would depend on how long it's been for the group since they quit smoking. Perhaps if they all quit 20 years ago, the answer might be no, but if it's a mix of more recent and longer term quitters, the answer would be yes.
Q. That would be one's expectation as an abstract proposition; correct?
A. Well, I think based on the kind of evidence that's shown here, that would be a correct statement.
Q. Now, you testified with respect to the MRFITs study that when you added ex-smokers to the never smokers, the quote relative risks showed little change. Do you recall that testimony?
Let me show you. Page 195 at line 21.

These relative risks showed little change when the former smokers were included in the analysis or when the adjustment was made for other risk factors for coronary heart disease. Do you see that?

A. Yes, I do.

Q. That's not quite right, is it, Doctor? Let's take a look at JD 003035. I'd like to see page 783. The summary on the first page.

Let's focus on this. The smokers who quit prior to entry were included in the analysis. Relative risks for men whose wives smoked compared with men whose wives did not smoke for the above end points were 1.45 and 1.72, respectively.

Let's take a look at those results, Doctor, and I've compared them in a demonstrative, 060300. At least I hope I have.

All right. This was the original report or result 2.11. Do you recall that from the abstract on page 1?

A. Yes, I do.

Q. When we throw in former smokers, the relative risk drops substantially, doesn't it? It goes from 111 percent elevated risks to 48 percent relative risk, correct?

A. Well, the numbers speak for themselves. 2.11.

Q. And they drop rather than going down and they drop rather substantially, don't they?

A. It dropped from 2.11 to 1.48.

Q. And again, among women, the same thing. It dropped rather than increasing; correct?
A. That's correct.
Q. I want to go back to the demonstrative that you created, and it's regarding the British smoker study. Your chart is U.S. 17121.

Do you recognize this, Dr. Samet? This is your graphic depiction of the results of the 50-year follow up and the British doctors study?
A. Well, it's not what you said it is. It's a follow-up of the British doctors study at different points. 10 years, 20 years, 40 years and 50 years.
Q. I accept that correction, Doctor. Thank you. This is your demonstrative.

Now what I've done is to take your figure, and let's see JD 060301. What we've done is just carry forward the graphic depiction of the results you just had with the MRFITs results, and as you can see, according to MRFIT, exposure to environmental tobacco smoke is -- imposes greater risks than smoking 25 cigarettes a day for 50 years; correct?
A. This is a little bit of an apples and/orange comparison. Again, just to make clear, that British physicians studies the relative risk comparison is smokers in these different groups to nonsmokers, and the 2.11 is a comparison to people passively exposed to not passively exposed and the comparison numbers is

there.
Q. According to MRFIT, the people in that study would have done better to move out of their houses, take up smoking for 20, 40 or 50 years that would have lowered their risk of coronary heart disease; right?
A. I can't --
Q. If we are to credit the results of that study.
A. I can't answer that question.
Q. Isn't that the logical implication of the results reported, Doctor?
A. That's your logic in answering that question, not mine.
Q. You would agree with me that it would be an entirely anomalous result for exposure to environmental tobacco smoke to produce a greater relative risk of coronary heart disease than smoking more than 25 cigarettes a day for 50 years, wouldn't you? That doesn't sound right, does it?
A. I think what's not being shown is the 2.11 is the confidence interval around the estimate. This is one estimate from a single study, and its value 2.11 is what was estimated by the investigators.
Q. I understand. Let's move on.
THE COURT: I think actually we will probably take a lunch break at this point. It's a good time, Mr. McDermott.
Dr. Samet, you may step down.
How much longer do you think you're going to be this

afternoon?

MR. McDERMOTT: Maybe a couple of hours, Your Honor. I'm not sure. I'll try to pare it back.

THE COURT: Are there going to be -- is there going to be other questioning from other defense counsel?

MR. BERNICK: Not at this time.

MS. SCHWARZSCHILD: No.

THE COURT: 2:00 o'clock everyone. Did you have a question? You will have a few questions, okay.

2:00 o'clock everyone.

(Morning proceedings concluded at 12:40 p.m.)

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CERTIFICATE

I, EDWARD N. HAWKINS, Official Court Reporter, certify that the foregoing pages are a correct transcript from the record of proceedings in the above-entitled matter.

Edward N. Hawkins, RMR

UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF COLUMBIA

UNITED STATES OF AMERICA,	.	
	.	
Plaintiff,	.	Docket No. CA CA99-02496
	.	
v.	.	
	.	
PHILIP MORRIS USA, et al.,	.	Washington, D.C.
	.	September 29, 2004
	.	
Defendants.	.	
. . . . .	.	

VOLUME 6
AFTERNOON SESSION
TRANSCRIPT OF BENCH TRIAL PROCEEDINGS
BEFORE THE HONORABLE GLADYS KESSLER,
UNITED STATES DISTRICT JUDGE

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P R O C E E D I N G S

(2:02 p.m.)

THE COURT: Mr. McDermott, please, you are going to finish this afternoon, aren't you or do you think not?

MR. McDERMOTT: I certainly hope so, Your Honor, I'm planning on it. If the Court would like, we would take up the matter of the backdrop for the chart that I -- or the exhibit that I showed the witness earlier, if I may approach.

THE COURT: All right, that's fine.

CONTINUED CROSS-EXAMINATION OF JONATHAN M. SAMET, M.D.

BY MR. McDERMOTT:

Q. Dr. Samet, let me show you what I have here. These are the numbers in the charts, these were the studies referenced, eight being yours and the five in the Surgeon General's Report, and the studies are listed here. The 13 comes from the --

THE COURT: Mr. McDermott, don't forget our court reporter is trying to take it down.

MR. McDERMOTT: Thank you. I'm sorry, Your Honor.

BY MR. McDERMOTT:

Q. And the final set of numbers right there. So, if you would like to make that comparison and satisfy yourself we've transposed the numbers correctly or translated them.

A. I don't think I want to review them all now. I trust that's been done correctly.

Q. All right. Thank you.

We spoke briefly, earlier, of the Dr. He, study, H-E, and that appeared in the New England Journal of Medicine.

A. Yes, I do.

THE COURT: And "He" appears where?

MR. McDERMOTT: That is a very good question, Your Honor.

THE COURT: It's in 180s, 190s. I think it's 194. Mr. McDermott, am I right, you're talking about the study referenced at the top of page 194.

MR. McDERMOTT: Yes, Your Honor. Yes, Your Honor, I'm sorry.

BY MR. McDERMOTT:

Q. In that same volume of the journal, New England Journal of Medicine, a Dr. John Baylar wrote a letter which I'll have handed to you right now, it's JD 002898.

And Dr. Baylar --

MS. EUBANKS: Excuse me, if I could have a copy.

BY MR. McDERMOTT:

Q. This is a -- Dr. Baylar is a professor emeritus at the University of Chicago.

THE COURT: Now wait a minute. Does Dr. Samet have this letter? It's not up on the screen.

THE WITNESS: And if I could make a correction, it's not a letter, it's an editorial. It was not a letter, but it was an editorial commentary prepared by Dr. Baylar on the article published in the New England Journal.

BY MR. McDERMOTT:
Q. I'm sorry, I thought it was a letter to the editor. I will accept your interpretation.
In that, if we can focus on the language, "I regretfully conclude -- "
MR. McDERMOTT: Maybe we shouldn't have taken a lunch break, Your Honor. I'm sorry.
THE COURT: Well, maybe I could have taken a few more steps in the open air.
MR. McDERMOTT: At least there isn't rain.
BY MR. McDERMOTT:
Q. "I regretfully conclude that we still do not know with accuracy how much, or even whether, exposure to environmental tobacco smoke increases the risk of coronary heart disease."
Now, my question to you, Dr. Samet, is that a scientifically legitimate viewpoint to express given the state of the science?
A. I just have to say I was quite disappointed when I saw that John Baylar had published this commentary, because he does not like met analysis and that's what he talks about in the editorial. He really does not go into the heart disease risk except to say he doesn't like the way He and colleagues did their analysis, and reached that conclusion. But he cites in his three references none of the many other people who have commented on this. He does not comment on any of the other

expert reports, and his background is not really in this area, so he's a respected is scientist, and this is his opinion, but in this commentary, it is basically all on the weaknesses of metanalysis.

Q. But it is a scientifically legitimate opinion to express, is it not?

A. It's an opinion. Again, all I can say is that in the page and a half of commentary here, it's a critique of met analysis of observational studies and he really does not go into the plausibility of the association, the experimental studies of passive smoking, you know, the way one would if you were addressing the whole topic. And in a way I thought my comment about this was that he was shooting the message because of the messenger, he just simply did not like metanalysis.

Q. And he certainly is not a fan of tobacco to your knowledge, is he?

MS. EUBANKS: Objection, Your Honor. Characterization. What does the questioner mean by "fan"?

THE COURT: If you would ask the question I might sustain the objection, but since the objection comes from the defendants' counsel I'm going to overrule it, it's their characterization.

THE WITNESS: I've not addressed this issue with Dr. Baylar.

BY MR. McDERMOTT:

Q. You certainly have no knowledge of the effect that he

has, do you?
A. I'm sorry?
Q. You have no information that he is a partisan of tobacco?
A. I don't have information one way or the other.
MR. McDERMOTT: Can we see JD 024502?
THE COURT: 005?
MR. McDERMOTT: 024502.
BY MR. McDERMOTT:
Q. Let's focus on page 505. What would appear on the screen, I hope, is a comment from the editor of the British Medical Journal, Dr. Richard Smith, that appeared in August of last year. You've read it before?
A. Yes, I have.
Q. Okay. And the British Medical Journal is one of the most prestigious peer review journals in the world, is it not?
A. It's a well respected journal.
THE COURT: And could we have the date on this, please?
MR. McDERMOTT: I'm not sure I know more than August -- August 30th, I'm sorry. August 30th, 2003.
BY MR. McDERMOTT:
Q. And in his comment he says, "We must be interested in whether passive smoking kills, and the question has not been definitively answered. It's a hard question and our methods are inadequate."
Dr. Samet, there was a legitimate scientific basis for

the British Medical Journal's editor to make that statement, was there not?

A. I have simply no idea why Dr. Smith made this two-sentence statement. I mean, it's a comment from the editor. I don't know Dr. Submit's background, and I don't -- I was quite surprised, actually, to see, again, this statement. And I don't know why Dr. Smith made it. He doesn't give the scientific basis, he says the question has not been definitively answered, but gives no, no commentary as to why he reached that conclusion.

Q. There's an extensive article on environmental tobacco smoke and its link to disease in nonsmokers that appears in that volume of the journal; isn't that correct?

A. Are you referring to the --

Q. Excuse me. I'm sorry.

It was the edition before that. The Enstrom, E-N-S-T-R-O-M, and Cabot article about which we spoke earlier. Do you recall that? Do you recall the article by Enstrom --

A. Of course we do.

Q. -- that we spoke of. And wasn't this a comment in response to a flood of criticism that came into the British Medical Journal?

A. Dr. Smith was commenting here on the articles that came into the British Medical Journal.

Again, why, in 2003 with so much evidence and so many

accumulated reports Dr. Smith raises questions about the other -- I can't speak to.

Q. Okay. Dr. Samet, would you agree that it is legitimate for Dr. Smith to voice his views?

A. I agree that Dr. Smith can voice his views. I think if he's raising questions, it would be useful if he supplied the basis for those views, which he did not do here.

Q. You wouldn't say his view should be silenced?

A. Dr. Smith is the editor of the journal.

Q. I want to move on to another topic, Dr. Samet, and I think we can be fairly brief on this.

I want to talk a little bit about your involvement in the model that has been developed in this case by Doctors Wyant and Zeger that estimates health care costs that they say are attributable to smoking.

Am I correct that your personal role in the development of this model, the design of this model, was very limited?

A. That's correct.

Q. And is it also the case that with respect to this model, even though you did not personally review all of the specifics, you thought and said it was important that the model address diseases that are actually caused by smoking?

A. Well, I said that the model suggests both those diseases that are caused by smoking and the consequences of the reduced health of smokers compared to nonsmokers.

Q. Okay. But you want to be certain, did you not, that they were identifying persons as having the disease who really had it and try to avoid false positive identifications of persons having a smoking caused disease?
A. I had discussed with them the approach to using a diagnostic information to try and avoid false positive diagnoses.
Q. And one of the suggestions you made was that they use age 40 as a cutoff because smoking related diseases only appear very rarely in persons under the age of 40, correct, at least that are attributable to smoking?
A. The major specific smoking related diseases are relatively uncommon before age 40, correct.
Q. Okay. And so, for example, if a person had heart disease at 40, and had a congenital defect condition, it would be inappropriate to include that person in a model that attributed that person's healthcare costs to smoking; is that correct?
A. Well, congenital heart defects, to my knowledge, have not been linked to smoking, not to say active smoking does not influence degenerative heart defects.
Q. But your recommendation is that you wanted people who were diagnosed with a condition over 40, correct?
A. Well, this is a conservative assumption to avoid false positive classifications.
Q. And you urged that assumption on them, correct?

A. I made that recommendation.
Q. I want to move on to another area very quickly, low tar, but let me just --
MR. McDERMOTT: Can I see the ELMO real quickly.
BY MR. McDERMOTT:
Q. I want to clarify one thing. I think I'm correct, but let me direct your attention to this bit of testimony which appears on 161 of your report, lines 21 through 23. I think you may have that backwards. Did you mean to say that studies found a 20 percent lower risk of disease in smokers of high yield cigarettes?
A. Your sharp eyes caught something that should be reversed, yes.
Q. Okay. That's just reversed. All right. Thank you.
You spoke very briefly in your direct testimony about the measurement of tar and nicotine by the FTC method, and in fact, you testified in your written direct that the FTC test method is not informative with respect to lung cancer risk or to the risks of smoking caused diseases generally. Do you recall that testimony?
A. Yes, I do.
Q. Okay. You understand, don't you, that the FTC method was not intended to measure the yield of tar and nicotine that the average smoker gets, do you not?
A. It's a measurement -- excuse me -- made by a machine of

yield under the specified circumstances. I understand that.
Q. And it was not intended to mimic human smoking as such, correct?
A. Well the original data on which the machine patterns of puffing are based were of human observations, so at least as far as how the machine smokes the cigarette, the pattern of puffing and so on was based on, I think, early observations.
Q. It was not intended to mimic every person smoking, or to represent an average person smoking, was it doctor?
A. Not to my knowledge.
THE COURT: Well, then, what was it? What, to your knowledge now, was it intended or designed to do?
MR. McDERMOTT: Your Honor, perhaps I can clarify if we can put up the next exhibit.
THE COURT: All right.
MR. McDERMOTT: JD 040254.
BY MR. McDERMOTT:
Q. And let's, at page 2. "In determining the testing method the commission is not attempting to gauge the test to the amount of smoke to tar and nicotine which the average smoker will draw from any particular cigarette." And then on to the next -- excuse me, second to the last paragraph on that same page.
"Thus, to reiterate the uniform method determined by the commission has, as its purpose, measurement of the tar and nicotine generated by cigarettes when smoked according to that

procedure."

Just a standardized test, Your Honor, to permit inter brand comparisons. Do you agree with that Dr. Samet?

A. Well, certainly the information has been attractive over time as tar and nicotine yields have changed. So it would be, I think, used for inter brand comparisons. Results have been reported on how these yields have changed over time as well.

Q. But it was a standardized test to permit comparisons. The EPA gas mileage for example is not a guarantee that you are going to get the number on the sticker, right, but, if you get a Volkswagen and compare it to a Hummer, you know, no matter how you drive the Volkswagen you're probably going to do a little bit better than if you drive the Hummer the same way, correct?

MS. EUBANKS: Objection, Your Honor. This is certainly beyond the scope of this witness's area of expertise, and it's also beyond the scope of his direct testimony. He's not here as an expert on the FTC method, he did have some passing testimony about it, but he's not a competent witness to testify to these matters.

THE COURT: Well, I think that's probably right, and I'm the one who started asking him the more detailed questions on that, so as to understand his understanding of the FTC method. We're obviously going to have people in this trial who are much closer to that issue than Dr. Samet.

BY MR. McDERMOTT:

Q. Let me just ask you this, Dr. Samet: Would you agree, the real question with respect to the FTC method is not whether it reflects the average smoker's intake, but rather the cigarettes with lower FTC yields, on average, actually deliver less tar to smokers than cigarettes with higher FTC yields?
A. Could you repeat that one more time?
Q. I'll actually -- maybe the court reporter can repeat it or we might not get it quite right.
THE COURT REPORTER: "Let me just ask you this, Dr. Samet: Would you agree, the real question with respect to the FTC method is not whether it reflects the average smoker's intake, but rather the cigarettes with lower FTC yields, on average, actually deliver less tar to smokers than cigarettes with higher FTC yields?"
THE WITNESS: I'm not sure -- I want to hear it again because I'm not sure I know what the real question is by what is meant by that. This is one of the questions that might be raised about interpretation of the FTC machine yields, how does it relate to what actually gets into a smoker.
MS. EUBANKS: Your Honor, I would object to this question, particularly in light of this expert witness's exam -- statement just now about his understanding or lack of understanding.
THE COURT: Sustained.
BY MR. McDERMOTT:
Q. In your direct testimony, Doctor, you refer to an article

written by Dr. -- I may not get this right, Djordjevic, D-J-O-R-D-J-E-V-I-C, that appears in the journal of the National Cancer Institute in 2000. Let's take a look at that, JD 003743.

THE COURT: And where is that discussed, please, in the direct?

MR. McDERMOTT: It's in his direct testimony at 147, line 22 over to line 13 on the following page, Your Honor.

BY MR. McDERMOTT:

Q. And you cite that for the proposition that smokers actually receive more tar and nicotine than the FTC numbers indicate, correct?

A. I think, as the article says, it's a proportionate assumption, but they would not receive proportionately to what is measured by the machine what actually goes into people, correct.

Q. Okay. But it also contains information, does it not, on whether the smokers of low-yield cigarettes do get less tar and nicotine than the smokers of medium tar cigarettes, right? It makes a direct comparison between those two?

A. Let me just look at the article.

Q. Certainly. Let me put up on the screen table 3 of page 109, and maybe that will assist you. I'm sorry -- okay.

I'm sorry, I have the wrong table up there. Let me turn to table 1, page 108. That's where we are.

You see there, Doctor, the highlighted line? If you look

on the screen. Low-yield cigarettes, the number of cigarettes per day on average, 14.9 versus medium-yield, 15.9. Do you see that?

A. Yeah, but let me clarify, but again, I think this table refers to what the individuals were smoking and then they were assigned in this study to smoke low-yield or medium-yield, if I remember correctly. So this was a number of cigarettes they were smoking under their usual smoking circumstances, and then they came in and were given cigarettes, I think, -- there may have been smokers of low-yield or medium-yield, and then the measurements were made with experimental cigarettes, if I recall.

Q. All right. Let's go to table 3, and we may be able to clarify this. During the course of the test were they assigning people different cigarettes, the results are displayed on the screen with respect to tar, milligrams per day. You'll see the low-yield cigarettes delivered an average daily amount of 312 milligrams, and the medium-yield cigarettes delivered 571 milligrams, correct, Doctor?

A. Just let me just spend a minute.

Q. Certainly.

A. This is -- I think again, I'm trying to reconstruct this relatively quickly here, but I think these are the author's estimates of how much tar in milligrams per day. And I think this is --

Q. Doctor, let me draw your attention to the tar category in the middle line. It says "measured" -- do you that?
A. Yes, I do.
Q. That's contrasted with the top line that says "rating", so that the measured amount is reported?
A. Right.
Q. Let me direct your attention to the line dealing with nicotine, again the middle line. 27.3 milligrams daily for the low-yield cigarettes, 42.6 for the medium-yield cigarettes; is that correct?
A. Yes. Let me have another minute, please.
Q. Certainly.
THE COURT: Now, is this chart from the National Cancer Journal? Is this what you started out referring to?
MR. McDERMOTT: Yes, ma'am.
THE COURT: Okay.
BY MR. McDERMOTT:
Q. While the Doctor is reading, for the record, it's volume 92, number 2, January 19th, 2000, Journal of the National Cancer Institute.
A. Okay.
Q. All right. Let me direct your attention down to the bottom now, carbon monoxide, again same pattern, measured amounts, 242 milligrams per day for the low tar versus 483 for the medium tar, correct?

A. Yes, I see those numbers.
Q. All right. Let's go to the next page, table 4. There's also a report on some of the carcinogens with respect to benzopyrene. You'll see he observed delivery, 17.9 anigrams in the low tar yield, low-yield category versus 21.4 for the medium-yield, and again, with NNK, 186.5 nanograms per cigarette on a daily basis for the low-yield category versus 250.9 for the medium-yield category, correct?
A. Correct.
Q. Let's go back to the first page of that article to the abstract.
In fact, the authors report, do they not, that smokers of --
MR. McDERMOTT: Can we call that up? It's right -- let me see, right here. Can you highlight that?
BY MR. McDERMOTT:
Q. "Smokers of medium-yield cigarettes compared with smokers of low-yield cigarettes received higher doses of all components."
So this gives us a lot of information suggesting that on average smokers of medium-yield cigarettes do get more than smokers of low-yield cigarettes by comparison, receive less of important constituents and components of smoke; isn't that right, Doctor?
A. This is one conclusion of the authors, the others being,

of course, that the reductions were not proportional to the machine measured yields.

Q. I understand that, but still statistically significant, and quite conceivably biologically significant, correct?

A. Well, biological significance I can't comment on it.

Q. All right. Let's move on.

You also discussed a study by Coultas and colleagues from 1988, correct?

A. That's correct.

Q. I'm going to ask my assistant to hand you a package of articles from 1988. We could not find the reference that you apparently had in mind, and maybe you can find it in those articles.

You say you discussed this at your transcript 147, line 22, and over on to the next page, and you cited for the proposition, and I quote, "After taking account of numbers of cigarettes smoked, the levels of biomarkers were not associated with the yield of tar and nicotine of the current brand that was being smoked." That's what you cite in this article?

A. That's correct, and to help, there should be an article in the American Review of Respiratory Disease.

Q. Well, there are a bunch of articles. One, two, three, four, five articles in that journal that were authored by Dr. Coultas that year, and we have got those for you, and we couldn't find the reference. And I don't want to make a mystery

of this Doctor, I think I found what you are referring to, but I want to give you an opportunity to --

MS. EUBANKS: Your Honor --

BY MR. McDERMOTT:

Q. -- to set this straight, if you would like.

MS. EUBANKS: Your Honor, if Mr. McDermott has the material that he thinks is being referenced, it seems to me it would expedite matters if he would show that to the witness instead of a stack of articles for the witness to go through.

BY MR. McDERMOTT:

Q. I just don't want to misconstrue your testimony, Doctor. I think what you're referring to is what I'll put up on the screen now, JD 060632, an article published by Coultas in 1993 entitled Cigarette Yields Of Tar And Nicotine And Markers Of Exposure To Tobacco Smoke published in the American Review of Respiratory Diseases volume 148 in 1993. I think this is the article that you were referring to but I may be wrong.

A. It is, and there must have been a misstated reference there.

Q. Okay. Well, so, this is the article that you meant to refer to. And you're listed as a coauthor, correct?

MS. EUBANKS: Before we go forward, could I have a copy, too, please?

MR. McDERMOTT: I'm sorry, I thought you -- Coultas -- let's turn to page 439 to table 3.

BY MR. McDERMOTT:
Q. And let me just start with a general question. It's true, Dr. Samet, that salivary cotinine is a biomarker for nicotine, correct?
A. That's correct.
Q. Okay. And isn't it true that these data show that both FTC tar and FTC nicotine are statistically significant predictors of salivary cotinine?
A. I think my comment about variability was in reference to the r-squared values at the bottom, which is the measure of variability.
Q. Okay.
A. And again, the -- if there is a perfect explanation value that is 100, the comment had to do with the degree to which the r-squared value increased with consideration of either the FTC tar or FTC nicotine yield in these statistical models.
Q. Okay. Let me see if I can get you to answer my question, because I'm not quite sure you did.
Isn't it true that these data show that both the FTC tar yield and the FTC nicotine yield are statistically significant predictors of salivary cotinine?
A. That's what's shown here. And my comment, again, was to the variability which is the r-square at the bottom, value at the bottom.
Q. All right. Now, one thing I noticed, you ran an analysis

for tar separately and you ran an analysis for nicotine separately. Why did you run those analyses separately?
A. Because these were quite correlated, they would not be appropriate statistically to put them both in the same statistical equation or model.
Q. Okay. So you shouldn't run them both together, in a statistical sense it would be a mistake to do that; is that right?
A. It might have been -- I can't remember the exact correlation here, but that's the reason that this was done.
Q. Okay. Because they're highly correlated?
A. That's correct.
Q. What would you expect to happen if you had run the analysis and put those two together?
MS. EUBANKS: Objection, Your Honor.
BY MR. McDERMOTT:
Q. Or can you say?
MS. EUBANKS: That -- I know this is an expert witness, but that calls for complete speculation that he's not really competent to answer. If he had put those thing together and then run an analysis statistically what he would have found.
MR. McDERMOTT: Your Honor, I'm not sure he can't -- I may he may know the answer, he may not.
THE COURT: If the witness knows it he may answer it. The objection is overruled.

THE WITNESS: I'll just say if one puts highly correlated data into models they become very difficult to interpret.
BY MR. McDERMOTT:
Q. Unstable or not reliable?
A. Well, the correlations are high, "unstable" is the wrong word. They just become difficult to interpret depending on patterns in the data.
Q. All right. Thank you. Let me move on to another issue. Vent blocking.
You referred at page 148 of your transcript to the phenomena of vent blocking smokers blocking the holes in filter cigarettes, and you note -- you observe there that these holes are, "generally covered by smokers". Do you recall that testimony?
A. Yes, I do.
Q. Okay. Actually, Dr. Samet, that's only true for smokers of the very lowest tar category cigarettes, isn't it, with yields in the range of 1 milligram of tar?
A. I'm not sure I can answer that question. I don't have a specific study in mind.
MR. McDERMOTT: Let's see US Exhibit 58770. Can you show page 28?
BY MR. McDERMOTT:
Q. Let me direct your attention to the bottom of the page. "Reviewing the literature" -- excuse me -- "vent blocking

appears to be a significant mode of compensation for reduced yield among smokers of lowest tar cigarettes, e.g. 1 milligram FTC tar, but not likely among most smokers of light and ultra light cigarette brands."

So, according to Monograph 28, vent blocking is not likely for most smokers of filter tip cigarettes, correct?

A. That's the statement here. I haven't seen table 2.3 to look at the basis.

MS. EUBANKS: Excuse me, but you said "Monograph 28", I just want to make sure that's what you intended.

MR. McDERMOTT: Excuse me, Monograph 13. My mistake, my Freudian slip is showing once again.

BY MR. McDERMOTT:

Q. Doctor, you understand even though you're not an expert in the FTC method as such, that the FTC is and has been aware of the possibility of vent blocking for years, correct?

MS. EUBANKS: Objection, Your Honor. There's no evidence in the record to suggest that this witness would be competent to offer testimony about the FTC's belief or knowledge with respect to these matters.

THE COURT: Overruled. The witness may be able to answer.

THE WITNESS: I can't answer that question.

BY MR. McDERMOTT:

Q. You attended the proceedings that resulted in the issuance of monograph 7, correct?

A. I attended part of those proceedings.
Q. And you do not recall the issue of vent blocking being discussed during the course of those proceedings, you weren't there?
A. I was there briefly for that meeting, and frankly, I don't remember whether I was there for that piece of discussion or not.
Q. Okay. You simply don't know?
A. Whether I was there or --
Q. Whether it was discussed during those proceedings or not?
A. I'm aware that the meeting was on potential limitations of the FTC method, but I don't recall if I was there for that particular discussion or not.
Q. And you don't recall whether possible vent blocking was one of the issues, or one of the concerns, that gave rise to that notion of revisiting whether this was the right test?
A. Well this is -- certainly has been a control about the FTC method because the posts, the vent holes in the filter, of course, are not blocked by the machine.
Q. Okay. And despite this and other criticisms with respect to the FTC method, you are also aware that as of today the FTC has not chosen to change the method that it requires for cigarettes sold in the United States, correct?
A. I'm aware that the same methodology is used.
Q. Doctor, let me talk to you about some of the epidemiology

studies that relate to the relative risks of low tar cigarettes. But let me start by asking you whether you agree that epidemiologic research has had a central role in characterizing the consequences of the changing cigarette?

A. Sounds suspiciously like something I wrote, so I think I will agree with it.

Q. Correct. And that statement is as true today as when you wrote it in 1996, isn't it Dr. Samet?

A. Well, it's as true today as then, yes.

Q. Okay. And isn't it -- and isn't it also true that epidemiology supplies direct information on the consequences of varying tar and nicotine yield products?

A. Also sounding familiar, and I will agree.

Q. Okay. And you agree with that today, correct?

A. Correct.

Q. And isn't it also true that only epidemiologic studies can provide information on modification of the risks of smoking as the cigarette has evolved?

A. I think you're reading from something I wrote from monograph 7.

Q. That is correct, and you agree with that today?

A. Yes.

Q. And isn't it also true that only epidemiologic data can measure the risk of cigarettes under "natural" circumstances of use?

A. Correct.
Q. And that is true today?
A. Yes.
Q. Would you agree that the clear impression from the epidemiological studies taken as a whole is that there is a lower risk of lung cancer among populations of smokers who use lower yield products?
A. I think that's a question that has to be addressed in different contexts. I mean, unfortunately for this issue, we're always looking backwards, so if we have done an epidemiological study, we're looking at the consequences of cigarettes used in the past. There's evidence that shows that users of filter, compared to nonfilter, cigarettes had lower lung cancer risks.
Q. And isn't it fair to say that the clear impression from these studies, taken as a whole, is that there is a lower risk of lung cancer among populations of smokers who use lower yield products?
A. Again, I mean, this all has to be put within a time context, because today we only have information from the past, and in terms of risks of recently used products or products in use now we have to wait. These kinds of comments are made in reference to a body of epidemiological studies that looked at people who smoked during the '40s, '50s, '60s and moved from higher yield nonfilter products to filter products.
Q. I understand.

MR. McDERMOTT: Let's see U.S. 587000. This is Monograph 13. Can you go to page 81 at the bottom of the page.
BY MR. McDERMOTT:
Q. Do you see that highlighted language? "The clear impression from these studies taken as a whole is that there is a lower risk of lung cancer among populations of smokers who use lower yield products."
That's a fair assessment of --
A. And table one refers largely to these studies I mentioned, studies published in the '60s, '70s and '80s.
Q. Thank you. And it's also true, is it not, that studies published in the epidemiological literature supported difference in lung cancer and possibly heart disease risks between populations of individuals who smoke filtered or lower yield cigarettes?
A. I'm sorry, I'm not sure I understand the question.
Q. Okay. Let's go to page 108 in the middle of the page. "Studies published in the epidemiological literature support a difference in lung cancer and possibly heart disease risks, but not in chronic lung disease risks, between populations of individuals who smoke filtered or lower yield cigarettes compared to individuals who smoke unfiltered or higher yield cigarettes."
That's the summary of the epidemiological evidence that appears in Monograph 13, is it not?

A. Apparently, and again, I think the lung cancer statement is consistent with the one that was just read. The heart disease information is somewhat mixed, and then no evidence of reduced -- correct me if I'm wrong -- lung disease risk is shown here.

Q. All right. And it's also true, is it not, Dr. Samet, that the epidemiological observation of lower risks with use of filtered and lower tar cigarettes has been reproduced in multiple populations and cannot be dismissed as an artifact of a single analysis or a single population?

A. Could I hear that one more time?

Q. Sure.

MR. McDERMOTT: Why don't we put up -- go to page 7, please.

THE COURT: Is this all from Monograph 13?

MR. McDERMOTT: Yes, Your Honor.

Middle of the page.

BY MR. McDERMOTT:

Q. "Epidemiological observation of lower risks with the use of filtered and lower tar cigarettes has been reproduced in multiple populations and cannot be dismissed as an artifact of a single analysis or a single population."

Do you agree with that.

A. Again I think this is in reference to the studies summarizing in 401, and there were multiple populations that

were studied, yes.
Q. Okay. And I want to step back a little bit in time, Dr. Samet, to 1996, when you were participating and then wrote an article with respect to monograph 7.
You personally reviewed at that time the prospective and retrospective of epidemiological studies concerning lung cancer and lower tar for that proceeding, didn't you?
A. I reviewed a number of studies that looked at lung cancer risk in relationship to type of product smoked.
Q. Okay. And you concluded, did you not, based on the CPS-I data that, "For all causes of mortality, and for lung cancer lung cancer mortality, the standardized mortality ratios declined as estimated tar and nicotine intake declined." Correct?
A. That's a description of the results of --
Q. Okay?
A. -- CPS-I.
Q. Okay and that represented a state-of-the-art interpretation of those studies at that time period, didn't it Dr. Samet?
A. This is my own interpretation.
Q. We'll credit you with that.
And you note in your publication, you actually quote the 1981 Surgeon General's Report.
MR. McDERMOTT: If we can put up monograph 7, which is J E

045979, at page 86.
BY MR. McDERMOTT:
Q. "Today's filter tip lower tar nicotine cigarettes produce lower rates of lung cancer than do their higher tar and nicotine predecessors."
This is from the '81 Surgeon General's Report, correct, that you are citing and quoting in your article?
A. It's a direct quote.
Q. And then this is your language: "The more recent case control evidence remains consistent with the first component of this conclusion." That being the sentence that I just read, correct?
A. Yes, also I think in light of what I just said in both the Surgeon General and I were wrong, of course. Today filter tip cigarettes we have to wait a while, but I think that was in reference to perhaps -- again, as I said, we always have to look forward, but I think what they meant was today's in general here.
Q. I understand your point that the cigarettes that people are smoking today cannot be precisely assessed until many years down the road, but with respect to the state of existing scientific knowledge at that time period your view was that the case control evidence available was consistent with the conclusion that lower tar and nicotine cigarettes produced lower rates of lung cancer than do their higher tar and nicotine

predecessors, correct?
A. I'm sorry, in the new studies, again, were primarily a comparison of filter to nonfilter users.
Q. All right. In 1999, indeed when this lawsuit -- about the time this lawsuit was filed, it was your opinion, was it not, that smokers of filter tip -- excuse me, of filter tip cigarettes had about a 20 percent lower risk of lung cancer than smokers with nonfilter cigarettes?
A. Well, that again is essentially the finding of the analysis of CPS-I data. In 1959, just to put it in context, that was 1959 to '72 the window covered by that study.
Q. We'll bring it up-to-date in a little while, Doctor, but it was your view in 1999 that there was about a 20 percent reduced risk of lung cancer for lower yield product smokers, correct?
A. Again, that was what was in the literature, correct.
Q. Now, in your direct testimony in this case, you have basically modified your review; is that right, on the issue we just discussed?
A. I'm sorry, I have not modified my view of what the evidence showed in CPS-I.
Q. Let me be more precise. You believe today that those results may not be truly representative and that they may present an incomplete picture of the risks, or the risk reduction that may attend smoking lower tar, lower nicotine

cigarettes, correct?
A. I'm not sure I phrased anything quite that way.
Q. All right. Is it your opinion that there is no benefit to low-yield cigarettes?
A. Well, again, that's different from what the lung cancer risk estimates showed 30 years ago. I don't -- I'm not sure that providing an opinion on the overall benefits of lower yield cigarettes conflicts with my earlier comments about the literature.
Q. Let's be more precise, and let me talk about lung cancer risk only.

Is it your opinion today that the smokers of low-yield cigarettes do not receive any benefit, with respect to lung cancer, by virtue of the fact they're smoking lower yield rather than higher yield products?
A. Well, I think there's no evidence of clear benefit.
Q. Do smokers of lower yield products have a lower risk of lung cancer, in your judgment today, than smokers of high yield cigarettes?
A. Again, I can't speak to today, and I think the -- my expert report covers other aspects of this problem. The increasing relative risks of smoking comparing the two CPS-I and 2 studies, so there's other lines of evidence that have been looked at for this question.
Q. Having examined those other lines of evidence, I'm just

trying to get your opinion, Doctor.

Do you believe, yes or no, that smokers of lower yield cigarettes have a lower risk of lung cancer than smokers of high yield cigarettes?

A. Again, I'm just going to comment that I find it very hard to speak to what today's products will produce in the future.

Q. Your testimony is you don't know?

A. I can't predict the future.

Q. What is the existing state of evidence on that question, Doctor, with respect to the people who were smoking these cigarettes 10, 20 or 30 years ago? What is the existing state of the evidence?

A. I think the state of the evidence has been well summarized in the reports of the Surgeon General, IARC, the Institute of Medicine, each group that's looked at the question of whether today's lower yield cigarettes are likely to produce -- are likely to produce lower risk of lung cancer, has said, you know, no clear benefit. They've looked to the past evidence, IARC commented on the relationship between machine measured yields and what gets into people. This is a tough area for scientific inquiry.

Q. I understand that, Doctor, and I'm not sure I caught all of your answer. So let me try to be precise.

In 1999, you testified, or you said in an expert report, that the smokers of lower yield cigarettes, on average, had a

20 percent decrease in the risk of lung cancer compared to the smokers of high yield or nonfilter cigarettes, correct?

A. And again, I've said very clearly what time window of evidence that was in reference to. It was in reference to these past studies, and that's approximately the risk reduction observed in the CPS-I and some other studies.

Q. Do you believe that's still true today, based on the evidence that we have today, that smokers of lower yield products have approximately 20 percent risk reduction with respect to lung cancer when compared to smokers of high yield or nonfilter cigarettes?

MS. EUBANKS: Objection, Your Honor. The witness has been asked this question several times and has answered it the same way each time.

THE COURT: Objection's overruled.

BY MR. McDERMOTT:

Q. Is that your opinion today, Doctor?

A. I don't, -- I don't have a reason to extend the risk reduction that was observed 30 plus years ago, 40 years ago, for the comparison of filter to nonfilter smokers to what will happen in the future when people consume today's products into the future. That's an unanswerable question with great certainty.

Q. Is the answer to my question no?

A. I'm sorry, which aspect of your question at this point?

Q. Do you believe there is a 20 percent risk -- today do you believe there is a 20 percent risk reduction for smokers of lower yield products with respect to lung cancer compared to the smokers of high yield products?
A. I believe that's been observed in the past, but I don't know if it will hold in the future.
Q. You don't know. You don't have an opinion?
A. I said I don't know if that will hold in the future.
Q. All right. Thank you.
Doctor, you don't consider yourself an expert on compensation as such, do you.
A. Not specifically, no.
Q. Okay. Dr. Benowitz is?
A. I believe so.
Q. He would know more about that subject than you do, you will concede?
A. He's likely to, yes.
Q. Okay. Nevertheless, based on your own personal review of the literature and your own thinking and experience in this field, you do not believe, do you, that smokers of lower yield products compensate completely when they switch from higher delivery cigarettes, do you?
MS. EUBANKS: Objection, Your Honor. Given the testimony from this witness, how can it be relevant what he believes when he's already testified that he's not an expert on the area of

compensation and that the expert is Dr. Benowitz, someone that the United States will bring forward to testify?

THE COURT: It doesn't mean he can't answer the question, and certainly it is a subject to which he has to have considered in the area that is his specialty. If he can't answer it, he'll tell us.

THE WITNESS: Well, I will say when I answer a question firmly I like to know I'm in command of the whole literature and on this topic I don't think I am, that is, the compensation topic.

BY MR. McDERMOTT:

Q. So you don't have an opinion as to whether compensation is 100 percent complete, you don't have an opinion one way or the other, is that correct?

A. Sitting here today I did not come here today prepared to answer that question.

Q. All right. In your direct testimony, Doctor, you talked about biomarkers, certain levels of biomarkers and how they do or don't correlate with FTC measurements, that's at transcript pages 147 to 148. I want to ask you about that.

You suggested in your testimony that a low correlation between a smokers biomarker levels and the FTC ratings was evidence, in your view, that smokers compensated for the reduced yield in their lower tar and nicotine products, correct?

A. Yes.

Q. All right. Doctor, if I tell you a correlation coefficient between FTC nicotine yield and a biomarker for nicotine is .57, and that's all you know, and that's the only information you know, can you tell me what the percent of compensation is?
A. That's not a -- an answerable question for me.
Q. That's not enough information to answer the question, is it?
MS. EUBANKS: Objection, Your Honor. That mischaracterizes, by way of the question, what the witness's answer was.
THE COURT: Objection's overruled. You may answer, if you can.
THE WITNESS: I simply can't answer the question as it's posed.
BY MR. McDERMOTT:
Q. That's not enough information, is it?
A. Well, I don't know what biomarker and I don't -- this is so hypothetical I can't answer it.
Q. Okay. Correlation coefficients and the presence or absence of compensation, they're not the same things, are they, Doctor?
A. Correlation is a statistical measure. Compensation is a physiological phenomena.
Q. Okay. It is possible, is it not, Dr. Samet, to calculate

the level of compensation from some of the biomarker studies in Monograph 13? Isn't there -- there is a formula for doing that, correct?

MS. EUBANKS: Objection, Your Honor. Again, we're going into an area that the witness has expressly stated that he's not competent to testify about.

THE COURT: Objection's overruled. If you can answer it, you may.

THE WITNESS: Again, I will say that I've not come here today prepared to discuss compensation.

BY MR. McDERMOTT:

Q. So, of the biomarker studies that are reported in Monograph 13, and they are set forth for the record at table 3.1, you don't know what the level of compensation for any of those studies is, correct?

A. Well, if I could see table 3.1.

Q. Okay. Sure. Let's look at U.S. 58700, table 3.1. It's on page 51 and 52, I believe.

These studies set forth, do they not, Dr. Samet, the results of some studies that report levels of biomarkers when comparing actual smoking versus machine yield?

A. Well, this is a series of studies, not of biomarkers generally, but of nicotine and -- possibly the blood specifically.

Q. The right-hand column?

MS. EUBANKS: Excuse me, could the witness be allowed to finish his answer please?

MR. McDERMOTT: I'm sorry. I thought he had.

BY MR. McDERMOTT:

Q. Sorry Doctor, I did not mean to cut you off.

A. Again, it seems to be a set of correlations of nicotine yields from different sources versus levels of various measures, whether it appears to be measures of nicotine or codeine concentration.

Q. Okay. They're comparing the yield of nicotine by the FTC method and then looking at biomarkers and seeing how much the smokers actually receive with some recognized biomarkers for nicotine intake, correct?

A. That appears to be the case, yes.

Q. Okay. Nowhere in this table is there any information on the level of compensation that may have been found in any of these studies, correct? That information is not reported?

A. Well, this is a comparison, or set of correlations, coefficients is what is given, apparently of the two measurements, nicotine field verse the machine yield versus what was measured in people.

Q. Okay. To your knowledge, Doctor, do you know whether a percent of compensation, if any, was computed with respect to these studies in connection with the preparation Monograph 13?

A. If it was, it's not shown in this table.

Q. All right. And do you know of any place else in the volume where it's reported?
A. I can't say that I've looked for that.
Q. And do you recall whether or not, based on your personal contact with the people in the development and evolution of this volume, whether that was done and not reported, whether somebody did it and just didn't bother to put it down?
A. I never made any such inquiry.
Q. Okay. Dr. Samet, I want to return to one of the points you made in monograph 7 that epidemiology addresses the risk of cigarettes under natural circumstances of use. Talking about, for the moment, within cigarette compensation as opposed to numbers of cigarettes per day, I want to put that aside for the moment -- with respect to within cigarette compensation, the epidemiology studies do take account of whether individuals smoking lower tar cigarettes smoke more intensively, do they not?
A. Well, they observe the risks as people are actually smoking the cigarettes.
Q. And that can include more puffs per cigarette, or deeper puffs, or more frequent puffs, that kind of compensatory smoking behavior, correct?
A. That's correct.
Q. And would you agree with Dr. Burns that the majority of compensation which has been found, is this within cigarette

compensation, with a smoker changing the way he smokes a cigarette, as opposed to increasing the number of cigarettes per day?

MS. EUBANKS: Objection again, Your Honor. This witness is not brought forward as an expert on compensation and these continual questions about that really are a waste of the Court's time to get his opinion. We'll have witnesses to testify about these matters.

THE COURT: I'm going to sustain the objection at this point.

MR. McDERMOTT: Let's approach it this way. Can I see Dr. Samet's direct testimony in this case, starting at page 164, line 15?

BY MR. McDERMOTT:

Q. Dr. Samet, take a moment and -- go over to the next page, please. See your answer?

Starting again, "The Surgeon General's Report did not fully take into consideration the phenomena of compensation, and how smokers smoke to get a certain amount of nicotine and will even adjust their smoking behavior to get the amount of nicotine they seek to be accustomed too. As I stated before, we now know that smokers do this through adjusting their pattern of smoking to smoke more cigarettes inhale deeper, and take more puffs."

Did I read your testimony correctly, Doctor?

A. Yes, you did.

Q. All right. Is it your opinion that smokers compensate both by changing the way they puff individual cigarettes, and by smoking more cigarettes, when they switch from higher delivery products to lower delivery products?
A. That appears to be the case, yes.
Q. It appears to be the case? Is that --
A. Yes.
Q. -- your opinion or isn't it?
A. Yes.
Q. Okay. Now, and again, you said that epidemiological studies inherently take account of the within cigarette compensation, smoking more intensively, taking more puffs per cigarette and the like, correct?
A. Yes, I did.
Q. Okay. And now I want to focus on cigarettes per day. Excuse me, before I change I want to go back to the question I asked before.

Do you agree with Dr. Burns that the majority of compensation occurs from this intensive smoking per cigarette rather than from increasing the number of cigarettes per day?

MS. EUBANKS: That objection was sustained and I'd like to add, in addition to that, we don't even have an establishment of any foundation that he knows what Dr. Burns has said. Dr. Burns is an expert who is scheduled to testify in this case. And I think if this witness is going to be asked about the opinions of

Dr. Burns, in fairness, he should be shown those so he can comment on them in a competent fashion.

MR. McDERMOTT: I'm happy to do that, Your Honor, but these gentlemen were coauthors of a chapter of Monograph 13 that dealt with this very subject, but I'm happy to show Dr. Burns' testimony if the Court or the witness would like.

THE COURT: Do you need to see the testimony, or do you remember it since you co-wrote it?

THE WITNESS: If I can make the comment the chapter I contributed to is the epidemiological chapter, not the chapter on compensation.

BY MR. McDERMOTT:

Q. Your chapter did discuss compensation on cigarettes per day, did it not doctor?

A. It included analysis of some data, but again, it was a chapter on epidemiology that I contributed.

MS. EUBANKS: Your Honor, we're going to have Dr. Burns here and --

THE COURT: I understands that, but they want to question this witness to see if they can get certain answers.

Dr. Samet, are you able to answer the previous question that was asked of you?

THE WITNESS: I'm sorry, which previous, Your Honor?

THE COURT: Back about one question. I know which one you're referring to.

THE WITNESS: All right.
BY MR. McDERMOTT:
Q. Do you agree with Dr. Burns that the majority of the compensation occurs with compensatory smoking within cigarettes rather than through smoking more cigarettes per day when somebody switches from a higher yield product to a lower yield product?
A. Again, I will say that I did not come here today prepared to discuss compensation, and I don't have, really, a strong opinion one way or the other as to what Dr. Burns has proffered and I haven't seen that statement, in any case, from Dr. Burns.
Q. And you do not personally have an opinion on the issue of where the majority of compensation takes place or whether it's within cigarettes or cigarettes per day?
MS. EUBANKS: Your Honor, his personal opinion would have no relevancy here. He's here to testify as an expert, and what his personal opinion is, if it's not premised upon his studies or his expertise has no relevance to this Court and doesn't assist the Court in the decision.
THE COURT: Mr. McDermott?
MR. McDERMOTT: I'm not asking for personal views, I'm asking for his professional opinion, whether he has one on this issue, that's all, Your Honor.
THE COURT: And you may answer if you do or if you do not.
THE WITNESS: I think I said I really do not sitting here

today.
BY MR. McDERMOTT:
Q. Okay. Well, should the Court and the parties ignore the testimony that you provided in your direct on compensation because that's just not an issue that you're really prepared to talk about?
A. I did not come here today prepared to talk about compensation. I've said that very clearly.
Q. All right. One possibility with respect to compensation, which you addressed on page 164 of your testimony, and we saw it earlier, is that when smokers switch from higher yield to lower yield products, they may increase the number of cigarettes they smoke per day, correct?
A. That's correct.
Q. And that's a theory, or a hypothesis, that was considered in Monograph 13, correct?
A. It was addressed in Monograph 13, correct.
Q. Okay. In your direct testimony at page 167, lines 9 and 10, you say, "Smokers of lower yield products tend to smoke more cigarettes per day than smokers of higher yield products." Do you see that? And if you want to see more text, you're welcome.
A. I see that, yes.
Q. All right. You don't cite anything in this portion of your testimony in support of that proposition. Is it the Monograph 13 analysis that you principally rely on to support

this?
A. Monograph 13 would be one source of data. There are other reports that have been published on this phenomena.
Q. Okay.
MR. McDERMOTT: Let's look at Monograph 13, page 81, please. U.S. 58700.
BY MR. McDERMOTT:
Q. "However, control for intensity of smoking across populations using number of cigarettes smoked per day as the measure of dose may result in a model mis specification if smokers who switched to low-yield cigarettes compensate by increasing the number of cigarettes that they smoke per day."
Now, as that's phrased, it says "may result", that's just a hypothesis, correct?
A. It's a description of what such a model would produce under the circumstances that smokers of lower yield cigarettes have increased numbers of cigarettes smoked.
Q. Okay. And the concern is that, if I understand the thrust of what you're trying to say, if a smoker of a higher yield product switches to a lower yield product, he or she may end up smoking more cigarettes, and so, if you do an epidemiological study that simply controls for number of cigarettes, you may not line up right, that the lower yield, you may have a false or an illusory reduction in risk because you're not comparing apples to apples, right, in your theory?

A. You would be not using the -- you're making -- you're making the smoker the of the lower yield products appear to be smoking the same as they smoked under the higher yield circumstances.
Q. Okay. The possibility that smokers who switch to lower yield products might end up smoking more cigarettes per day is not a new theory, is it, Dr. Samet?
A. Well, "theory", I would probably not use that word, but it's certainly not a new hypothesis, no.
Q. Okay. And in fact, this has been examined empirically in the scientific literature prior to Monograph 13; isn't that correct, Doctor?
A. There are studies in this regard.
Q. All right.
MR. McDERMOTT: Let's look at NCI monograph 28. I final got to the right number. JD 063324.
BY MR. McDERMOTT:
Q. And that monograph is entitled Toward a Less Hazardous Cigarette, published in June of 1968, and I want to --
MS. EUBANKS: You misread that.
MR. McDERMOTT: I'm sorry.
MS. EUBANKS: "Less harmful".
MR. McDERMOTT: I have a typo here, I apologize.
BY MR. McDERMOTT:
Q. Toward a less harmful cigarette.

Direct your attention to an article entitled Relationship, -- or a portion of that articled entitled Relationship to Number of Cigarettes Smoked to Tar Rating by Selwyn Waingrow and Daniel Horn, and in particular, let me direct your attention to page 30.

Okay. About mid page the authors note, "There's little relationship, however, between the tar rating score and the number of cigarettes smoked." Do you see that?

A. I see that.

Q. Okay. And then they at the bottom of the page, they say, "In examining the relationship between a change in the tar rating score and any change in the number of cigarettes smoked, no statistically significant relationship was found."

That finding is inconsistent, is it not, with the hypothesis that was advanced in Monograph 13?

A. The words as stated are inconsistent. I haven't seen the evidence for the analysis, so it's hard for me to comment in further detail, but that's what the words say.

Q. Well, let's look at a -- let's -- of particular interest, where is that quote? It's about -- it's I think at the bottom of the page. Here we go. "Of particular interest is the observation that those who were categorized as having shown a reduction in their tar rating score, exactly the same number, 27.3 percent, were classified as smoking more cigarettes as were classified as smoking fewer cigarettes than previously with 45.3 percent showing no change."

That observation is inconsistent with your testimony that smokers of lower tar cigarettes tend to smoke more cigarettes per day than smokers with higher yield cigarettes, isn't it, Doctor?
A. Again, I can't comment in great detail because I don't know what population, what study, or what methods. I only see the conclusions of the authors.
Q. And the conclusion is not consistent with your direct testimony, correct?
A. As stated, I've seen none of the evidence supporting the conclusion.
Q. Let's go to JD 000659. And this is the Banbury Report, which is a volume of papers that were produced. This was published in 1980, correct? You're generally familiar with it?
A. Yes, I am.
Q. Okay. Let me turn to a -- the paper by Garfinkel. Let's go to page 19.
Okay. Dr. Garfinkel worked for the American Cancer Society, correct?
A. Yes, he did.
Q. Okay. And he examined in this paper, did he not, the relationship between cigarettes per day and tar and nicotine yields among individuals who switched to CPS-I?
A. Well, that's not apparent from what is here. I suspect this was a paper related to CPS-I smokers, yes.
Q. Okay. Let's go to page 24, focus on the sentence under

the chart, please.

Starting at the beginning of that sentence, "Our data demonstrate that although nicotine dependence may have some effect in the short run, over a long period of time people tend to smoke the same number of cigarettes a day regardless of tar and nicotine level." Do you see that?

A. I do, and again, I think some of these same data were reanalyzed in Monograph 13.

Q. All right. As reported here by one of the chief health officials of the American Cancer Society, "Cigarettes per day do not increase as you reduce the yield of cigarettes" and this is inconsistent with your opinion that smokers who switch tend to smoke more cigarettes, correct?

A. Again, this is Dr. Garfinkel's statements about his -- the evidence shown here.

Q. Okay. And it's inconsistent with your opinion, correct?

A. It's inconsistent -- as stated, again, I have not seen this in evidence here, but his conclusion suggests that there was not a change.

Q. Okay. And the 1989 Surgeon General's Report also concluded that smokers' daily consumption of cigarettes did not change from 1974 to 1983, even though the sales weighted average of tar and nicotine yields declined, correct? Let me show you JE 063621, page 293, bottom of the page.

"Because sales weighted average nicotine yields declined

from 1974 to 1983, one might expect to have observed an increase in average daily cigarette consumption. Compensatory changes in smoking behavior to maintain relatively constant nicotine intake have been shown to occur when smokers switch from high-yield to low-yield cigarettes. Although daily cigarette consumption did not increase from 1974 to 1985, other compensatory changes may have occurred, e.g., increased frequency, puffing or depth of -- frequency of puffing or depth of inhalation as the smoking population moved toward lower yield cigarettes."

So the Surgeon General was saying in his report in 1989 that the compensation is within cigarette if it occurs, not increasing the number of cigarettes per day, correct?

A. I think the statement says that across that time window as estimated, however they did it, there was not a change in daily cigarette consumption with whatever changes there was in sales weighted tar and nicotine level across the 11-year interval.

Q. Okay. And in any case, Doctor, this is not consistent with your testimony that smokers of lower tar cigarettes tend to smoke more cigarettes per day than smokers with higher yield cigarettes, is it?

A. Well, this particular evidence would conflict with that statement, correct.

Q. Okay. And you explore this issue in your 1993 article with Dr. Coultas, didn't you?

MR. McDERMOTT: Let's see JD 063062.
BY MR. McDERMOTT:
Q. Are you familiar with the study?
A. Yes.
Q. Okay.
MR. McDERMOTT: Let's see page 437.
BY MR. McDERMOTT:
Q. You report the results of your study and you notice relationships, a number of relationships. I can read the whole quote, but what I want to direct your attention to is the bottom line: "FTC tar and nicotine yields were not significant."
You were studying, were you not, the behavior of smokers switching from -- high tar smokers versus low tar smokers, and tar and nicotine yields were not significantly correlated with cigarettes per day, were they?
A. This was not a study of switchers, this was simply a cross sectional survey at this point.
Q. Okay. But the number of cigarette -- tar and nicotine yields were not a significant predictor -- I'm sorry, I've got that back -- you did not find a statistically significant relationship between FTC yields and cigarettes per day, did you doctor?
A. Not in this particular population.
Q. Okay. So this study is not consistent with your opinion in this case that smokers of lower tar cigarettes tend to smoke

more cigarettes per day than smokers with higher yield cigarettes, is it?

A. This particular piece of evidence is not.

Q. Okay. And it's a piece of evidence that you personally participated in generating, correct?

A. I was involved in the study.

Q. All right.

MR. McDERMOTT: Let's look at figure 4.7 of Monograph 13.

THE COURT: I think, actually, we'll take a break at this point. Let me just get down something.

Okay, let's take a 15-minute break, everybody.

(Thereupon, a break was had from 3:23 p.m. to 3:40 p.m.)

MR. MCDERMOTT: May I proceed, Your Honor?

THE COURT: Yes, please.

BY MR. MCDERMOTT:

Q. Dr. Samet, I want to go back and just clear up one thing that I'm not sure we got quite straight. I'm putting on the ELMO the Garfinkel study that we -- which you saw earlier and I want to direct your attention to the reference here, the prospective study we analyzed. And then let me show you the next page where they describe the study population.

As you can see here, Doctor, there's a reference to cigarettes smoked in '59 and in 1972 follow-up. Does that refresh your recollection that this is dealing with the ACS-I or

CPS-I population?
A. Oh, I understand that, yes.
Q. Okay. Let me move on, then, Doctor, to Monograph 13. And I want to look at -- that's U.S. 58700. And I'd like to look at Figure 47-7, which appears at page 115.
Now, Doctor, this is one of the previously unpublished statistical analyses that's presented in Chapter 4 of Monograph 13, isn't it?
A. It's presented in that monograph, yes, and I believe it was not previously published.
Q. To your knowledge it was not?
A. To my knowledge, no.
Q. All right. And this, if I understand it correctly, purports to analyze whether people who switch brands -- change brands from higher tar to lower tar or when they switch brands at all change the cigarettes they smoke per day, correct?
A. This is an analysis directed at whether there was a change in the number of cigarettes smoked in relationship to the change in nicotine level as going from the present -- the former brand to the present brand.
Q. Okay. And one of the things that this figure is trying to address, if I understand it, is whether or not when people switch, when smokers switch from a higher yield product to a lower yield product, they tend to smoke more cigarettes per day, correct?

A. Yes. Actually, I think it's sort of bi-directional, it's just which way --

Q. True.

A. -- whether it went up or down. My comment was this was bi-directional; it was whether there was a change in cigarettes smoked per day in relationship to either an increase or decrease in the nicotine yield.

Q. Okay. Now, as you see down at the bottom, Doctor, where it says "Source," this is dealing with the CPS-I population, correct?

A. That's correct.

Q. So the population being examined here is the same population that Dr. Garfinkel examined and published on in the Banbury report, correct?

A. The overall study was the same. I don't know whether exactly the same people are in the two sets of analyses.

Q. It's pretty clear, though, that the results of those analyses are completely different, correct? Dr. Garfinkel, as we saw just a moment ago, said, and I quote: "Over a long period of time, people tend to smoke the same number of cigarettes per day regardless of tar and nicotine level."

Right? That's what he said?

A. That's correct.

Q. Okay. This suggests that as people move up and down the scale of tar and nicotine deliveries, they smoke different

numbers of cigarettes. That's the thrust of this graph, correct?
A. I think the thrust of it is the line that's shown there that would describe that relationship, correct.
Q. Okay. And the negative slope, the fact that it's sloping downward as it goes from left to right, suggests that as people decrease the measured nicotine yield of their cigarettes, they tend to smoke more cigarettes, correct? That's the thrust of this analysis?
A. Well, if they decrease, the line goes up. That's right. If they increased, they smoked less.
Q. Okay. If the slope had been positive, if it had gone up from left to right, that would mean as people increased the nicotine yield -- it would mean just the opposite, right? As people lowered their cigarette yield, they would reduce the number of cigarettes, correct? That's what the opposite slope would mean?
A. That's correct. For the negative, minus, I think probably what's important is the minus 2.31 up there that describes the slope of that line and it says that as nicotine goes up by a milligram, on average, the number of cigarettes smoked goes down by roughly two.
Q. Okay. And if this line had been level, just flat dead level, that would mean that there is no change in the number of cigarettes per day smoked regardless of whether you moved up or

down the tar and nicotine -- the nicotine yield line, correct?
A. Correct.
Q. Okay. You can't tell us, based on -- you did not, I gather, Doctor, participate in the underlying analysis, underlying data -- number crunching that produced this chart; is that correct?
A. Myself personally, no.
Q. Okay. And you don't know, based on your lack of familiarity with the underlying work, whether this analysis of the CPS-I data or Dr. Garfinkel's analysis of the CPS-I data got it right? You can't of your own personal knowledge say which was the better analytically, which was the more accurate, which is the one that should be relied on; isn't that true?
A. They're very different. I mean, Dr. Garfinkel's analysis grouped things and said: Did the amount go up or down of cigarettes smoked or did it say the same?

This analysis, I think, deals more with the quantitative relationships. So Dr. Garfinkel's table had this qualitative expression: Did the number of cigarettes increase or decrease?

Here there's an attempt to describe that quantitatively, so I think they're somewhat different approaches.
Q. But you haven't basically gone behind the Court, either with respect to Dr. Garfinkel's analysis or with respect to this analysis, and looked hard at the numbers and the analytical approach taken; isn't that correct?

A. I haven't looked at the source data or the analysis, no.
Q. Do you know whether or not defense experts have look at the source data and looked at the way in which this figure was constructed and the underlying data that went into this figure?
A. No, I have no knowledge.
Q. Okay. So you have not been shown Dr. Wecker's analysis or reanalysis of this data?
A. No, I have not.
Q. Okay.
THE COURT: I don't really understand the distinction that you tried to draw between this analysis in Monograph 13 and Dr. Garfinkel's analysis in the Banbury report. Would you try and explain that.
THE WITNESS: Let me explain. Dr. Garfinkel said: If people changed their brands, did they smoke more, the same or less? Just those three bins, if you will.
What this analysis says is: Let's look at whether there's a relationship between the reported numbers of cigarettes smoked on the first brand and on the second brand and the difference in nicotine yield on the first brand and the second brand. So this is a more quantitative analysis and Dr. Garfinkel's was more sort of: Did people change from smoking more than they used to to less than they used to? And this one tries to get at quantifying that relationship.
BY MR. MCDERMOTT:

Q. Doctor, in fairness, you haven't really drilled down into either the Garfinkel analysis or this analysis, have you? You really can't speak beyond what appears on the page here, can you?
A. The page is all I have. I don't have the source data to look at.
Q. You didn't prepare it? You didn't participate in the construction of this chart? You're not familiar with how the underlying data was analyzed as such, are you?
A. Well, from what is shown here in the Garfinkel study as well, I can certainly surmise what kind of analysis was done.
MR. MCDERMOTT: Let's see on the screen JD 065989.
Excuse me, Your Honor. We have a technical glitch.
BY MR. MCDERMOTT:
Q. This is an expert report prepared by Dr. Wecker in this case. And as you see -- and he performed a reanalysis of the same data and came out with a perfectly flat line. That's an overstatement, a flat line. No statistically significant slope.
Now, let me ask you, Doctor: Have you seen this analysis before?
A. No, I have not.
Q. Okay. But if this analysis is correct, if this is a more accurate analysis of the underlying data, this slope would indicate, would it not, that people who switch do not tend to smoke more cigarettes; is that right?

MS. EUBANKS: Your Honor, there is a lack of foundation. The witness has testified regarding the previous exhibit, which is similar to this, that he didn't have anything but that document before him and couldn't offer testimony on it. He's certainly not competent to offer testimony on one page of defense -- the exhibit that's put before him that purportedly defendants' expert has put together. It's not appropriate examination. It's certainly beyond the scope of cross and it goes far beyond the witness's qualifications to testify.

MR. MCDERMOTT: Your Honor, I'm not asking Dr. Samet to choose sides and say which is the more accurate analysis. We'll present testimony on that. I'm asking if he agrees that if this analysis is accurate, what it means is smokers who switch from high yield to low yield cigarettes do not on average smoke more cigarettes per day.

MS. EUBANKS: The objection is the same, Your Honor. There's a complete lack of foundation to throw up a chart like this.

THE COURT: If the doctor can answer the question, he may.

THE WITNESS: The only comment I can make is that there is a difference between a flat line shown here and the negative line shown in the prior exhibit.

BY MR. MCDERMOTT:

Q. And a flat line would mean that smokers would not increase their cigarettes per day as they switch from a higher

yield to a lower yield product, correct?
A. That would be the interpretation of a flat line.
Q. Thank you. Now, Dr. Samet, the hypothesis that Monograph 13 was dealing with; that is, that smokers who switch to lower yield products might smoke more cigarettes per day -- it's possible to test that hypothesis, isn't it, simply by removing the control of cigarettes per day and seeing what happens?
A. I don't think I follow you.
Q. You don't follow me? The concern you have is, to pick numbers out of the air, if somebody who smoked 20 cigarettes per day of a high yield product switched to a low yield product, that person might end up smoking 25 cigarettes per day, correct? That's the kind of cigarette-per-day compensation you're concerned about?
A. Concerned about an increase in numbers, correct.
Q. Okay. And one of the reasons that you argue or contend that the apparent lower relative risk experienced by low tar smokers, low yield cigarette smokers, is an artifact of comparing 20 cigarette-a-day smokers of high yield with 20 cigarette-a-day smokers of low yield when in fact you should be comparing 20 cigarette-a-day smokers of high yield products with 25 cigarette-a-day low yield products.
Again, the numbers are approximate. I'm just trying to get the idea.
A. I think the general point that one would want to use the

actual number of cigarettes smoked, whether higher yield or lower yield smokers, is correct.

Q. Okay. But my question is, Doctor, if that's the concern, one way to test whether that's a problem is just to eliminate the controls for cigarettes per day, right, and just use all smokers of high yield and all smokers of low yield product and not control or adjust the cigarettes per day?

A. Well, I think probably the way to do it would be to estimate the risks associated with the actual numbers of cigarettes smoked within the groups and make the proper comparison, using the information available if one had it.

Q. But, Doctor, if you just removed the controls of cigarettes per day and the lung cancer risk and the difference in lung cancer risk between high yield and low yield smokers remained the same or are approximately the same, wouldn't that suggest that the cigarette-per-day compensation hypothesis that you are advancing was not correct?

A. Again, it's not the comparison I would make. I think I said what the comparison I would make would be.

Q. All right. Do you know Dr. Jay Lubin?

A. Yes, I do.

Q. In fact, you and he have co-authored several different articles, haven't you?

A. Yes, we have.

Q. Let me show you JD 061657. This is an article entitled

"Patterns of Lung Cancer Risk According to Type of Cigarettes Smoked," published in the International Journal of Cancer in 1984.

Let me turn to page 571, the second sentence under the heading: "The risk of lung cancer was higher among exclusive nonfilter smokers and mixed smokers than among lifetime filter smokers after adjusting for cessation in either the years of use or number per day."

That's number of cigarettes per day, correct?

A. Yes, that's what it says.

Q. All right. Let's turn to Table 5 at page 572.

And there are two separate analyses, correct, Doctor, one where you control and one were you do not control for cigarettes per day?

A. I guess you're referring to the fact that there's analyses that are broken up or stratified by number per day and then they have what is called the "adjusted."

Q. Correct. And at the very top of the chart, it reports that for males, smokers of exclusively nonfiltered cigarettes, they had a relative risk of 1.8 compared to the smokers of nonfilter cigarettes, correct, that number?

Do you see that?

A. I'm sorry, no. Where are you --

Q. Okay. If you just --

A. Yes, I do. Yes.

Q. Filter cigarettes, 1, compared to 1.8?
A. Yes.
Q. See that?
THE COURT: Compared with 1.8?
MR. MCDERMOTT: Excuse me. Compared with 1.0. I'm sorry, Your Honor.
BY MR. MCDERMOTT:
Q. And smokers of exclusively nonfilter cigarettes had a relative risk of lung cancer of 2.5 compared to exclusively filter, correct?
A. That's within the group of one to 29 years of smoking.
Q. The same basic relationship obtains, though, does it not, when you adjust?
A. I guess I will assume that the data by years of use are not adjusted and the one that says "adjusted" is adjusted for years of use.
Q. Let's go back to 571, where it talks -- let me see, where is that adjustment?
Okay. "After adjusting simultaneously for years of use, number smoked per day and years since cessation, smoking nonfilter cigarettes exclusively, on mixed brands, resulted in a 1.7-fold -- 1.6-fold excess risk for males and a 2.0 for excess risk for females compared to smoking filter brand cigarettes."
So that when you make that adjustment, the relative risk doesn't change, right? 1.8 versus 1.7?

A. I'm sorry. Are you referring to the 1.8 in Table 5?
Q. Yes, that's correct.
A. So that is -- at least I think that is adjusted for years of use, as I interpret this table.
Q. And this is adjusted for cigarettes per day. There's no change in the relative risk; there's no material change whether you adjust for cigarettes per day or not, correct?
A. I don't think the one -- again, I think that 1.8 is not adjusted for cigarettes per day, but for years of use, as I interpret this table.
Q. And, Doctor, we probably don't have time for you to give me a tutorial. Let's move on.
Dr. Lubin is with the National Cancer Institute, isn't he?
A. Yes, he is.
Q. And so is Dr. Bob Tarone?
A. Yes, he is.
Q. Okay. And neither one of these gentlemen is affiliated with the tobacco industry, to your knowledge, correct?
A. That's correct.
Q. They commented on Monograph 13, didn't they, as it was being prepared?
A. I believe they wrote peer review comments.
Q. Okay. And you reviewed their comments during the course of your participation in the preparation of Monograph 13, didn't

you?
A. I reviewed the various peer review comments on the monograph.
Q. Okay. And didn't they specifically note that: "The lung cancer risk amongst smokers of lower tar cigarettes are lower both in studies that control for cigarettes per day and in studies that do not control for cigarettes per day"?
A. I don't remember those particular comments.
Q. Okay. Let's look at Dr. Tarone's comments. JD 063687.
A. Excuse me. I think I've been handed the wrong exhibit.
Q. I think you have been, too. Here you are.
"Analytic studies -- i.e., case control studies -- and cohort studies have been very consistent in showing lower lung cancer risk with reduced cigarettes per day."
Carrying on: "The Burns et al. chapter" -- and that's the chapter we were just looking at, Chapter Four, right? -- "attributes the low risk estimates obtained from analytic studies largely to bias. In particular, Burns et al. claimed that adjustment for smoking intensity, the number of cigarettes per day, induces bias because people who switched to lower tar and nicotine cigarettes increased the number of cigarettes per day."
And then he goes on to say: "Not all studies have adjusted for smoking intensity. However, even those that do not adjust for smoking intensity tend to show reduced risk."

That is at odds with and tends to -- that is inconsistent with your opinion, isn't it, Dr. Samet, that smokers who switch tend to smoke more cigarettes per day?

A. This is not particularly relevant to that. I think, again, my comments about Table 4.1 and what the evidence in Table 4.1 shows are consistent with what Lubin and Tarone are saying here. @@

Q. Let's go on: "Moreover, Burns et al. present little direct evidence that such bias exists, even though they could investigate the presence of such bias; e.g., using a CPS-I study. Lubin et al. published two papers reporting a large case control study of lung cancer in Europe."

All right. Down here: "The risk estimates reported for filter cigarettes compared to nonfilter cigarettes were virtually identical in the two papers."

I'm sorry. I got that backwards. Let me go on.

"As summarized in Table 4.1, Burns et al. analysis of one paper" -- and that's the paper that Dr. Lubin himself wrote -- correct -- we just saw?

A. Yes.

Q. Okay. "Adjusted for duration of smoking, number of cigarettes per day, while analysis in the second paper, British Medical Journal, adjusted only for duration of smoking. The text incorrectly" -- and so forth. That's not material.

"Burns et al. conjecture that the evidence for reduced

risk of low tar cigarettes from CPS-I study might have resulted from such bias in adjusting for smoking intensity. It is puzzling that Burns et al. do not present direct evidence for such a bias."

Correct?

A. That's what it says, correct.

Q. Didn't Dr. Tarone and Dr. Lubin take strong issue with the approach that Dr. Burns took in analyzing the CPS-I data and attributing all of the risk reduction that has been shown in prior EPI studies to an increase in cigarettes per day?

A. I'm not aware that the monograph where Dr. Burns attributed all, to use your words, of the risk reduction to this potential bias. Clearly, Drs. Lubin and Tarone are expressing their point of view here about the analyses that were included in the draft monograph.

Q. They disagree with it, right?

A. Their viewpoints are stated here.

Q. Okay. Dr. Samet, can you point me -- you cannot point me, can you, to a single study that you considered wherein the observed risk -- excuse me -- observed reduction in lung cancer risk among smokers of lower tar cigarettes disappeared entirely or substantially when control for cigarettes per day was removed, can you?

A. I probably could not.

Q. All right. You stated in your direct examination that:

"If changes in cigarette yields had any benefit, you would expect the risks of lung cancer to be dropping over time as FTC tar and nicotine yields decline." Correct?

A. Correct.

Q. In other words, your testimony is predicated in large part on the expectation that the lung cancer rates should have dropped with the lower tar and nicotine yields, but they didn't decline; is that right?

A. Relative risks in particular.

Q. Correct. And so the hypothesis, I take it, is that if all other factors affecting lung cancer risk in smokers remained constant except for the tar and nicotine yield, when you delivered less tar and nicotine on average, you would expect the lung cancer -- the relative risk to go down if those cigarettes conferred any benefit -- risk reduction benefit, right?

A. That's the hypothesis.

Q. All right. But if other factors besides the tar delivery of the cigarettes affected the lung cancer risk and didn't remain constant, the question becomes a little bit more difficult to analyze, doesn't it?

A. Well, if such factors are changing over time in the same way that tar and nicotine yield were, they would need to be taken into account.

Q. Okay. Isn't it true, Dr. Samet, that the increase in lung cancer rates which we've seen in recent years, could be

accounted for or could be the result of unmeasured differences in early-life smoking in the different populations of smokers studied?

A. I'm sorry. Can you be more specific?

Q. All right. Let's look at JD 000540. It's an article written by Dr. Michael Thun entitled "Changes in Mortality From Smoking From Two American Cancer Society Prospective Studies Since 1959."

Let me direct your attention to page 425, where Dr. Thun writes: "So the rise in lung cancer death crates from CPS-I to CPS-II." That's the increase you were talking about, correct, Doctor?

A. This is one of the few opportunities that we have to observe changing relative risks over time.

Q. Okay. "May reflect unmeasured differences in early life smoking."

That's what Dr. Thun posits, correct?

In fact, one of your charts that you showed earlier today showed a huge increase in the cigarette consumption from the '30s to the '50s, correct, Doctor?

A. That's correct.

Q. From the chart? Okay. So unmeasured differences in early life smoking.

Let's go to JD 064118. This is an article written by Dr. Thun, "Excess Mortality Among Cigarette Smokers: Changes in

a 20-Year Interval," published in the American Journal of Public Health in 1995. And let me direct your attention to page 1228, right-hand column, second paragraph.

"Thus the increase in death rates from lung cancer seen from CPS-I to CPS-II may reflect unmeasured heavier smoking in CPS-II during the 1940s and '50s even when current daily cigarette consumption and years of smoking appear to be equivalent among middle aged smokers in the two studies."

If that's true, if smoking intensity was much heavier in the CPS-II population, that could very well account for the rise in lung cancer rates which you have commented on, correct, Doctor?

A. I think Dr. Thun is being a very careful and cautious interpreter of his information here.

Q. Let me show you JD 000540, Thun et al., "Changes in Mortality From Smoking: Two American Cancer Society Prospective Studies Since 1959," published in Preventive Medicine in 1997.

Let me direct your attention to page 424: "Early life smoking increased markedly around World War II when CPS-II smokers were adopting the habit." Do you see that.

A. Yes, I do.

Q. That's consistent with your chart showing a huge jump in smoking, World War II and thereafter, isn't it, Doctor?

A. This is one of the points where there was an increase.

Q. Okay. Let me show you a letter from Sir Richard Peto to

Dr. Benowitz, dated July 17th, 2000. It's JD 004426.

This was something that relates to your work on Monograph 13, correct?

A. I would need a minute to look at this letter.

Q. Certainly. Take your time.

A. I believe this was written in relation to Monograph 13, although it doesn't say specifically.

Q. Okay. Do you recall seeing this particular exhibit during your involvement in Monograph 13?

A. I've seen this information a number of times, probably in the context of Monograph 13 as well as others.

Q. All right. And let's go to -- let me see.

Can you scroll through it, where it says "among older men"? Do you have "among older men"?

"Among older men and among women, the trends are dominated by the massive increase caused by replacement of smokers who have not smoked substantial numbers of cigarettes throughout adult life by smokers who had done so. This change involves not only the age when the first cigarette is smoked, but also the intensity of cigarette smoking in early adult life."

Correct?

A. That's what he says, yes.

Q. Okay. Do you agree with Dr. Peto that increases in early life smoking during the 20th century have affected lung cancer

rates?
A. Well, I think, first, in the context, I think Richard Peto here is referring to the UK experience and describing his impression of what smoking patterns would have been. I think it's likely that -- we know that there was a starting of smoking earlier in life across at least the earlier part of the century in men and across the mid century in women.
Q. So you think this comment is confined to the UK; is that your recollection?
A. My recollection of Richard's discussion of this relates to interpretation of the data below, which are for the UK, correct.
Q. Okay. And he also made the same case with respect to the U.S. populations, didn't he, Dr. Samet?
A. My -- I don't believe that Richard has extended these -- his arguments about the change in rates in the younger age cohorts have, to my knowledge, been confined to the UK rates.
Q. Let me show you JD 000529. This is an article authored by Peto and Sir Richard Doll and others and appears in the British Medical Journal and was published in 2000.
Let me turn to the discussion, pages 327 and 328.
Okay. "By 1950, the increase in smoking was too recent to have had its full effects on disease rates except perhaps among men in early middle age. The fact that by 1990, many of the current smokers would have smoked substantial numbers of

cigarettes throughout adult life is the chief reason for the large increase in the cumulative risks of lung cancer among continuing smokers. For the same reason, increases in risk associated with smoking were also seen between the first 20 years, 1951 to 1971, and the next 20 years, 1971 to 1991, of follow-up in the prospective study of smoking death among British doctors and between the two large prospective studies carried out by the American Cancer Society in the 1960s and the 1980s."

Same phenomenon, in Sir Richard's opinion, apply to both sets of populations, correct?

A. That's true. But again, the sentence not seen, which is right above, is that his information that's all in relationship to the UK and he's now extended these arguments to the United States.

Q. And he reviewed the literature with respect to that, didn't he? He has citations here?

A. I don't know the basis for -- he's citing simply here the American Cancer Society studies, but not patterns of smoking in the U.S.

Q. Okay. Do you agree with Dr. Peto that smoking in early life is the chief reason for the increases seen in lung cancer risk in the second half of the 20th century? Let me ask you first with respect to the UK.

A. Lung cancer has gone up for many reasons in the second

half of the 20th century. Certainly there were more smokers; the number of women smoking has gone up, so the rise in smoking is far more than an increase in -- I'm sorry -- lung cancer is far more than an increase in early life smoking alone.

Q. I'll ask you again: Do you agree with Dr. Peto that increases in early life smoking are the chief reason for increases in lung cancer risks observed in the second half of the 20th century?

A. I need some help, whether you mean lung cancer risks, lung cancer relative risks -- what you're referring to, to answer that question.

Q. Lung cancer relative risks.

A. I think that is one potential theory. I think that is a contributing explanation. There may be others.

Q. So basically, we may have two conflicting -- we may have two different forces in play. One could be an increase in intensity of smoking in early life with later study populations that would tend to drive up the relative risks; and then we have the operation of the changes in cigarette designs and the lowering of tar.

And whether that would move it up or down or level it off, it's pretty hard to say in the abstract, isn't it, Doctor?

A. Well, I think perhaps the point is that -- again, with the limited information we have, particularly within comparable populations, CPS-I and II, the relative risks of smoking have

gone up approximately 12 in men in CPS-I and roughly 20-fold increase in CPS-II.

Q. But it is certainly possible, isn't it, Doctor, that the influence of increased smoking intensity in the latter half of the 20th century could outweigh real benefits that might be conferred by smoking lower delivery products -- again, talking about lung cancer risk; isn't that correct?

A. I think I've commented elsewhere on the difficulty of trying to sort this out. I think, again, the evidence shows increasing relative risks and an increase -- a decrease in the age of starting to smoke may have contributed to that.

Q. Let's go to JD 00540. Again, this is the Thun article. And let's go to page 424. It's a discussion in the second paragraph, 424; the second paragraph, page 424, starting with "First."

"First, although the sharp increase in lung cancer death rates from CPS-I to CPS-II is clearly an adverse consequence of cigarette smoking, we cannot tell whether lung cancer risk might not have increased even more but for the reduced tar yield in cigarettes. Early-life smoking increased markedly around World War II, when CPS-II smokers were adopting the habit."

This is clearly dealing with the U.S. population, correct?

A. Yes.

Q. And Dr. Thun is positing that while the intensity of

smoking may have pushed the relative risks of lung cancer up, it is quite possible that this was mediated or ameliorated by reduced yield products, correct?

A. Certainly he does not say it's quite possible. He offers that possibility, but he did not say "quite possible."

Q. All right. We'll use his words. "We cannot tell whether the risk -- lung cancer risk might not have increased even more but for the reduced yield -- tar yield in cigarettes."

What he's saying there is if it weren't for low yield products, it might have been worse, correct?

A. He offers that hypothesis.

Q. Okay. And this is the same hypothesis -- this is the same argument that Sir Richard Peto advanced with respect to the UK population, based on his work in England -- correct? -- that the advent of lower yield products did, in fact, lower the relative risk of lung cancer even in the face of contrary forces as a result of increased smoking intensity, correct?

A. Richard has interpreted temporal patterns of changes in rates and in relative risks as possibly reflecting consequences of yield of products.

Q. Okay. Let's look at JD 004426 and let's go to page 2. "My" -- second page.

"My interpretation is that the hazard among UK male cigarette smokers age 40 is a bit lower in 1995 than it was in 1955 despite the fact that smokers age 40 in 1995 probably

smoked a lot more in their teen-age years than smokers age 40 in 1955. Hence, I conclude that if" -- a big "if" -- "we ignore the extent to which changes in cigarette competition may have directly or indirectly increased cigarette consumption in various ages, the lung cancer hazards of the way smokers actually use one pack a day are less than half of what they used to be."

Do you agree with Sir Richard that today's cigarette -- with today's cigarettes, the lung cancer hazards of the way cigarette smokers actually use one pack a day are less than half of what they used to be?

A. Again, this is Richard's interpretation, based on the changing patterns of lung cancer rates in the UK. It's one of the pieces of information that those of us who have taken on the challenge of trying to understand changing cigarette yields have used. It argues for some reduction in risk associated with change in yield.

How much? It certainly offers no quantification. And I respect Richard's opinion and here it is.

Q. Okay. And his opinion is inconsistent with the opinion you're offering in this case, isn't it, Doctor?

A. This is, again, one line of evidence that I have considered and others have considered, including the IARC working group, of which Richard was a member.

Q. His opinion that lower yield products did confer some

benefit, i.e., contributed to the reduction of relative risk with respect to lung cancer -- you disagree with that, don't you? Or he disagrees with you?

A. I -- again, this is his interpretation of the information. It's -- I don't know how he's precisely come to this quantification, so I can't speak to that. I have a letter in front of me, not a scientific report.

Q. Okay. Well, let's look at JD 004427. This is a letter to Dr. Burns, again in connection with Monograph 13, correct? It's dated August 14, 2000.

You've seen this before, have you not?

A. I believe I've seen this letter. And again, it's -- while not specifically stated in relation to Monograph 13, I believe it was.

Q. Okay. And he says: "At the start, I would like to put on record my main comments about how best to interpret the U.S. and the UK lung cancer time frames in relation to cigarette consumption and composition."

Correct?

A. Correct.

Q. Okay. And he argues that instead of just looking at cohort data, huge populations, that instead you look at age-specific trends, correct?

A. Well, he says: "Analyses of age-specific trends may be a more reliable indicator." That's what he says.

Q. Okay. Let's go to the next page. And he presents data with respect to U.S. males age 35 to 39 that run from 1968 to 1997, right? At the top of the page there?
A. Correct.
Q. Okay. And he comments: "Men in early -- for men in early age, the decrease in lung cancer mortality is substantially larger than can plausibly be explained by changes in the prevalence of smoking among adults."
Correct?
A. Sir, give me a moment. Where is that?
Q. It's highlighted right there on the screen, Doctor.
A. That's his comment, yes.
Q. Okay. He also notes in the letter that --
Can you scroll down a little bit.
He makes a specific reference to -- here we go: "Since 1986, the UK and the U.S. trends among men in early middle age have been very favorable." Right?
And then he encloses copies and he says: "Don't let mathematical models obscure this." Right?
He then cautions at the bottom of this letter: "European countries such as France and Poland illustrate how bad male lung cancer mortality trends could have been in middle age. Copies enclosed."
And then he encloses some analyses, right? Let's look at those.

Let's go to page 11 of this exhibit. Do we have the attachments?

I think it's page 11 of this exhibit.

MS. EUBANKS: We don't have that.

BY MR. MCDERMOTT:

Q. All right. And in particular --

(Discussion had off the record.)

BY MR. MCDERMOTT:

Q. Doctor, we're having technical difficulties. Let me move on.

THE COURT: Well, it is 4:30. I had hoped we could finish. How much more do you think you have?

MR. MCDERMOTT: Not very much more, Your Honor. Ten or 15 minutes.

THE COURT: I think, given that we're all going to be here for the long haul, I'm going to do my best to keep to the schedule that we announced. There will be days, naturally, when we'll go later than 4:30, but at least for now, I'm going to keep to it.

There is going to be some cross by Lorillard; is that right?

MR. MINTON: Just a few minutes, Your Honor.

THE COURT: And then, Ms. Eubanks do you have any sense yet on how long your redirect will be?

MS. EUBANKS: Probably no more than ten or 15 minutes,

Your Honor.

THE COURT: So you'll be getting to your nonexpert witnesses tomorrow?

MS. EUBANKS: That's correct, Your Honor.

THE COURT: All right, everybody. Thank you.

Dr. Samet, you may step down.

I'll see everybody at 9:30 tomorrow.

(Proceedings adjourned at 4:31 p.m.)

C E R T I F I C A T E.

I, Scott L. Wallace, RDR-CRR, certify that the foregoing is a correct transcript from the record of proceedings in the above-entitled matter.

Scott L. Wallace, RDR, CRR
Official Court Reporter

Scott L. Wallace, RDR, CRR
Official Court Reporter

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