

**UNITED STATES DEPARTMENT OF LABOR**

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**OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION**

**PUBLIC HEARING**

**PROPOSED STANDARD FOR INDOOR AIR QUALITY**

**PAGES: 14673 through 14989**

**PLACE: Washington, DC**

**DATE: March 10, 1995**

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**BAYLEY REPORTING, INC.**  
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UNITED STATES DEPARTMENT OF LABOR

OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION

PUBLIC HEARING  
PROPOSED STANDARD FOR INDOOR AIR QUALITY

Friday,  
March 10, 1995

Department of Labor  
Washington, D.C.

The above-entitled matter came on for hearing,  
pursuant to notice, at 9:00 a.m.

BEFORE: HONORABLE JOHN VITTONI  
Administrative Law Judge

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AGENDAPAGE

Dr. Steven Bayard  
EPA

14677

## Questions:

Mr. Furr

14747

Mr. Davis

14886

Mr. Furr

14927

Ms. Sherman

14974

Mr. Myers

14981

Mr. Weinberg

14985

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EXHIBITS

<u>EXHIBIT NO.</u>	<u>IDENTIFIED</u>	<u>RECEIVED</u>
277	14742	14742
278	14742	14742

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P R O C E E D I N G S

1

2

9:04 a.m.

3

4

5

JUDGE VITTONI: We resume our hearings this morning into the proposed indoor air quality rule by the Occupational Safety and Health Administration.

6

7

We have one witness today and it's Dr. Steven Bayard from the Environmental Protection Agency.

8

9

It's my understanding that Dr. Bayard has a direct presentation of at least one hour.

10

Is that right?

11

DR. BAYARD: I expect an hour and one half, Judge.

12

13

JUDGE VITTONI: An hour and a half. How many slides do you have?

14

DR. BAYARD: Fifty-seven.

15

JUDGE VITTONI: Fifty-seven slides?

16

DR. BAYARD: Yes.

17

18

JUDGE VITTONI: You have copies, I hope, for the record?

19

DR. BAYARD: Yes, I do.

20

JUDGE VITTONI: Okay

21

22

DR. BAYARD: I've give copies both to the attorneys for OSHA and for the attorneys for the public.

23

And I've also left copies outside.

24

JUDGE VITTONI: Okay. Thank you, Dr. Bayard.

25

So we will begin with his direct presentation and

1 then after that we will open it up for direct examination by  
2 the participants.

3 Are there any preliminary matters we have to take  
4 up before we get started?

5 (No audible response)

6 JUDGE VITTON: Okay. All right.

7 Who is going to be operating the slides for you,  
8 Dr. Bayard?

9 DR. BAYARD: Ms. Jinot.

10 JUDGE VITTON: All right. Thank you. I'll get  
11 out of your way and you can begin as soon as I get out of  
12 your way.

13 DR. BAYARD: Thank you very much. Thank you,  
14 Judge.

15 (Pause)

16 JUDGE VITTON: Doctor, go ahead.

17 DR. BAYARD: Thank you, Judge Vittone, Ms.  
18 Sherman, members of OSHA, members of the public. My name is  
19 Steven Bayard. I will be making a presentation of  
20 approximately an hour and a half. I have presentations both  
21 from my prepared testimony on March 10, 1995 which is very,  
22 very similar to the testimony which I submitted in August or  
23 September, I forget, I think it was August.

24 I will read the first two pages of my testimony  
25 and from there I will go on to slides and I will then finish

1 with the last couple of pages of the testimony.

2 My name is Steven Bayard. I am the United States  
3 Environmental Protection Agency project manager and  
4 co-author for the report on the respiratory health effects  
5 of passive smoking.

6 I am here today to discuss EPA's findings on ETS  
7 and comment on the ETS sections of OSHA's proposed rule on  
8 indoor air quality. In addition to my testimony, I have  
9 submitted copies of the EPA report on passive smoking; a  
10 report of the EPA Science Advisory Board on its review of  
11 the second external draft of the EPA report, November 20,  
12 1992; number three, the EPA fact sheet on the respiratory  
13 health effects of passive smoking, January 1993; number  
14 four, EPA fact sheet on setting the record straight, June  
15 1994; number five, EPA's policy brochure on secondhand  
16 smoke, July 1993; number six, a letter from EPA's Office of  
17 Research & Development, Acting Assistant Administrator Gary  
18 Foley to Dan Mulholland, Director, Congressional Research  
19 Service, June 23, 1994; and, finally, two journal articles  
20 rebutting criticism of the EPA risk assessment by a tobacco  
21 industry consultant.

22 MR. FURR: Your Honor, I really hate to have to  
23 object here. The problem we have today, as we all know,  
24 we're going to be very short of time. Is there any reason  
25 to take up time with reading the testimony that he submitted

1 back in August?

2 JUDGE VITTON: I don't think that up to this  
3 point we have stopped anybody from making a direct  
4 presentation in the manner that they wanted to. That goes  
5 for all of the witnesses. He's going to take an hour, an  
6 hour and a half. As I said earlier, we're going to be here  
7 for a long time today, I assume. He feels comfortable doing  
8 this and it's not going to take an inordinate amount of  
9 time. I'm going to let him go ahead.

10 MR. FURR: I apologize for interrupting but could  
11 we also identify the other people at the table?

12 JUDGE VITTON: Yes. I'm sorry.

13 Dr. Bayard, before you go too much further, would  
14 you identify your colleagues?

15 DR. BAYARD: I've never seen these people before  
16 in my life.

17 MR. SHEEHAN: We can identify ourselves, I think,  
18 Your Honor. I'm John Sheehan with the Environmental  
19 Protection Agency.

20 JUDGE VITTON: Okay.

21 MS. NEUWIRTH: And I'm Laura Neuwirth from the  
22 Environmental Protection Agency.

23 MR. FOOTE: I'm Greg Foote, also from the  
24 Environmental Protection Agency. We are all representing  
25 the Office of General Counsel.

1 JUDGE VITTON: Okay. You're all with the General  
2 Counsel's office?

3 MR. SHEEHAN: That's correct.

4 On the point just raised, Mr. Bayard's only going  
5 to read the first couple of pages of his testimony and then  
6 he's going to do the slides.

7 JUDGE VITTON: No, I understand that. Okay.

8 Dr. Bayard, go ahead.

9 DR. BAYARD: Thank you.

10 I hold a B.S. degree in mathematics from Tufts  
11 University and a Ph.D. in biostatistics from the Johns  
12 Hopkins University. I have also received training in  
13 biochemistry and toxicology.

14 From 1970 to 1974, I was Assistant Professor of  
15 Biometry at Yale University, where I taught courses in  
16 biostatistics, epidemiology and demography.

17 I have worked as a statistician for the  
18 Environmental Protection Agency for the last 16 years, where  
19 one of my primary functions is to assess various chemical  
20 compounds and mixtures for potential carcinogenicity. I  
21 work in the Office of Health and Environmental Assessment,  
22 which performs these assessments for several of EPA's  
23 program offices. The reports provide the scientific support  
24 for regulatory policy development.

25 Among those substances for which I have performed

1 assessments are asbestos, dioxin, vinyl chloride, methylene  
2 chloride, ethylene oxide and nickel. I have been employed  
3 only by the United States Government since 1974.

4 In recent years, comparative risk studies  
5 performed by EPA and its Science Advisory Board have  
6 consistently ranked indoor air pollution among the top five  
7 environmental risks to public health. Environmental tobacco  
8 smoke is one of the major indoor air pollutants and, given  
9 the known health impact of tobacco smoking, there has been  
10 concern that non-smokers may also be at risk of serious  
11 health effects.

12 As part of its efforts to address all types of  
13 indoor air pollution, EPA's Indoor Air division in 1988  
14 requested that EPA's Office of Research and Development,  
15 which is my chief office, undertake an assessment of the  
16 respiratory health effects of smoking. Because of both  
17 resource and time limitations, the assessment was limited to  
18 the respiratory health effects, both cancer and non-cancer,  
19 rather than a broader investigation.

20 The report was prepared by ORD's Office of Health  
21 and Environmental Assessment and was written with both  
22 in-house staff and outside contracting assistance.

23 Before being released in draft form for public  
24 review, the passive smoking report received many internal  
25 reviews, mostly from within ORD. Various parts of it were

1 also reviewed by outside experts, both from other federal  
2 agencies and from academic institutions. Revisions  
3 incorporated the reviewers' comments wherever possible.

4 A first external draft of this assessment was  
5 released for public review and comment in June of 1990. In  
6 December 1990, the EPA Science Advisory Board, a committee  
7 of independent outside scientists, conducted a review of the  
8 draft report and submitted its comments to the EPA  
9 administrator in April of 1991. In its comments, the SAB's  
10 indoor air quality total human exposure committee concurred  
11 with the primary findings of the report but made a number of  
12 recommendations to strengthen it.

13 Incorporating recommendations from both the public  
14 and the SAB, a revised draft was transmitted to the SAB in  
15 May 1992 for a second review.

16 Following a July 1992 meeting, the SAB panel  
17 endorsed the report and its conclusions, including a  
18 unanimous endorsement of the classification of environmental  
19 tobacco smoke as an EPA Group A or known human carcinogen.

20 The EPA also received and reviewed public comments  
21 on the second draft and integrated all appropriate material  
22 into the final risk assessment. The final report was  
23 released in January 1993.

24 I will now proceed to make a discussion from the  
25 slides.

1 (Slide 1)

2 DR. BAYARD: This is just the front cover of the  
3 report. While it's not on the slide, it's one of the two  
4 slides I forgot, I feel it is as important as any of the  
5 slides I'm going to present because I want to tell you who  
6 the authors of this report are.

7 Jennifer Jinot, who is showing the slides right  
8 now, and I are the EPA co-authors. However, there are five  
9 co-authors who are contractors and were a very integral part  
10 of the team.

11 Kenneth Brown from North Carolina wrote chapters  
12 five and six and several of the appendices, did a lot of the  
13 analyses on lung cancer. He is a biostatistician Ph.D.

14 Fernando Martinez is an M.D. pulmonologist from  
15 the University of Arizona Medical Center in Tucson. He did  
16 chapter seven and part of chapter eight. Chapter seven  
17 dealt with the non-cancer respiratory effects in children  
18 and adults.

19 Brian Leaderer is an associate professor of  
20 environmental sciences, I think, at Yale. He did our work  
21 on chapter three on exposure assessment.

22 Neal Simonsen is a Ph.D. epidemiologist, did much  
23 of the Appendix A which provides details of the 30  
24 epidemiology studies on environmental tobacco smoke and lung  
25 cancer.

1           Judson Wells is a Ph.D. physical chemist, now  
2 retired, formerly a DuPont executive, was also an OSHA  
3 expert witness. Wrote our Appendix B on smoker status  
4 misclassification.

5           Thank you.

6           Oh, if you want a copy of this, by the way, you  
7 can just call 513-569-7562. They're free.

8           (Slide 2)

9           DR. BAYARD: Now, while we covered several health  
10 effects in the report, there were several we didn't cover.  
11 The ones that we did cover were lung cancer, non-cancer  
12 respiratory effects in adults. In children, we covered the  
13 lower respiratory tract which includes bronchitis,  
14 pneumonia, asthma, wheezing and bronchial  
15 hyporesponsiveness. Upper respiratory tracts includes  
16 coughs, colds and sore throats, sputum and phlegm. We  
17 covered sudden infant death syndrome, middle ear fluid and  
18 infections, lung function. In adults, we included lung  
19 function and respiratory symptoms.

20           What we didn't cover. In children, we didn't  
21 cover development effects, behavioral effects and childhood  
22 cancers. There is evidence on leukemia and brain. And also  
23 there's evidence in other cancers, including breast,  
24 cervical, nasal sinus cancer. We didn't cover those.

25           We didn't cover heart disease. I understand OSHA

1 has done an evaluation of heart disease. The EPA did not  
2 cover that. We also did not cover irritation.

3 For purposes of these hearings today, we will  
4 limit our discussions to adults, lung cancer, lung function  
5 and respiratory symptoms.

6 (Slide 3)

7 DR. BAYARD: The outline of the report, and I  
8 won't go into this in detail but we will include it in the  
9 record, in the interests of brevity, it covers the  
10 conclusions on chapter three, which was the composition and  
11 exposure assessment that was done by Dr. Leaderer. Lung  
12 cancer has an identification and population risk estimates.  
13 And we also did the non-cancer respiratory effects and  
14 population estimates for those.

15 (Slide 4)

16 DR. BAYARD: Slide 4, the major conclusions.  
17 Based on the weight of the available scientific evidence,  
18 the U.S. EPA has concluded that the widespread exposure to  
19 environmental tobacco smoke in the United States presents a  
20 serious and substantial public health impact.

21 In adults, ETS is a human lung carcinogen  
22 responsible for approximately 3000 lung cancer deaths  
23 annually in the U.S. non-smokers.

24 In children, ETS exposure is causally associated  
25 with an increased risk of lower respiratory tract infections

1 such as bronchitis and pneumonia. This report estimates  
2 that 150,000 to 300,000 cases annually in infants and young  
3 children up to 18 months of age are attributable to ETS.

4 ETS exposure is causally associated with increased  
5 prevalence of fluid in the middle ear, symptoms of upper  
6 respiratory tract irritation and a small but significant  
7 reduction in lung function.

8 ETS exposure is causally associated with  
9 additional episodes and increased severity of symptoms in  
10 children with asthma. This report estimates that 200,000 to  
11 one million asthmatic children have their condition worsened  
12 by exposure to ETS.

13 ETS exposure is a risk factor for new cases of  
14 asthma in children who have not previously displayed  
15 symptoms.

16 (Slide 5)

17 DR. BAYARD: Slide 5. We had other primary  
18 findings. EPA estimates that --

19 MR. FURR: Judge, I would object to this. Why are  
20 we taking up time today talking about alleged findings with  
21 respect to children that have absolutely no relevance to the  
22 workplace rule.

23 DR. BAYARD: It's just a few more sentences,  
24 Mr. Furr.

25 JUDGE VITTON: Mr. Furr, let's let him get his

1 presentation out. If it's not relevant to what's happening  
2 here, you can point that out in whatever comes down later in  
3 this process, okay? But let's just let him get his  
4 statement out on the record and let's find out what he has  
5 to say, okay?

6 DR. BAYARD: Thank you, Judge.

7 EPA estimates that ETS exposure may be responsible  
8 for 8000 to 26,000 new cases of asthma annually in children  
9 who have not previously displayed symptoms.

10 Passive smoking has subtle but significant effects  
11 on the respiratory health of non-smoking adults including  
12 coughing, phlegm production, chest discomfort and reduced  
13 lung function.

14 There is strong evidence that infants whose  
15 mothers smoke are at an increased risk of dying from sudden  
16 infant death syndrome. However, available studies did not  
17 allow us to differentiate whether and to what extent this  
18 increase is related to in utero versus postnatal exposure to  
19 tobacco smoke products.

20 We were able to make no conclusions on upper  
21 respiratory tract infections or acute middle ear infections.

22 (Slide 6)

23 DR. BAYARD: Slide 6. I'll be talking about Group  
24 A carcinogens so I just thought that I would put up a slide  
25 so that you could know what EPA has concluded are Group A

1 carcinogens. These include arsenic, asbestos, benzene,  
2 benzidine, bis(chloromethyl)ether, chromium 6, coke oven  
3 emissions, diethylstilbestrol, the three benzidine based  
4 dies that are black, blue and brown, environmental tobacco  
5 smoke, 2-naphthalamine, nickel, radon, vinyl chloride.

6 Furthermore, environmental tobacco smoke is the  
7 only carcinogen in Group A for which the cancer risk in  
8 humans was detected at typical environmental exposure levels  
9 rather than occupational or pharmaceutical levels.

10 EPA's environmental tobacco smoke risk assessment  
11 also classifies mainstream tobacco smoke and sidestream  
12 tobacco smoke as Group A carcinogens.

13 (Slide 7)

14 DR. BAYARD: Table 7 is a table on the components  
15 of environmental tobacco smoke and in deference to Mr. Furr  
16 I will assume that most people know what happens when you  
17 smoke a cigarette and I will move on to the next slide.

18 (Slide 8)

19 DR. BAYARD: Slide number 8. Number 8 is a table  
20 from table 3 and a lot of which is produced in OSHA's notice  
21 of proposed rulemaking which details some of the components  
22 of mainstream and sidestream smoke, mainstream smoke  
23 comprising roughly 15 or so percent of environmental tobacco  
24 smoke and sidestream smoke composing the rest. The two  
25 largest components on slide 8 are carbon monoxide and carbon

1 dioxide and the concentrations go down from there.

2 (Slide 9)

3 DR. BAYARD: Notice from slide 8 and slide 9 that  
4 while all the components are in both mainstream and  
5 sidestream smoke their distributions vary.

6 Next, slide 10, please?

7 (Slide 10)

8 DR. BAYARD: Also in our exposure assessment we  
9 looked at all the exposures that we could find of markers of  
10 environmental tobacco smoke and what slide 10 presents in  
11 two different charts are the concentrations of markers of  
12 environmental tobacco smoke in multiple settings using two  
13 different markers, one is respiratory suspended particulates  
14 and one is nicotine.

15 The conclusions we draw from both of these are  
16 that -- well, let me tell you what they are. The thick bars  
17 in the middle represent the range of average values of all  
18 the studies that we found and the upper limits are similarly  
19 demarcated by the more slender bar.

20 What these charts tell us is that residential  
21 exposures and occupational exposures tend to have fairly  
22 similar average concentrations with the exception that  
23 restaurants tend to be higher, transportation in some places  
24 is higher, others such as hallways or stairways or  
25 elevators, train stations, may have more extreme values. So

1 some of the averages, some of the occupational places went  
2 up considerably.

3 So in general, the message that I think I would  
4 like you to take from this is that the residential and  
5 workplace exposure levels average about the same with the  
6 exception that the workplace exposure levels tend to have  
7 some higher extremes.

8 Next, please?

9 (Slide 11)

10 DR. BAYARD: The weight of evidence. For lung  
11 cancer, we determined that environmental tobacco smoke was a  
12 known human carcinogen, it belonged in EPA's category Group  
13 A by means of weight of evidence. And that weight of  
14 evidence includes these categories.

15 Strong dose relationships for active smoking for  
16 all four major lung cancer types with no evidence of a  
17 threshold.

18 These relative risks for active smoking range up  
19 to 20 or 30. They are highly dose responsive in terms of  
20 intensity, number of years smoked, age at which smoking  
21 began. And while the dose response relationships may differ  
22 for the different major lung cell types, they may differ in  
23 slope, there are all four types.

24 The chemical similarity of mainstream and  
25 environmental tobacco smoke both mainstream and ETS contain

1 over 40 known or suspected human carcinogens. Supporting  
2 evidence from animal bioassays and genotoxicity studies  
3 which include rat lung implantation of sidestream smoke  
4 condensate, mouse skin painting with sidestream smoke  
5 condensate in which sidestream smoke condensate actually  
6 produced more tumors than the mainstream smoke.  
7 Genotoxicity studies, there were studies showing that  
8 environmental tobacco smoke extracts and sidestream smoke  
9 extracts produced genotoxic results. And biomarker evidence  
10 of ETS uptake by non-smokers which are correlated with air  
11 measures.

12 In addition, there are various analyses of 30  
13 epidemiology studies on environmental tobacco smoke and  
14 cancer.

15 Now, slide 12 please.

16 (Slide 12)

17 DR. BAYARD: For the 30 epidemiology studies,  
18 there were 27 case controlled studies and 3000 cases and  
19 6100 controls. There were four cohort studies, 250 cases,  
20 280,000 follow-up. One of those case control studies was  
21 nested in a cohort study and so in the final analysis only  
22 26 case controlled studies were used and four cohort  
23 studies. These came from eight different countries.

24 Now, the ETS exposure surrogate we used was  
25 spousal smoking and that is we looked at the effect of women

1 with and without spouses who smoked to find out whether or  
2 not their lung cancer experience and risks differed. The  
3 benefit of this, their actual environmental exposure,  
4 spousal smoking and marriage habits tend to be of fairly  
5 long duration, so that would tend to be a stable measure  
6 over a long period of time.

7 Now, these are also actual environmental exposure  
8 levels and so if there's an effect here, there's very little  
9 from which to extrapolate downward.

10 Two types of exposure misclassification, however,  
11 and both types tend to decrease the likelihood of observing  
12 an effect if one exists. First, people with spouses who  
13 don't smoke are still exposed to environmental tobacco smoke  
14 from other sources, when they go out and go on a bus or  
15 wherever smoking is. And they'll say, no, my spouse doesn't  
16 smoke and so they'll be recorded as unexposed but then in  
17 reality they are going to be exposed and this is the problem  
18 with getting a clean control group, you just can't do it.

19 Some people whose spouses smoke may not actually  
20 be exposed to appreciable amounts of ETS also. The spouses  
21 may smoke, they just may not smoke in the home. Either that  
22 or he may not be home.

23 Smoker misclassification status, on the other  
24 hand, may provide an upward bias and we'll be getting into  
25 that a little later. In fact, right now.

1 (Slide 13)

2 DR. BAYARD: Slide 13. Some of the potential  
3 sources of bias in these studies, epidemiology studies are  
4 rather difficult from which to determine a true causality.  
5 And that's because there are so many sources of bias.

6 Misreporting or misclassification of smoking  
7 status, Dr. Wells has testified on this but essentially it's  
8 that smokers have higher lung cancer risks and tend to marry  
9 smokers and for spousal smoking, the EPA considered that  
10 such a bias was a potential bias and might exist.

11 I will add, though, that it's not necessarily a  
12 bias. We took what I thought was a conservative approach  
13 but there are reasons why even though smokers may not have  
14 proper recall of their actual smoking status that this might  
15 not be a bias. There are times when misclassification rates  
16 do not translate into bias, it's only when there is  
17 differential between exposed and non-exposed.

18 For example, I don't think we have any indication  
19 at all that lung cancer cases tend to misclassify their  
20 status at the same rate as controls. In fact, when lung  
21 cancer cases are asked about smoking, there's this problem  
22 of recall bias which means that they remember too much and  
23 so they may remember things about their own smoking that  
24 controls do. If that's the case, then the positive  
25 systematic bias could actually be a negative bias.

1           Furthermore, the tendency for a smoker  
2           misclassification bias to exist depends for the most part on  
3           current surveys of smoker concordance. That is, smokers  
4           tend to like smokers and so they marry smokers and  
5           non-smokers marry non-smokers. But the cases that we're  
6           getting are from case controlled studies where exposure was  
7           developed 40 and 30 years ago when that type of concordance  
8           may not have existed. Just an aside.

9           Background exposure for unexposed and that is  
10          we've just gone into that and that is that the unexposed  
11          people are getting exposure from other sources, the people  
12          whose spouses don't smoke.

13          Misdiagnosis. Misdiagnosis is interesting because  
14          it can be both positive and negative and you can have both  
15          false positives and false negatives. And that is that lung  
16          cancer cases -- we said -- let me stop for a minute. We  
17          said in our draft that these are non-systematic biases and  
18          so we didn't bother trying to correct for them. While we  
19          did try to correct for one, the bias in one and the bias in  
20          two, we didn't try to correct for the bias in three, four or  
21          five because we said, okay, they're non-systematic, they're  
22          probably going to bias the estimates downward but we don't  
23          have the methodology right now to correct for them.

24          However, there are studies which indicate that all  
25          of these biases will estimate, will drive the estimates

1 downward. so while we expect that there may be some upward  
2 biases or confounders which some of the studies did not take  
3 into account which might bias the estimate in an upward  
4 position, it's these which I am also pointing out to you  
5 which could drive the estimate downward.

6 For example, misdiagnosis. Let's say we have a  
7 cohort study and we don't diagnose all our primaries so we  
8 have false negatives, primary lung cancers. Well, if  
9 there's any effect of passive smoking, that will be lost  
10 because you won't diagnose all these people. It will also  
11 affect the population risk estimate. Where we said there  
12 were 38,000 lung cancer deaths in a single year and the we  
13 partitioned them for a population risk estimate, if in fact  
14 there were more that we just missed, then our risk estimates  
15 are too low and OSHA's would be similarly affected.

16 Proxy respondent. I'll present evidence later on  
17 that will show you that proxy respondent doesn't seem to be  
18 a major problem for spousal studies. However, it seems to  
19 be a fairly major problem for childhood exposures and for  
20 workplace exposures. And I think that's important to know.  
21 People in general will know about their own smoking habits,  
22 their spouses will know about their spouses' smoking habits,  
23 but when you start asking someone else about what happened  
24 to this case's exposure either as a child or in the  
25 workplace, you're not going to be so sure and that's going

1 to produce some bias.

2 Publication bias. We did the best we could to  
3 eliminate publication bias and let me define publication  
4 bias as the tendency to publish results either positive or  
5 negative but I think it's been argued that people want to  
6 publish results that are positive and what we tried to do in  
7 this case was to find every study we could, whether it was  
8 positive or negative, whether it was published or not. I'm  
9 sorry. And that way we hoped to avoid publication bias.

10 So our cutoff was determined not by -- for lung  
11 cancer, was determined not by whether or not the study was  
12 published but by whether it was available to us by a certain  
13 cutoff date.

14 Next, please.

15 (Slide 14)

16 DR. BAYARD: Now, EPA looked at the spousal  
17 smoking studies. We also had -- a lot of these spousal  
18 smoking studies also had information on workplace and  
19 childhood. We found that there's a lot of problems with  
20 workplace exposure measures and one is that for the  
21 workplace you have to have one measure represent 30 or 40  
22 years of exposure.

23 Now, people just don't -- at best, their exposures  
24 change and they can change for several reasons so that you  
25 don't get a stable type of exposure in the workplace.

1           Also, the recall is just not as reliable as it is  
2 for spousal smoking, especially by the proxy respondents.

3           Workplace ETS exposure is certainly less stable  
4 than the home because people change jobs and offices, their  
5 co-workers change, the workers may not know if they're  
6 exposed since ETS can come through air circulating systems  
7 and other chemicals could even mask the smell of ETS.

8           Furthermore, high levels of hazardous chemicals in  
9 the workplace can create lung cancer risk and maybe because  
10 of these problems only two studies even published a response  
11 by workplace exposure level.

12           Next, please, slide 15.

13           (Slide 15)

14           DR. BAYARD: By the time we went out, just staying  
15 on this idea of amount of information with workplace and  
16 childhood exposure, by the time we went out with our first  
17 draft in 1990, this is the information that was available to  
18 us and this is in explanation of why we stuck with spousal  
19 studies because we wanted as large a database as we could  
20 possibly have and as uniform a database as we could possibly  
21 have.

22           We had 24 studies in 1990, May 1990, available to  
23 us. Of those 24 studies, all those 24 studies had spousal  
24 smoking. Some of them also had workplace. In fact, I think  
25 eight of them had information on workplace exposure, eight

1 of them had information on childhood exposure. I'm sorry,  
2 it was seven and seven. These were for females. For males,  
3 there were eight studies and about two of them or three of  
4 them had information on workplace exposure, not on  
5 childhood. For the U.S. males, U.S. females, eight of them  
6 had spousal, four of them had workplace and two, I guess,  
7 had childhood exposures. And there were far fewer for the  
8 U.S. males.

9 Even more significant than the number of studies,  
10 of the total number of cases, 516. For spousal, there were  
11 2053 cases total. For the workplace, there were 456 cases  
12 on females. For childhood, there were 740 cases. You can  
13 see that there were far fewer cases both for male spousal  
14 and male workplace and in general the picture here is if you  
15 want a homogeneous data base, pick your largest data set, it  
16 tends to be female never smokers married and look at the  
17 history of the husband.

18 (Slide 17)

19 DR. BAYARD: And slide 17 merely gives the numbers  
20 that you see in slides 15 and 16.

21 So the story that I want you to get from this is  
22 that we chose the biggest and the strongest database which  
23 we felt would allow us to determine whether or not there was  
24 an effect from typical environmental levels to environmental  
25 tobacco smoke.

1 (Slide 18)

2 DR. BAYARD: Slide 18 just is an update on this.  
3 It shows the number of studies available to us as of May  
4 1992 which was our cutoff date for literature review and the  
5 day we went out, the time we went out with our second review  
6 draft.

7 Again, we had 30 studies and the spouse which had  
8 some -- nine in the male and nine in female in the workplace  
9 and two male in the workplace, we actually had a little bit  
10 more information on childhood.

11 Interestingly, more studies now are including  
12 workplace and childhood and socials and we're getting better  
13 pictures of total human exposure and we'll see that later.  
14 However, for our report, there just wasn't enough  
15 information, we felt, to provide a good story on what's  
16 going on either in the workplace or the male or childhood.

17 Number 19, please.

18 (Slide 19)

19 DR. BAYARD: Anyway, so for hazard identification  
20 analysis, this is what we did. We accumulated this database  
21 and we adjusted every one of the studies first before we did  
22 anything else for potential smoker misclassification. Every  
23 one of them, okay? So every one is already adjusted for the  
24 one potential source of downward bias.

25 Then we did five analyses of individual studies,

1 five separate analyses. We did the ever versus never  
2 exposed. We had 30 studies, all 30 studies. Then we looked  
3 at the highest exposure group. If anything's going to show  
4 an effect, it's going to be the highest exposure group. I  
5 can't imagine anyone getting up before you and giving you an  
6 analysis of the environmental tobacco smoke data and not  
7 showing a highest exposure group analysis, okay?

8           You can't do it with the workplace because you  
9 just don't have enough dose level studies in the workplace  
10 and there's probably a reason for that.

11           Dose response trends. Of the 17 studies that  
12 provided information by level, 14 of them provided us enough  
13 information for dose response trend tests.

14           An analysis of pooled data, then we then tried  
15 pooling the 30 studies. We found that the studies were  
16 different by countries, that when you put them together, you  
17 put the odds ratios together, you have heterogeneity between  
18 countries and we found that -- we grouped the studies by six  
19 countries and we'll get into that in a short time.

20           We also pooled the studies by country with quality  
21 ranking procedures and that is that size of a study is one  
22 measure but some studies are just better than others. Some  
23 studies are really designed to study environmental tobacco  
24 smoke, some studies just stuck it in as a question or two  
25 when they were studying other indoor air pollutants and

1 they're just not as good. So we did what we could to  
2 provide a qualitative ranking scheme and we then evaluated  
3 the studies within countries and by quality ranking tiers.

4 We also did whatever examination we could for  
5 potential confounders.

6 Next, please, 20.

7 (Slide 20)

8 DR. BAYARD: On the analysis of individual  
9 studies, again, we have the exposed versus the unexposed.  
10 this is the next three groupings I'm going to show you for  
11 these next three data sets, I'm sorry, I'm going to show  
12 you. For exposed versus unexposed, the highest exposure  
13 group, and the exposure response trends.

14 (Slide 21)

15 DR. BAYARD: Slide 21. Slides 21 and 22, leave  
16 21, please --

17 (Slide 22)

18 DR. BAYARD: -- present the accumulation of the  
19 summary data and some tests by individual study, by country.  
20 We did -- you'll have all 30 studies here. Column three  
21 represents the relative weight within that country for the  
22 pooling within country. Column four represents the power of  
23 or the ability of that study to determine a 50 percent  
24 increase in risk at a 5 percent level.

25 You'll notice, or at least the statisticians

1 should know, that the power in most of these studies was  
2 quite small. OSHA in it's 1980 policy, I think, always  
3 wanted to have at least an 80 percent power of detecting an  
4 effect before it would consider a study properly negative.

5 You can see that very few of these studies have  
6 anything like that power. In fact, I see only two of them,  
7 it should be three, Fontham and Garfinkel and Wu-Williams,  
8 were the only studies large enough to have any kind of  
9 power.

10 Anyway, out of these 30 studies, 24 of them had  
11 increased overall odds ratios, relative risk estimates for  
12 the ever versus never exposed. Now, remember, this ever  
13 versus never exposures is a crude rate because it contains  
14 all that exposure misclassification because you don't have a  
15 clean control group and because of your background.

16 Now, we've also done trend tests. Of these 24 out  
17 of 30 studies, none of them of were statistically  
18 significant with a one tail test at the 5 percent level.  
19 Now, that may not sound like much but, again, the studies  
20 have very low power in general and if there is no effect at  
21 best at the 5 percent level of significance, we should have  
22 only seen one and a half significant studies, one and a half  
23 out of 30. But now we've seen nine.

24 That's what I wanted to tell you in these two  
25 slides.

1 Slide 23, please?

2 (Slide 23)

3 DR. BAYARD: Slide 23 presents the relative risk  
4 estimates for the highest exposure groups. At 1992, we had  
5 17 studies that had information on highest exposure groups.  
6 This slide happens to have three more, three of them that  
7 have come out more recently: Liu 1993, Brownson 1992,  
8 Fontham 1994 and it should have Stockwell in there,  
9 Stockwell 1992. The Fontham 1994 study substituted for the  
10 Fontham 1991 study.

11 The point here is that every one of these high  
12 exposure groups is already corrected for smoker  
13 misclassification. Every one of them is increased, I think,  
14 13 of them, 13 of the 20 are statistically significant,  
15 despite very small sizes, very low power.

16 That's my message there.

17 (Slide 24)

18 DR. BAYARD: On slide 24, I'm actually showing you  
19 what the power is and what the relative risks are for each  
20 one of these studies.

21 Again, the picture is emerging. You're seeing  
22 overall increases, you're seeing especially the highest  
23 exposure group increased and now we're going to talk about  
24 trend tests.

25 (Slide 25)

1 DR. BAYARD: Slide 25 -- I'm sorry, could we go  
2 back, please?

3 (Slide 24)

4 DR. BAYARD: There's some more information in  
5 slide 24 that I'd like everyone to know. At the highest  
6 exposure levels, these risks start getting pretty high by  
7 certain standards. For example, the median risk of these 20  
8 studies I think is about two on these, you can count it  
9 yourself, it's either 1.9 or 2.1, I forget. So the median  
10 increase at the highest level is about 100 percent. The  
11 average increase is about 70 or 80 percent.

12 But it's significant to know that when you have an  
13 increase this high then you have to start wondering what  
14 type of potential confounders or biases could explain this.

15 Again, you also have different studies on  
16 different countries. Some of them might be controlled for  
17 some potential confounders and not for others but when you  
18 start getting your increase this high in your highest  
19 exposure group, it becomes very difficult to explain by any  
20 confounders or biases.

21 Go ahead, please. Next.

22 (Slide 25)

23 DR. BAYARD: Then we get into the trend analyses.  
24 This one happens to be the Fontham 1994 analysis. It isn't  
25 the one we did with our paper but it's my way of moving from

1 1992 into 1995. And it's the largest U.S. study and, again,  
2 it just starts showing what happens when your dose  
3 increases.

4 Now, most of the high exposure information we have  
5 and most of the trend tests we have are based on intensity.  
6 Fontham happened to do hers on pack years of exposure,  
7 number of packs per day times the number of years of  
8 exposure. And the message that I want to bring here is that  
9 at the highest exposure levels you see what happens to the  
10 risk at 80 pack years, which is probably two packs a day for  
11 40 years or something like that. You're increasing to  
12 about -- it's 79 point -- the relative risk is 1.79 on that.  
13 You go down to 40 to 79 years and you have about 1.36.

14 Individually, only the highest exposure group is  
15 statistically significant at the 5 percent one tail test and  
16 it's not quite statistically significant at the two tail  
17 test. It's very, very close. But the P trend is  
18 statistically significant either by a one tail or a two tail  
19 test, the test for exposure response trend, that's P equals  
20 .015.

21 Next, please.

22 MS. SHERMAN: Dr. Bayard, could you read into the  
23 record the number of the slide so the transcript will be  
24 more understandable?

25 DR. BAYARD: Sure.

1           Would you like me to take some questions on the  
2 particular slides now?

3           JUDGE VITTON: No. I'd like for you to just go  
4 through all of them.

5           DR. BAYARD: Okay. Okay.

6           (Slide 26)

7           (Slide 27)

8           (Slide 28)

9           DR. BAYARD: Now, what we then did was take the  
10 exposure response trends for all the data that we had and  
11 what you'll see in slides 26, 27 and 28 are all the data and  
12 what I want you to notice about these data on the fifth  
13 column is how the relative risk estimates typically increase  
14 as the exposure goes up.

15           Now, a lot of these are by intensity but the point  
16 being that you're getting 14 -- I'm sorry, there are 17  
17 studies, 14 of them have enough information to determine the  
18 significance of trends. All the trends were positive. Of  
19 the 14 that enough information for testing, 10 of them were  
20 statistically significant.

21           At the 5 percent level, the probability of getting  
22 10 out of 14 significant trend tests, if there's no effect,  
23 if infinitesimal. It borders on something less than one in  
24 a billion. But it's a matter of continuously looking at the  
25 data and watching what happens there. It's not that you get

1 one small pooled relative risk of 1.19, it's just that all  
2 the data together are telling you a consistent story here.)

3 Next, please.

4 (Slide 29)

5 DR. BAYARD: We are now into slide 29.

6 Now, because different authors did different trend  
7 tests differently, we categorized them by intensity,  
8 duration and cumulative pack years. Most of the tests have  
9 been done by intensity but what I want you to know here, my  
10 message here, is that when you have significant trends by  
11 intensity it's awfully difficult to try to figure out a  
12 potential confounder which can explain these results.

13 We actually tried to find one. The one that  
14 really stuck out at us was radon and we thought that -- this  
15 was several year ago, we thought that if anything was going  
16 to have an effect with intensity of exposure, it's going to  
17 be radon and the reason was this: the radon particles in  
18 the home might tend to adhere to the particulates in the  
19 smoke so that when the smoke actually got into the lung the  
20 residence time, it stayed longer. They had longer residence  
21 time and therefore you might have an interaction effect  
22 between the radon and the particulates in the smoke.

23 We tried modeling this. It never worked out. We  
24 did the best we could. We then looked at the epidemiology  
25 studies that we could find on radon and environmental

1 tobacco smoke, there are only two. At the time, there was  
2 only one and that was Pershagen, the Swedish study, who  
3 found an effect for environmental tobacco smoke and I think  
4 a somewhat smaller effect for radon and I don't think he  
5 found much of an interaction, if any interaction effect.  
6 But there was nothing else that we could imagine that would  
7 have caused a dose response with intensity like that.

8 Now, some of the potential confounders could  
9 possibly have an impact on prevalence but you can't -- if  
10 you people have an explanation for it, I would love to hear  
11 it but I just haven't heard one yet.

12 Next, please.

13 (Slide 30)

14 DR. BAYARD: We then analyzed the studies pooled  
15 by country and, as I said before, we have six country  
16 groupings and then we have the qualitative ranking within  
17 country.

18 Next, please.

19 (Slide 31)

20 DR. BAYARD: Slide number 31. In slide 31, you  
21 start seeing the overall relative risks by country pooling.

22 For Greece, based on two studies, the relative  
23 risk was 2.01. For Hong Kong, it was 1.48. Japan was also  
24 highly statistically significant, 1.41 based on five  
25 studies. The USA was barely statistically significant based

1 on 11 studies at 1.19. Western Europe was far less powered,  
2 it had the same -- in four studies, it had the same overall  
3 relative risk as the U.S., 1.17. This is for pooled ever  
4 versus never now. It's a crude measure. China had four  
5 studies and it was actually below one.

6           However, when we then tried to do a qualitative  
7 ranking on these studies, we ranked three of the Chinese  
8 studies very low because they were not designed to study  
9 passive smoking, they were designed to study other indoor  
10 air pollutants. And so the results changed a little.

11           Slide 32, please.

12           (Slide 32)

13           DR. BAYARD: Well, we used several ranking factors  
14 and we did a tiering scheme. We don't wave our flag on it  
15 but we felt it was worth an effort to try to rank these  
16 studies by some qualitative measures. And then what we did  
17 was in country we did odds ratios and we did pooling by  
18 sequential tiering. So, for example, in Greece, Kalandidi  
19 had a tier one ranking as a good study, we felt, it had an  
20 overall relative risk of 1.92, it was significant at the .02  
21 level. There were no studies in tier two but Trichopoulos  
22 was a tier three study. He had a relative risk of -- I  
23 forget what it was but when you pooled Trichopoulos and  
24 Kalandidi you had a pooled relative risk of 2.01 and a P  
25 value of .0005, so it was highly statistically significant.

1 And that was the methodology that we used for all the  
2 countries. I just wanted to explain that.

3 Thank you.

4 Thirty-three.

5 (Slide 33)

6 DR. BAYARD: So what's our story from all these  
7 analyses?

8 We looked at all the confounding factors that we  
9 could and that's all in there. We looked at six of them and  
10 we found that they couldn't explain the results. We  
11 examined home heating sources, that's at the bottom there.  
12 We examined cooking with oil. We examined for lung  
13 diseases. I think there were nine studies that did a  
14 control on diet, several on occupation. And we found no  
15 consistent results which could explain the story that I've  
16 been telling you, the picture that emerges, the dose  
17 response trends, the consistent increases, and the fact that  
18 you're seeing them from all different countries. And that  
19 all argues against that any one confounder would do it.

20 So what do we have?

21 In test for effect, exposed versus unexposed, 24  
22 of 30 studies found an increased risk and nine of the 30  
23 were statistically significant. The probability of getting  
24 that was less than 1 in 10,000, of getting nine out of 30  
25 statistically significant results.

1           The test for effect in the highest exposure group,  
2 all 17 studies with exposure level data found an increased  
3 risk in the highest exposure group. Nine out of 17 were  
4 statistically significant.

5           Trend tests, 10 out of 14 with sufficient data for  
6 a trend test showed a statistically significant response  
7 relationship. The probability of that happening is less  
8 than 1 out of a billion.

9           Meta-analyses, four of the six country groupings  
10 including the U.S. had statistically significant pooled risk  
11 estimates.

12           Qualitative ranking, all six country groupings  
13 showed an increased risk when the qualitative considerations  
14 were taken into account. All results were previously  
15 adjusted for the only known source of upward bias which is  
16 the potential smoker misclassification status.

17           We concluded that environmental tobacco smoke is a  
18 known human carcinogen.

19           Slide 34, please.

20           (Slide 34)

21           DR. BAYARD: Slide 34 merely puts all that weight  
22 of evidence back together. The epidemiology studies on the  
23 right, the fact that active smoking causes lung cancer with  
24 no evidence of a threshold, the fact that even that you see  
25 increased lung cancer risks with pipe and tobacco in the

1 order of two and three where you don't have inhalation, you  
2 merely have the passive smoke from that.

3 ETS and active smoke both contain the same 40  
4 carcinogens. Documented exposure in everyday environments  
5 and supporting evidence from animal studies and genetic  
6 tests.

7 That's our conclusion on the hazard  
8 identification. However, I would like to bring in now the  
9 several recent studies all of which we believe have some  
10 support for our conclusions.

11 (Slide 35)

12 DR. BAYARD: Trichopoulos, JAMA 1992, did a lung  
13 autopsy study of women, this is slide 35, who did not die of  
14 lung cancer. It was a study of epithelial possibly  
15 pre-cancerous lung lesions. It wasn't a very live study of  
16 these, however, he did suggest that non-smoking women  
17 married to smokers had significantly higher possibly  
18 pre-cancerous lung lesions. It's a different type of  
19 design, still found among non-smoking women married to  
20 smokers that they had more possibly pre-cancerous lung  
21 lesions.

22 Reif did an epidemiology study on dogs with 51  
23 cases and 83 controls. Overall, he found a 60 percent  
24 overall increase in canine lung cancer with one or more  
25 smokers in the home. It was not statistically significant.

1 He found a 140 increase for short and medium nosed dogs.  
2 Not statistically significant.

3 Wang, International Journal of Epidemiology, 1994,  
4 a small Chinese study of ETS and lung cancer, found that  
5 household ETS exposure significantly increased the risk by  
6 250 percent for non-smoking women if they were exposed at  
7 ages 14 or less.

8 Liu, American Journal of Epidemiology, 1993, was a  
9 case controlled study of home indoor air pollution and lung  
10 cancer in China. Thirty-eight never smoking female cases,  
11 69 controls. He found statistically significant increased  
12 risk of lung cancer from spousal smoking in the highest  
13 exposure group with an odds ratio of 2.9 and a statistically  
14 significant exposure response trend, P equals 0.0342, cited.

15 I brought up those studies but the next three  
16 studies which I will bring up are probably much more  
17 pertinent to OSHA's analysis. They're much larger studies,  
18 they're U.S. studies.

19 (Slide 36)

20 DR. BAYARD: Stockwell, 1992, JNCI, 210 never  
21 smoking female lung cancer cases, 301 controls. Overall,  
22 ever versus never exposed to spousal smoking, there was a 60  
23 percent increase in relative risk. Significant exposure  
24 response trends for household exposure to ETS for both  
25 adulthood and all lifetime household exposure. Significant

1 increases in relative risk at the highest exposure levels  
2 for both childhood, OR equals 2.4 in adulthood, OR equals  
3 2.4 households. These are statistically significant.  
4 However, they found no statistically significant increases  
5 in risk from exposure at work or during social activities.  
6 Data not shown, that's their quote.

7 MS. SHERMAN: That was slide 34, Dr. Bayard?

8 DR. BAYARD: Thirty-six.

9 MS. SHERMAN: Thirty-six.

10 (Slide 37)

11 DR. BAYARD: Slide 37, another large study,  
12 Brownson et al., American Journal of Public Health, November  
13 1992, 431 never smoking female lung cancer cases, 1164  
14 controls. No increased risk in ever versus never exposed to  
15 spousal smoking. Spousal smoking, no increased risk.  
16 However, he found a 30 percent statistically significant  
17 increase in the relative risk from spousal smoking in the  
18 highest exposure group.

19 No increased risk observed for childhood exposures  
20 using a quantitative exposure, however, he found a  
21 statistically significant increase in subjects reporting a  
22 moderate exposure, odds ratio equals 1.7, in the heavy  
23 exposure, odds ratio equals 2.4 based on childhood exposure.

24 He found no increased risk in ever versus never  
25 exposed to ETS in the workplace. However, he found an

1 increased risk of about 30 percent in the two highest  
2 workplace exposure quartiles. This is unpublished data of  
3 Brownson submitted to OSHA by Butler, 1994. We'll get into  
4 this in a minute.

5 Slide 38.

6 (Slide 38)

7 DR. BAYARD: Slide 38 should be broken into two  
8 parts on your overheads, 38A and 38B, but those people who  
9 have the handouts will find that it's in one slide.

10 The largest and what we feel is the best designed  
11 and conducted U.S. study, Elizabeth Fontham, 1994, Journal  
12 of the American Medical Association, "Environmental Tobacco  
13 Smoke and Lung Cancer in Non-Smoking Women," Fontham et al.  
14 Largest case controlled study, U.S. or otherwise, 653 cases,  
15 1253 controls, five U.S. cities, Atlanta, Houston, Los  
16 Angeles, New Orleans, San Francisco. Los Angeles and San  
17 Francisco I think comprise about 80 percent of the final  
18 sample, less of the original three-year study in 1991.

19 Most carefully designed and run study specifically  
20 conducted for ETS and lung cancer in never smoking women.  
21 Controls for most potential confounders, including diet,  
22 other ETS exposures, age, race, education, study area,  
23 family history of lung cancer and high lung cancer risk  
24 occupations.

25 This completes the final two years of accrual of

1 the five-year National Cancer Institute funded study. EPA's  
2 risk assessment reported on the first three years.

3 Results. Very similar to earlier study findings  
4 with results achieving higher degree of statistical  
5 significance because of increased number of subjects.  
6 Highly consistent with EPA's conclusions. The study finds  
7 exposure to ETS during adult life increases the risk of lung  
8 cancer in lifetime non-smokers.

9 Overall increased risk is about 30 percent for  
10 spousal smokers smoking, 40 percent for occupational  
11 exposures, 50 percent for social exposures. All three  
12 increases are statistically significant by either a one tail  
13 or two tail test. Exposure response trends are also  
14 statistically significant.

15 Largest increase is seen for exposure index for  
16 combined exposure, which includes household, occupational  
17 and social exposures, about 75 percent higher at the higher  
18 levels.

19 (Slide 38)

20 DR. BAYARD: Now, the study didn't find an  
21 increased risk associated with childhood exposure by itself  
22 but it did find increased risks for women exposed -- this  
23 would be slide 38 and 39, we're moving into 39.

24 (Slide 39)

25 DR. BAYARD: Now, if you just look at childhood

1 exposure by itself, you don't find any increased risk but if  
2 you look at both childhood and adult exposure -- I'm sorry,  
3 if you compare those who were exposed both during childhood  
4 and adult with those exposed during adulthood only, you do  
5 find an increased risk and we think this is significant  
6 because it's similar to active smoking with the earlier the  
7 starting age the higher the risks.

8 Now, this slide was prepared by a colleague of  
9 mine, Dr. Dorfland, and he kindly lent it to me for display.  
10 But it shows, the lower line shows -- I'm sorry, the axis,  
11 the Y axis is the relative risk, slide 39, the X axis is  
12 smoke years as an adult.

13 If you weren't exposed during childhood, you get  
14 the little hollow circles. There's not much of an increase,  
15 depending on what your amount of smoke years as an adult  
16 was. There's a small amount. But if you were exposed  
17 during childhood, according to her data, and then exposed as  
18 an adult, the increase is significant. So there seems to be  
19 an effect of childhood exposure, a large effect of childhood  
20 exposure, if you were exposed as an adult.

21 Next, please.

22 (Slide 40)

23 DR. BAYARD: Well, let's put this new data  
24 together and see what they're trying to tell us. Slide 40.

25 It turns out that slide 40 will show us the

1 results for spousal smoking results of most recent U.S.  
2 studies on ETS and lung cancer in never smoking women. I've  
3 also -- I've put in Fontham '94 and '91. Now, they're not  
4 independent studies, of course, but I wanted to put them in  
5 so that you could compare them. Brownson, '29; Stockwell,  
6 '92; and Janerich, 1990. Those are the most recent U.S.  
7 studies.

8           The reason I'm dealing with U.S. studies is  
9 because I think they're more pertinent to what OSHA is  
10 dealing with here.

11           Now, I think for the spousal smoking, for the  
12 spousal component here, whether or not it's a proxy or  
13 whether or not it's direct, there seems to be fairly good  
14 reliability, especially if the proxy is the next of kin,  
15 it's the husband. But I just put it in so that you'll get  
16 the figures here.

17           Now, Fontham, 1994, had 653 cases; Brownson column  
18 two for 1992 had 431 cases. And you look at the overall  
19 relative risks down that column. Fontham's overall relative  
20 risk was the same, 1.29, for both studies. The relative  
21 risk in the highest exposure group actually went up in 1994  
22 but I don't think varied statistically significantly from  
23 the 1991.

24           The P-trend tests for exposure are statistically  
25 significant in both studies.

1           Brownson found no increased risk but he did find  
2           an increased risk in the highest exposure group, 1.3.  
3           Stockwell, 1992, found an overall increased risk of 1.6 but  
4           when you limit that to the direct only, there were 70 cases  
5           of direct and then the increased risk actually goes up to  
6           3.1 and we'll look at this in the next slide when we get to  
7           it in just a minute.

8           The highest exposure group in the direct only for  
9           Stockwell had an increased risk of 4.7. That is -- you'll  
10          see this in just a minute. Both were significant trend  
11          tests.

12          Janerich was a slightly different study because  
13          they used individual matched case controlled studies, so he  
14          actually had -- while he had 68 percent direct interviews,  
15          when you look at the 129 direct interview cases, you get an  
16          overall relative risk of .93, no increase, although there is  
17          a very slight increase in the highest exposure group. But  
18          when you look at proxies, the results actually fall apart.  
19          The overall relative risk goes down to .44 and the relative  
20          risk in the highest exposure group actually goes down to .2  
21          or a five-fold reduction and the P-trend is negative.  
22          Janerich himself comments on it.

23          The message that I'm trying to get across here or  
24          at least some thought provoking for OSHA is that proxy can  
25          have quite an effect, if not directly in spousal studies

1 then look what happens in childhood studies.

2 Look on slide 41.

3 (Slide 41)

4 DR. BAYARD: Now, slide 41 presents columns by  
5 self, proxy -- now, these are for case patient reviews, by  
6 self, husband proxy or other proxy. Remember the Stockwell  
7 study had 70 direct interviews for the cases and for  
8 adulthood those risks based on amount of smoke years were  
9 3.4, 3.6 and 4.7. For husbands, when they asked the husband  
10 based on spousal, it was 3.1, 1.8, 4.2, pretty much the same  
11 results. But when you asked another proxy, look what  
12 happens to the relative risks. They go way down: .8, .8,  
13 1.5. Your trend test falls apart. Again, it's a matter of  
14 being careful about who the other proxy is and all studies  
15 are not created the same and within studies not everything  
16 is the same.

17 Now, with childhood adolescence, watch what  
18 happens. Watch how much more quickly it falls apart. When  
19 you do the self-interview, you get 4.3, 2.4 and 6.5. Those  
20 are pretty high relative risks, with a trend test of .04.  
21 When you ask the husband, though, about what the wife's  
22 exposure was as a child, apparently he doesn't know that  
23 well because the risks seem to be falling apart here: 2.4,  
24 1.6 and 1.4. Then when you ask another proxy, the risks go  
25 way down to .6, .6 and 1.6. Okay. Now, that's childhood

1 exposure study.

2 Slide 42.

3 (Slide 42)

4 DR. BAYARD: What's happening in the workplace?  
5 We've made the claim that the workplace exposure is fairly  
6 unreliable even when you ask the direct interview. If  
7 you're going to ask husbands about what their wife's  
8 exposure is in the workplace, you're going to have to kind  
9 of imagine yourself how good a measure that is. I have  
10 trouble remembering myself what my own exposure was in my  
11 workplace five years ago, six years ago because that's when  
12 I started on passive smoke.

13 Now, what happens? In the workplace, the Fontham  
14 study with direct interviews, 63 percent and 66 percent  
15 direct interviews, was finding an increased risk, overall  
16 risk of 1.39 and 1.34. In the highest exposure groups, they  
17 were 1.86 and 1.3 with significant trends.

18 The Brownson study and the Stockwell study found  
19 no increases overall in the workplace but, again, their  
20 proxies were much, much higher. They had twice as many --  
21 twice as high a proxy rate. Stated in a complimentary way,  
22 they had one-third -- on-third of their interviews were  
23 direct versus two-thirds case interviews direct for the  
24 Fontham study. You have to look and see how significant  
25 these proxy interviews are, especially in workplace studies.

1 That's my message here.

2 Janerich, on the other hand, with 68 percent  
3 proxy, those 129 cases, they're all direct interviews. So  
4 here was a group in which he still found no adverse effects,  
5 even with direct interviews. I don't think the story is  
6 airtight. I'm presenting the data as I see them and a lot  
7 of the analysis remains to be done on this.

8 However -- slide 43.

9 (Slide 43)

10 DR. BAYARD: I would still make the claim that the  
11 Fontham study is still the best study to use even for the  
12 workplace. However, when you do use it for the workplace, I  
13 want you to make sure you decide what your proper study  
14 population is. Here is some data from Brownson submitted by  
15 Dr. Butler to you and I'm bringing it up to you again  
16 because I think it's worth a second look.

17 Now, the problem is who do you -- which group do  
18 you accept as your truly unexposed group?

19 Now, Butler did it two ways. He asked Dr.  
20 Brownson for his data and Dr. Brownson gave him his data and  
21 Butler presented the data to OSHA in two different ways.  
22 When you take all never smoking women, that includes women  
23 who worked and women who didn't work. And then you had 238  
24 cases and 710 controls who say they were never exposed to  
25 ETS in the workplace. But some of those who say they were

1 never exposed to ETS in the workplace didn't even work so  
2 they weren't exposed to ETS in the workplace. When you  
3 remove those 238 minus 151, 87, people who just didn't work  
4 for outside the home for more than six months, then your  
5 estimates change in the second row.

6 It turns out that your risk estimates increase  
7 when you exclude those women who didn't work outside the  
8 home. The message here is to measure the effect of  
9 occupational ETS exposure restrict the analysis to subjects  
10 with a history of employment outside the home. This is  
11 something Fontham did not do. If you are going to take the  
12 Fontham measure, my suggestion to you is that measure has to  
13 be adjusted upward, either it has to be adjusted upward,  
14 that risk, or you have to ask Dr. Fontham to redo an  
15 analysis using only women exposed outside the home, because  
16 otherwise the non-employed subjects could have greater  
17 exposure misclassification than the employed subjects as a  
18 result of being unexposed during 40 hours a week. You know  
19 they weren't being exposed at work, they were probably  
20 somewhere else being exposed and they're being treated as  
21 unexposed. It's a similar problem, it's a smoker  
22 misclassification, smoker exposure -- it's an exposure  
23 misclassification problem but it relates to a problem  
24 specifically for workplace exposure.

25 That's my message on hazard identification and on

1 the new studies. However, I also have a comparison -- and  
2 this will be rather short, I hope, of quantitative estimates  
3 of population risk.

4 Slide 44.

5 (Slide 44)

6 DR. BAYARD: Again, getting -- let me just take a  
7 minute.

8 (Pause)

9 DR. BAYARD: Now, getting back to the question of  
10 trying to derive population estimates, this is what EPA did,  
11 in deriving population estimates, now that you've decided,  
12 we've decided, that environmental tobacco smoke is a known  
13 human carcinogen, we have to clean up the control group, we  
14 have to adjust for the fact that our risk estimate was not  
15 high enough because it was driven down by a control group or  
16 a non-exposed group that was exposed.

17 This is shown in some extent in slide 45.

18 (Slide 45)

19 DR. BAYARD: Data from the IARC ten country  
20 collaborative study, this is by Riboli et al., 1990, I  
21 believe it is, 1369 subjects, ten different cities -- excuse  
22 me, 13 cities, ten countries.

23 The Y axis shows the level of urinary cotinine  
24 which is we consider a good marker for environmental tobacco  
25 smoke exposure by source of exposure. It turns out that

1 women who said they were not exposed at home and not exposed  
2 in the workplace still had urinary cotinine levels of 3.1  
3 nanograms per milligram. Women who said they were exposed  
4 at work but not at home had levels of 4.5. In the home, the  
5 levels went up quite a bit, 9.5 if they were exposed at home  
6 and not at work, 10.1 if they were exposed at work and not  
7 at home.

8 I don't mean to present these data to you as the  
9 be all and end all. There were 13 different cities. I do  
10 mean to show you that there is a background exposure of  
11 women who say they were not exposed. They are getting  
12 exposure some place and that has to be correctable.

13 JUDGE VITTONI: Excuse me, Dr. Bayard.

14 DR. BAYARD: Yes?

15 JUDGE VITTONI: Your last comment about the column  
16 to the right, 10.1?

17 DR. BAYARD: Yes.

18 JUDGE VITTONI: They were exposed at work and  
19 home?

20 DR. BAYARD: Yes, sir.

21 JUDGE VITTONI: Okay.

22 DR. BAYARD: That's saying yes, they were exposed  
23 at work and at home.

24 JUDGE VITTONI: And 9.5 if they were just exposed  
25 at home.

1 DR. BAYARD: And no work. That's correct.

2 You're following this, huh?

3 Well, in order to correct for background exposure,  
4 we used the model of the National Research Council and then  
5 expanded from there. What we had was the relative risk as  
6 observed, which was the risk to the quote exposed group over  
7 the risk to the quote unexposed group.

8 We then had to clean up that exposed group by  
9 getting rid of that -- by making the risk relative to people  
10 who were truly unexposed. In order to do that, the formula  
11 down there, we had to find an estimate of what we call Z.  
12 And what we call Z is the ratio between the mean dose level  
13 in the exposed group and the mean dose level in the  
14 unexposed group.

15 Once we could do that, slide 47 --

16 (Slide 47)

17 DR. BAYARD: We could make the proper adjustments.  
18 In our chapter six for lung cancer, you will find different  
19 ways in which we did that. And, in fact, we did our  
20 analyses three different ways.

21 Number one, we used -- this is slide 47, please.

22 We used the combined U.S. studies, 11 U.S. studies  
23 and when we used the combined U.S. studies, we took the  
24 information we could find on population surveys much like  
25 the 13-country IARC study that I just presented but for the

1 U.S. data only. And found the amount of exposure in women  
2 exposed at home versus women not exposed at home.

3 It turns out with a Z equal 1.75, for example,  
4 we're saying for every four parts of exposure or for every  
5 one part of exposure that a woman is getting outside the  
6 home, they're getting 1.75 parts inside the home.

7 Now, so we did our adjusted analyses, adjusted  
8 risk estimates, for both the 11 U.S. studies combined and  
9 for Fontham, which we felt was the best study at the time.

10 Now, these are not independent estimates and I  
11 don't mean to imply that they are because Fontham is one of  
12 the 11 U.S. studies and, I think, provides 35 percent of its  
13 weight. So the 1.19 pooled estimate includes the 1.28 from  
14 Fontham.

15 However, what Fontham had, which is what no other  
16 study had, was a sample of the women controls in her own  
17 study in which we actually had urinary cotinine levels for  
18 home versus non-home exposure. So we called Dr. Fontham, we  
19 asked her if she would provide us with a summary of those  
20 data, which she did. She provided us the means and the  
21 medians and the standards errors. We didn't ask for the  
22 individual data but we did ask for the summary. We asked  
23 her if we could use them and she gave us permission to use  
24 them, so the data are available. The data that we have are  
25 available to everyone. We always do that.

1           Now, the data that Fontham gave us actually had a  
2 mean cotinine level of 2 and a median of 2.6. We used them  
3 both. We suspected that the data are probably log normal in  
4 which case we probably would have used more of a median but  
5 we used the 2.0 or 2.6 and those are -- what you see are the  
6 differences in risk estimates that we get.

7           So I'll just read down, and I'll read down for the  
8 11 combined studies and try to tell you what they mean.

9           The 1.19 was the pooled relative risk that we got  
10 from 11 U.S. studies. When you clean up and say what's the  
11 risk of these women exposed at home after you have adjusted  
12 for background, assuming that you're comparing those to  
13 women with no exposure at all, then that risk jumps to 1.59.

14           The other calculation you can say is what's the  
15 risk of women who aren't exposed at home and they're just  
16 exposed in the background? Our background, I have always  
17 said, is just walking around but it isn't just walking  
18 around, it's workplace and social and other, okay?

19           So we've estimated that risk from 11 U.S. studies  
20 as increased by 34 percent. And that varies depending on  
21 the observed relative risk and the estimate of the cotinine  
22 ratio.

23           Next, please.

24           (Slide 48)

25           DR. BAYARD: Slide 48. So these are the estimates

1 that we get. Now, this happens to be the estimates for the  
2 11 U.S. studies and the rows by smoking status, non-smoking  
3 female, that's NS, non-smoking female, one is not exposed to  
4 spousal smoking, one is exposed to spousal smoke, and we did  
5 the same for males. We got the numbers of population at  
6 risk. And then we got former smokers.

7 Now, we only got former smokers who had quit at  
8 least five years. We figured after five years they're  
9 probably still going to have increased risks but we only  
10 tried to add on the increased risks -- we only used that for  
11 a population base so that we would add on the increased  
12 risks due to passive smoking. So the increased numbers that  
13 you're seeing for former smokers relate only to the actual  
14 increase due to passive smoking, not to increases that would  
15 be predicted to be due to their former smoking habits.

16 In any event, the results we got from the 69  
17 million at risk, bottom row, please, the 69 million at risk  
18 ages 35 to 74, we estimated from the 11 studies 2200 were  
19 due to sources other than spousal, 860 cases or deaths would  
20 be due to spousal for a total of 3060.

21 You can also sum these up by column, in which case  
22 you have 1500 for never smoking females, 500 for never  
23 smoking males, 430 from former smoking females, 630 from  
24 former smoking males.

25 Slide 49.

1 (Slide 49)

2 DR. BAYARD: We did the same thing for Fontham.

3 In Fontham, we used both the means and the medians and I  
4 will go right to the bottom line, literally. And if you use  
5 the means, you come up with 2360 backgrounds, 1210 for  
6 spousal, for a total of 3570. For a median, these are the  
7 same data just using the mean and the median, you come up  
8 with 2670 total.

9 Now, again, these estimates are susceptible to the  
10 model. They're susceptible to the value of the cotinine  
11 ratio that you insert but the three estimates that we get,  
12 2670, 3000 and 3570, we said, well, that seems to be close  
13 enough to 3000 so we're going to say that our estimate is  
14 3000.

15 And so now we want to try to put these estimates  
16 in perspective. We've decided that exposure to passive  
17 smoke causes in the U.S. about 3000 lung cancer deaths a  
18 year. The increased relative risk may not be that large but  
19 when you take even a small relative risk and project it to a  
20 total population of 69 million exposed, then you're going to  
21 get some numbers here.

22 (Slide 50)

23 DR. BAYARD: On slide 50, you'll find that we've  
24 tried to put this into perspective.

25 Of the 127,000 deaths expected in 1985 from lung

1 cancer, we found from active smoking that 110,000 would be  
2 attributed to active smoking, 3000 would be attributed to  
3 passive smoking and that's broken down two ways, both from  
4 spousal and background and from background plus spousal.

5 The non-tobacco related causes, both active  
6 smokers and passive smokers would also suffer from cancer  
7 from non-tobacco related causes. Their additional lifetime  
8 risk is 400,000 and we expect 14,000 of those.

9 Now, one in 1000 as a background risk may not seem  
10 that high, two in 1000 may not seem that high. But for EPA  
11 an action level is anywhere from one in 10,000 to one in a  
12 million.

13 Next, please.

14 (Slide 51)

15 DR. BAYARD: Well, after we did this and I got  
16 ready to try to give a presentation to OSHA, I said, gee, I  
17 wonder how their estimates compare with ours. So Jennifer  
18 Jinot worked up this table and I'm going to tell you.

19 OSHA -- well, let's go through ours first.

20 Again, we broke ours down by home, which that's  
21 quote home because it's spousal, background which is  
22 workplace, social and others, and then methodology that we  
23 used I won't go into because it's in the table and it's also  
24 in the book. But I will go into the population at risk for  
25 background which is everyone from 35 to 74 and that's 69

1 million.

2           Of those 69 million, 35.4 would have been exposed  
3 to spousal smoking. Now, we assume that the prevalence --  
4 I'm sorry, let's go on and talk about what OSHA did now.

5           OSHA used a completely different methodology than  
6 we did and they assumed that there was 74.2 million  
7 non-smoking workers, I imagine age 20 to 65, our population  
8 was 35 to 75. But OSHA used a different methodology  
9 completely and what they did was they took the background  
10 lung cancer incidence rate from a Garfinkel study which is a  
11 cancer prevention survey and then they calculated an ETS  
12 attributable rate using the occupational relative risk  
13 estimate from Fontham 1991, which is 1.34. So they actually  
14 took a background rate from one study, which is a live  
15 study, and they said, well, you multiply that by the  
16 relative risk from the workplace by getting the proper  
17 number exposed and the amount of exposure and then you can  
18 figure out the excess lifetime risk.

19           What we did was we actually took the 38,000 lung  
20 cancer deaths and decomposed them by attributable risk from  
21 active smoking and passive smoking and non-smoking sources  
22 and other effects. So the methods bear very little  
23 semblance to each other. They both look legitimate to me.  
24 As you go along and try to calculate these things, they look  
25 like fairly decent methods.

1           However, the OSHA prevalence of exposure I thought  
2 was a low prevalence and I thought it because -- they  
3 estimate that their prevalence of exposure is 18.8 to 48.7.  
4 The prevalence that you used for the 18.8, I think, was some  
5 national health examination survey and the question had to  
6 do with were you near anyone who smoked at work in your  
7 immediate location in the last two weeks or something like  
8 that. And that appears to be on the low end from my limited  
9 knowledge of workplace smoking policies.

10           The other estimate was an estimate by Cummings of  
11 48.7 was people exposed at work but not at home. And that  
12 was 48.7. But then when Cummings asked the question are you  
13 exposed both at work and at home, there was another 29  
14 percent. So an upper limit there would have been 77  
15 percent. OSHA used 48 percent. It seems a little low to  
16 me. It just doesn't seem like the right methodology.

17           Now, however, they calculate -- so their  
18 estimates, I think, based on this methodology should  
19 probably be up a little, okay? But they calculated excess  
20 lifetime risks of between .4 per thousand and one per  
21 thousand. EPA's estimates were about one in a thousand both  
22 for home and for background.

23           But when it comes to the total LCDs per year, look  
24 at the background for EPA because that's the component that  
25 you're going to be dealing with when you're dealing with

1 OSHA's workplace exposure.

2 If you use OSHA's estimate, you're going to come  
3 out with between 740 and 1480 cases of lung cancer deaths  
4 per year in the workplace. The EPA estimate, which includes  
5 workplace and social, is 1460 to 2360 per year. So my  
6 message is that these estimates come out about the same.  
7 The methodology looks pretty good to me. It still is a  
8 little bewildering why they come out so close, but that's my  
9 message there.

10 Now, 52 --

11 (Slide 52)

12 DR. BAYARD: I'd like to get into lung function  
13 and respiratory systems in adults.

14 The EPA analysis on lung function and non-cancer  
15 respiratory symptoms and lung function, I think our cutoff  
16 date for literature review was somewhere around November  
17 1991. This is different from lung cancer where our cutoff  
18 date was about May 1992. We had different authors. We did  
19 include some studies that the SAB recommended to us in the  
20 July meeting but other than that those were the basic  
21 literature review cutoff dates.

22 Our conclusion was that passive smoking has subtle  
23 but significant effects on the respiratory health of  
24 non-smoking adults including coughing, phlegm production,  
25 chest discomfort and reduced lung function.

1           The conclusion is not a very strong conclusion.  
2           It's based on our review of the studies produced by the  
3           Surgeon General and the National Research Council in their  
4           reports of 1986 plus six new studies. And those six new  
5           studies are presented in slide 53.

6           (Slide 53)

7           DR. BAYARD: In the interests of time, I will skip  
8           those studies, if that's all right with you, Mr. Furr. Is  
9           that all right?

10          JUDGE VITTON: Go ahead, Dr. Bayard.

11          DR. BAYARD: Because those are in our report and  
12          they are fairly self-explanatory. In fact, there's more  
13          explanation in the report. But in the interests of  
14          providing information to OSHA, I would like to read into the  
15          record the information that we got from the new Medline  
16          search on many new studies which I believe very strongly  
17          support the EPA conclusions.

18          First, there's a study by Spitzer, 1990, "Link  
19          Between Passive Smoking and Disease." Actually, this study  
20          is discussed in the EPA report but it's discussed in a  
21          different section. That's a report of the Working Group on  
22          Passive Smoking, Clinical and Investigative Medicine. What  
23          they did was actually a best evidence analysis. They got  
24          the best studies they could find and they had a review  
25          procedure which was actually the model of our review

1 procedure for qualitative evidence for lung cancer. And  
2 they concluded that there is convincing evidence for an  
3 association between residential exposure to ETS in both  
4 children and adults and many chronic and acute respiratory  
5 illnesses.

6 Now, most of the references were to children, when  
7 you look at their references on this. However, all  
8 references for adults were also in the EPA report.

9 Respiratory function, they made the conclusion  
10 there is good evidence demonstrating small reductions in  
11 physiologic measures of respiratory function among both  
12 children and adults exposed to residential ETS. The  
13 respective figures from adults were six positive studies and  
14 one negative study. All seven references are in the EPA  
15 report. These are quotes from their abstracts.

16 White, Froeb and Kulik, 1991, in Chest, July,  
17 Volume 100, No. 1, pages 39 to 43, "We evaluated CO levels  
18 as an index of cigarette smoke in the workplace and analyzed  
19 diary entries on respiratory symptoms, eye irritation, chest  
20 colds and lost days from work due to respiratory illnesses  
21 in 40 passive smokers and 40 control subjects matched for  
22 age and gender. Passive smokers experienced greater CO  
23 levels during the workday. Also, they reported  
24 significantly more cough, greater phlegm production, more  
25 shortness of breath, greater eye irritation, more chest

1 colds and more days lost from work due to chest colds than  
2 control subjects."

3 JUDGE VITTON: Excuse me, Dr. Bayard. One  
4 question.

5 DR. BAYARD: Sure.

6 JUDGE VITTON: When you said you were going to  
7 read the last couple of pages, are these the pages you were  
8 talking about?

9 DR. BAYARD: Yes. Would you like me to omit them?

10 JUDGE VITTON: Well, if you're going to repeat  
11 what is here, we've got four single spaced pages.

12 DR. BAYARD: If you enter them into the record, I  
13 will be glad to omit that.

14 JUDGE VITTON: Believe me, they're going to be in  
15 the record.

16 DR. BAYARD: Thank you very much.

17 All right. Now, I will end with page 10, my  
18 comments on the methodology in the ETS sections of OSHA's  
19 proposed rule.

20 Lung cancer. Hazard identification. In general,  
21 the hazard identification section, pages 15,979 and 15,981,  
22 provide an outline and rough summary of the data but no  
23 analysis or reference to an OSHA analysis. OSHA may want to  
24 rely on earlier assessments by the Surgeon General, the peer  
25 reviewed EPA analysis with a suitable update for the new

1 studies, NIOSH or the National Research Council.

2 However, unlike EPA, a unique OSHA concern is that  
3 of worker risk. The EPA lung cancer hazard identification  
4 section focused its analysis of the ETS epidemiology studies  
5 on spousal exposure and specifically excluded workplace  
6 exposures for the reasons stated above.

7 The EPA agrees with OSHA, page 15,994, first  
8 paragraph, that if lung cancer effects as seen from home  
9 exposures, then similar exposures elsewhere should produce  
10 these same effects. Correspondingly, excess risks based on  
11 home exposures can be used as the basis to estimate excess  
12 risk from workplace exposure.

13 Nevertheless, OSHA may want to discuss the 10 to  
14 15 epidemiology studies which do address workplace exposure  
15 and lung cancer rather than just the Fontham 1994 workplace  
16 results.

17 Most of the workplace lung cancer studies do not  
18 have risks stratified by ETS exposure levels making exposure  
19 response and highest exposure level analyses impossible and  
20 nearly all are potentially confounded by spousal exposure,  
21 further complicating analysis and interpretation.

22 A key difference between EPA's report on hazard  
23 identification conclusions and those of the OSHA is whether  
24 ETS can be classified as a known human carcinogen on the  
25 basis of its similarity to mainstream smoke in known body

1 uptake of ETS.

2 The EPA report in chapter four categorizes both  
3 mainstream smoke and sidestream smoke as known human lung  
4 carcinogens and quote, in the EPA report, "Because ETS is  
5 known to be inhaled and absorbed into the body, ETS would  
6 similarly be categorized as a Group A carcinogen."

7 The OSHA report concludes only that this  
8 similarity to mainstream smoke carcinogenicity "clearly  
9 establishes the plausibility that ETS is also a human lung  
10 carcinogen."

11 We believe the OSHA may wish to strengthen this  
12 conclusion, since tobacco smoke is a complex mixture which  
13 while varying in relative composition of its components  
14 depending on burning conditions should be expected to  
15 maintain its toxic properties. Considering the chemical  
16 similarities of ETS, sidestream smoke and mainstream smoke,  
17 the established carcinogenicity of mainstream smoke and the  
18 fact that mainstream smoke is a substantial component of ETS  
19 should be sufficient to establish ETS as carcinogenic  
20 hazard.

21 Furthermore, the OSHA seems to be putting all the  
22 weight of the evidence on ETS epidemiology studies when it  
23 states, "As a first step in this quantitative risk  
24 assessment, OSHA critically reviewed epidemiology studies  
25 associating exposure to ETS with adverse health effects.

1 The purpose of such a critical evaluation was to determine  
2 whether or not exposure to ETS is a causal factor in  
3 cancer."

4 The EPA believes that the ETS epidemiology studies  
5 represent only a portion of the weight of evidence databased  
6 and that this statement should be modified.

7 Furthermore, that amount of space that is  
8 dedicated to several animal and genotox studies seems  
9 disproportionate to their stated importance in OSHA's  
10 conclusions. More space is given to those than to the 32  
11 epidemiology studies mentioned. Furthermore, the proposal  
12 suggests that the animal evidence is very strong. The EPA  
13 position is that the human evidence on lung cancer is  
14 stronger both for mainstream smoke and environmental tobacco  
15 smoke.

16 Two minor points. One, if OSHA wants to cite  
17 Coggins et al. as supporting evidence for its lung cancer  
18 classification, it should at least point out the conclusions  
19 of Coggins. However, this study did not provide evidence of  
20 all lung cancer effects. And, two, in the Reif study on  
21 lung cancer in pet dogs, only the short nosed dogs had an  
22 increased risk of lung cancer and this was not statistically  
23 significant.

24 In summary, while the weight of the evidence for  
25 human lung carcinogenicity of ETS is strong, I believe that

1 the proposed rule does not provide an adequate summary for  
2 this conclusion.

3 I understand from my discussions with OSHA staff  
4 that a more complete analysis of the weight of evidence for  
5 ETS and lung cancer is forthcoming.

6 (Pause)

7 It's the last page. OSHA's preliminary  
8 quantitative risk assessment for ETS and lung cancer  
9 presents an analysis of 32 epidemiology studies. While the  
10 table lists nearly all of the studies analyzed by EPA, the  
11 three categories of positive, equivocal positive trend and  
12 equivocal are hardly self-explanatory and leave even an  
13 experienced reader somewhat perplexed.

14 For example, most of these studies analyze the  
15 effects of adult home exposure, some analyze workplace and  
16 social adult exposures and some childhood exposures. The  
17 summary table with no numbers or explanations hardly  
18 convinces the reader that "the relative risk of lung cancer  
19 in non-smokers due to chronic exposure to ETS ranges between  
20 1.2 and 1.5."

21 JUDGE VITTON: Doctor, if you don't mind, since  
22 this is already in here, this is going to be in the record.

23 DR. BAYARD: Thank you. This concludes my  
24 testimony, Judge. I'll be happy to answer any questions.

25 JUDGE VITTON: We're going to take ten minutes.

1 (Whereupon, a brief recess was taken.)

2 JUDGE VITTON: We will identify for the record.  
3 Dr. Bayard's testimony dated March 10, 1995 which is 12  
4 pages long as Exhibit 277.

5 (The document referred to  
6 was marked for  
7 identification as Exhibit  
8 277 and was received in  
9 evidence.)

10 JUDGE VITTON: The copy of the figures and tables  
11 used during the presentation of his testimony which total 57  
12 will be identified as Exhibit 278 for the record.

13 (The document referred to  
14 was marked for  
15 identification as Exhibit  
16 278 and was received in  
17 evidence.)

18 JUDGE VITTON: Let me ask for a show of hands of  
19 who has questions for Dr. Bayard.

20 (Show of hands)

21 JUDGE VITTON: All right. Let me start over  
22 here, this gentleman here.

23 Who are you, sir?

24 DR. DAVIS: Dr. Ron Davis representing the  
25 American Medical Association.

1 JUDGE VITTON: Okay. How long are you going to  
2 be, Dr. Davis? Do you know?

3 DR. DAVIS: Well, I wouldn't need more than an  
4 hour.

5 JUDGE VITTON: Mr. Meyers? Approximately.

6 MR. MEYERS: We won't need more than 40 minutes.  
7 Again, it will depend on what happens before. It could be  
8 much shorter.

9 JUDGE VITTON: Okay. Let me start in the back.  
10 Mr. Eli?

11 MR. ELI: I may need about 30 minutes but if  
12 Mr. Furr has sufficient time, he may well cover much of what  
13 I have.

14 JUDGE VITTON: Mr. Andrade?

15 MR. ANDRADE: We're in the same position, Your  
16 Honor. We might 30 minutes to an hour but, again the areas  
17 that we examine on would be the same area that Mr. Furr is  
18 interested in and if he is going first and has ample time,  
19 our questioning might not be more than five or ten minutes.

20 JUDGE VITTON: Okay. Mr. Weinberg?

21 MR. WEINBERG: Ten to 20 minutes, possibly.

22 JUDGE VITTON: Okay. Mr. Furr?

23 MR. FURR: Well, since you're asking what we need,  
24 I need about three or four days.

25 (Laughter)

1 MR. FURR: I should say before he started talking  
2 today I need three or four, I need five or six now. I can  
3 condense into six hours, though.

4 JUDGE VITTON: Mr. Furr, I can tell you right now  
5 you're not getting six hours.

6 Okay. Ms. Sherman?

7 MS. SHERMAN: Depending on what comes out,  
8 possibly a half an hour.

9 JUDGE VITTON: Okay.

10 All right. Let me make sure I've got everybody.

11 Is there anyone else out there whose hands I  
12 didn't call on?

13 (No audible response)

14 JUDGE VITTON: Okay. All right.

15 Ms. Sherman indicated last week that she would  
16 prefer to go last.

17 MS. SHERMAN: It's not a matter of preference. I  
18 will accede to the Reynolds request that we go last as long  
19 as we do have time available.

20 JUDGE VITTON: I understand. All right. Okay.

21 (Inaudible comment from audience.)

22 JUDGE VITTON: Is there any objection if I let  
23 Mr. Furr go first?

24 (No audible response)

25 JUDGE VITTON: I see nobody objecting.

1           Mr. Furr, in view of comments of everybody, I'm  
2 going to give you three hours. I'll allocate up to three  
3 hours for you, starting right about now. You know I'm not a  
4 hard timer on anybody here but I'm going to allocate three  
5 hours for you right now and then we'll see how we go and  
6 then I'll see where everybody else is, okay?

7           MR. FURR: Are we on the record?

8           JUDGE VITTON: Yes, we are. Do you want to come  
9 on up?

10           MR. FURR: I don't want to engage in any argument  
11 taking up my time. I did want to make sure I state for the  
12 record that we object to being limited to three hours. It  
13 is wholly insufficient for the testimony that Dr. Bayard has  
14 offered including all the new material he offered today but  
15 we'll proceed and do the best we can.

16           JUDGE VITTON: Well, I'm going to give you three  
17 hours and we'll see where we are. If there isn't a lot of  
18 other examination and we have adequate time and it's a  
19 reasonable time during the day, I may give you more but  
20 we'll see where we are and who else has questions and where  
21 everything is at that point.

22           MR. FURR: Thank you, Your Honor.

23           DR. BAYARD: Your Honor, could we turn the lights  
24 down a little? They're really glaring at me.

25           JUDGE VITTON: Yes, they seem to be a little

1 brighter today. Maybe it's because we're not used to them  
2 any more.

3 Can we turn them down just a little bit?

4 Maybe OSHA should do a study on the effect of  
5 lights and blindness or something on Administrative Law  
6 Judges, a very small study group.

7 (Laughter)

8 MS. SHERMAN: Well, what would be test of  
9 significance?

10 JUDGE VITTONI: There probably wouldn't be a lot  
11 of sympathy, either.

12 (Laughter)

13 DR. BAYARD: I'm sorry, they're just glaring.

14 JUDGE VITTONI: Is that still a little too bright  
15 for you?

16 DR. BAYARD: Yes, please.

17 JUDGE VITTONI: Is there one that's maybe -- maybe  
18 this one over here -- is that any better?

19 DR. BAYARD: I'll try. Thank you.

20 JUDGE VITTONI: Do you want to wear a pair of  
21 sunglasses? Maybe that will help.

22 DR. BAYARD: These are the best I can do. I don't  
23 have sunglasses with me.

24 JUDGE VITTONI: I'm sorry about that.

25 I guess you guys can still get a pretty good

1 picture of everything.

2 This one right here seems to be a little brighter,  
3 the second one that's one -- or the third one, I guess.

4 Yes, it's the third one.

5 (Pause)

6 JUDGE VITTON: Yes, that's a little better.

7 I'm sorry, Mr. Furr. Go ahead.

8 MR. FURR: My three hours are starting now, Your  
9 Honor?

10 (Laughter)

11 MR. FURR: Good morning, Dr. Bayard.

12 DR. BAYARD: Good morning.

13 MR. FURR: As I think you know, I'm Jeff Furr. I  
14 represent the R.J. Reynolds Tobacco Company as well as a  
15 number of other parties to this hearing.

16 I want to begin by asking you if we can agree on a  
17 very simple ground rule that I'm sure His Honor will approve  
18 in an effort to be as efficient as we can today and develop  
19 the best record that we can for the decision maker and what  
20 I would like to ask is that I'm going to ask as precise a  
21 question as I can of you and I would ask you to try to  
22 answer exactly the question that I ask as opposed to some  
23 other question so that when the decision maker goes to  
24 review the record, we'll have questions and answers that  
25 match up. That's fair, isn't it?

1 DR. BAYARD: I always do that.

2 MR. FURR: I appreciate that.

3 JUDGE VITTON: Of course, you understand,  
4 Dr. Bayard, you always have an opportunity at some point if  
5 you want to make an explanation later on of any answer.  
6 I'll give you an opportunity when we finish to day if you  
7 want to make some kind of clarification statement on any  
8 point, I'll let you do that at the end of the day.

9 DR. BAYARD: I tend to want to answer the question  
10 as well as I can --

11 JUDGE VITTON: Sure.

12 DR. BAYARD: -- which means that --

13 JUDGE VITTON: No, I understand that. But I'm  
14 just saying --

15 DR. BAYARD: It's important to me to answer the  
16 question when it's asked because I have a short memory.

17 JUDGE VITTON: Okay.

18 DR. BAYARD: Thanks.

19 MR. FURR: Dr. Bayard, you told us earlier today  
20 that you spent the last 20 years working for the Federal  
21 Government. Has all that time been for the EPA?

22 DR. BAYARD: I think I told you it was 18 years  
23 but, no --

24 MR. FURR: Let me ask it this way. You've spent a  
25 large portion of your professional life working for the

1 Federal Government and you've spent a large part of that  
2 time conducting risk assessments. Is that correct?

3 DR. BAYARD: That's correct.

4 MR. FURR: You would hold yourself out as an  
5 expert in risk assessment, wouldn't you?

6 DR. BAYARD: People tell me I am but an expert is  
7 someone who lives in another city.

8 MR. FURR: Well, do you think you're an expert?

9 DR. BAYARD: I do my best. I don't want to  
10 categorize myself like that. I do my best.

11 MR. FURR: And your experience with risk  
12 assessment has largely been in the context of risk  
13 assessment performed under federal statutes, hasn't it?

14 DR. BAYARD: Yes. But I have certainly reviewed  
15 risk assessments that have come in -- I'm sorry. Mostly  
16 under federal statutes. That's correct.

17 MR. FURR: You reviewed OSHA's proposed rule,  
18 obviously, so you understand, don't you, that smoking would  
19 be limited to designated smoking areas in which no work of  
20 any kind could be performed?

21 DR. BAYARD: You know, the answer is yes, I  
22 reviewed OSHA's rule but I only reviewed the part of the  
23 OSHA rule relating to the science behind the risk assessment  
24 of environmental tobacco smoke and I expect that the OSHA  
25 rule is a pretty comprehensive ban but I spent very little

1 time looking at what the rule was because I was interested  
2 in the risk assessment part of it.

3 MR. FURR: So you're not familiar with the  
4 restrictions that the rule would place on smoking in the  
5 workplace.

6 DR. BAYARD: I'd probably agree to anything you  
7 said on that.

8 MR. FURR: Okay. Well, let me ask you a question  
9 that goes straight to the heart of whether OSHA's rule is  
10 necessary. You do not believe that completely eliminating  
11 smoking from the workplace is necessary to eliminate any  
12 significant risk of material impairment to health from ETS  
13 exposure in the workplace, do you?

14 DR. BAYARD: I'm sorry, I missed it, Jeff.

15 MR. FURR: You don't believe that a zero exposure  
16 level for ETS is necessary to eliminate any significant risk  
17 of disease from occupational ETS exposure, do you?

18 DR. BAYARD: I suspect there's a threshold, if  
19 that's what you're asking. But I don't know where it is.

20 MR. FURR: But you believe that there probably is  
21 a threshold below which there is no significant risk.

22 DR. BAYARD: Significant in the terms of 10 to the  
23 minus six, say? Is that what you would like to describe  
24 significant as?

25 MR. FURR: Well, I want to use the word

1 significant as you use it when you're talking about risk  
2 assessment in the context of federal statutes.

3 DR. BAYARD: I want to help you any way I can  
4 here. You know, we use significant when we talk about  
5 statistical significance but significant risk is probably  
6 used by EPA as anything around 10 to the minus six range and  
7 above.

8 MR. FURR: You've been asked this question before  
9 in hearings, haven't you?

10 DR. BAYARD: In terms of whether or not I believe  
11 there's a threshold below which there's no risk for  
12 environmental tobacco smoke?

13 MR. FURR: Well, actually, let me refocus it. As  
14 to whether you believe that a complete elimination of  
15 smoking in the workplace is necessary to eliminate any  
16 significant risk. You were asked this in the Maryland OSHA  
17 hearing.

18 DR. BAYARD: Yes. And I'll tell you -- if you ask  
19 me the same question I can respond the same way and the  
20 question says do I believe there's a threshold and I said,  
21 yes, I said, if I go in for 20 minutes to a restaurant then  
22 I'm not going to worry a whole bunch about it, I probably  
23 wouldn't take my kid there but I would also be concerned  
24 that it's not me that has to worry about 20 minutes, it's a  
25 waiter and a waitress.

1 MR. FURR: Well, let me ask you the question the  
2 same way you were asked it in the Maryland hearing.

3 DR. BAYARD: Oh, good.

4 MR. FURR: And tell me whether you still agree.  
5 That was a hearing before the Maryland OSHA, correct?

6 DR. BAYARD: December 16, 1993.

7 MR. FURR: That's right. And it was before  
8 Maryland OSHA?

9 DR. BAYARD: Yes, sir.

10 MR. FURR: And that was a hearing on whether some  
11 rules being considered by the state of Maryland regarding  
12 workplace smoking were necessary.

13 DR. BAYARD: That's correct.

14 MR. FURR: And the issues in that hearing were  
15 very much like the issues in this hearing and they included  
16 whether occupational exposure to ETS posed a significant  
17 risk of lung cancer and heart disease.

18 DR. BAYARD: I think their definition of  
19 significance was different than OSHA's, isn't it?

20 MR. FURR: Well, I want to ask you the question  
21 you were asked there. You didn't qualify your answer and  
22 you didn't debate what the term significant means.

23 DR. BAYARD: No, I didn't. I'm sorry I'm having  
24 trouble with the question.

25 MR. FURR: Isn't it true that during that hearing

1 you were asked questions by a Dr. Farrel --

2 DR. BAYARD: I don't know the name.

3 MR. FURR: -- who was a member of the OSHA board?

4 DR. BAYARD: I don't know the name.

5 MR. SHEEHAN: Mr. Furr, I might suggest if you  
6 have the question and answer you just read those.

7 (Mr. Furr proffers document to Dr. Bayard.)

8 MR. FURR: Dr. Bayard, I hope in our short time  
9 together today that you and I get to discuss lots of  
10 documents.

11 DR. BAYARD: Sure.

12 MR. FURR: I made copies of all of them; extra  
13 copies for you if you wanted to look at one.

14 DR. BAYARD: Oh, good. May I have that?

15 MR. FURR: You can. What I would ask is you go  
16 ahead and keep all of the documents up there so we can refer  
17 back to them without having to go back and forth, and we'll  
18 enter them into the record at the end of the examination.

19 DR. BAYARD: Thank you.

20 MR. FURR: During the Maryland OSHA hearing, Dr.  
21 Bayard, what --

22 MR. SHEEHAN: I'm sorry, Mr. Furr. Could he have  
23 a chance to read this?

24 MR. FURR: I'm going to read it. He can read as I  
25 go.

1 MR. SHEEHAN: Okay.

2 MR. FURR: Weren't you asked the question by Dr.  
3 Ferrell, as follows:

4 "I would ask the same question I asked Dr.  
5 Bascomb, which is basically: 'Do you believe that there is  
6 a level of environmental tobacco smoke at which a person who  
7 is exposed will not suffer material impairment of health or  
8 functional capacity?" and wasn't your answer, in part, "The  
9 answer is certainly"?

10 DR. BAYARD: But I can't tell you what it is, and  
11 you raise a question, which I think is a real good question.  
12 Not only that, but you know smokers and nonsmokers are  
13 probably different physiologically. People don't smoke a  
14 lot.

15 MR. FURR: Dr. Bayard, my only question to you now  
16 is, wasn't your answer, in part, "The answer is certainly"?

17  
18 DR. BAYARD: I'd like to read the whole answer.

19 MR. FURR: We're going to enter that into the  
20 record. It will all be in.

21 JUDGE VITTON: Wait a minute. Were you reading  
22 from the record or speaking there?

23 DR. BAYARD: I was giving the same answer that I  
24 gave to that question.

25 MR. SHEEHAN: He was reading from the record, Your

1 Honor.

2 JUDGE VITTON: All right. That's what I want to  
3 know.

4 MR. SHEEHAN: Your Honor, I would note we're given  
5 one page here, and his answer goes onto the next page, which  
6 isn't there, and so this is obviously not a complete  
7 response.

8 MR. FURR: We will submit the whole transcript to  
9 the record, Your Honor.

10 JUDGE VITTON: All right.

11 MR. FURR: Dr. Bayard, let me --

12 MR. MYERS: I want to object. The witness gave a  
13 complete answer to a question, and we're not getting a full  
14 representation. The witness doesn't remember his full  
15 answer. He's entitled to see the full answer and for us to  
16 see it.

17 MR. FURR: The full answer will be in the record.

18

19 MR. MYERS: If you've got the full answer, let's  
20 see it.

21 JUDGE VITTON: You're going to put the whole  
22 transcript into the record?

23 MR. FURR: I'll put the whole transcript into the  
24 record.

25 JUDGE VITTON: Mr. Myers can read it as long as

1 he wants to.

2 MR. MYERS: No, but the witness should be able to  
3 see his entire answer. He's being grilled on it.

4 MR. FURR: It's before him now.

5 MR. SHEEHAN: His entire answer is not before him.  
6 We just have an excerpt of his answer. If you have the  
7 rest, I suggest you present it to him.

8 MR. FURR: Let me ask you a different question.

9 MR. MYERS: No. He's --

10 MR. FURR: I don't want to waste anymore time on  
11 this.

12 MR. MYERS: -- playing games. This is something  
13 important.

14 MR. FURR: That is the entire answer to this  
15 question.

16 JUDGE VITTON: All right. I want you to give him  
17 an opportunity, though, to explain his answer that he has  
18 given in the Maryland hearing, beyond just the one or two  
19 words that has come out into the record.

20 Dr. Bayard, are you finished with your answer with  
21 respect to that first question?

22 DR. BAYARD: My lawyer asked if we could see the  
23 whole answer, and -- I mean, that's why I brought my lawyer  
24 up here.

25 JUDGE VITTON: Can you show it to him, Mr. Furr.

1 (Mr. Furr proffers document to Dr. Bayard.)

2 MR. SHEEHAN: Just for the record, we have now  
3 been presented page 298, which completes the answer that Dr.  
4 Bayard started.

5 MR. FURR: Isn't the explanation of your answer  
6 that EPA believes that there are levels of exposure to ETS  
7 that don't pose a significant risk to health; you believe  
8 there is probably a threshold of some sort?

9 DR. BAYARD: I believe that from nearly everyone  
10 you're going to find a threshold somewhere. It's a matter  
11 of --

12 MR. FURR: Below that threshold --

13 DR. BAYARD: -- I'm sorry.

14 MR. FURR: -- further exposure would no longer  
15 pose a significant --

16 DR. BAYARD: -- I didn't --

17 MR. FURR: -- health --

18 JUDGE VITTON: Both of you can't talk at the same  
19 time.

20 DR. BAYARD: I'm sorry. I just didn't finish my  
21 answer.

22 JUDGE VITTON: Go ahead and finish.

23 DR. BAYARD: Okay. The problem is, where do you  
24 find that threshold. For most of these chemicals that we  
25 look at, we look at animal studies, and we're looking at

1 anywhere from 10,000 to a million times what a normal  
2 environmental exposure would be.

3 For even occupational exposure studies, we're  
4 seeing anywhere from 10,000 to a million times what a normal  
5 exposure will be.

6 So you can pick out any number, and say, okay, you  
7 can go up a little and you're probably not going to have too  
8 much risk. But with environmental tobacco smoke, I think  
9 you have to be a little bit more careful because lung cancer  
10 has been shown at very typical environmental levels.

11 So when you pick a threshold, the answer is, where  
12 do I pick one. I suspect that one could say, okay, let's  
13 pick a 10 to the minus 6th risk level, which we will  
14 determine as your significant depending on who's asking.

15 It becomes a little bit more dangerous with an  
16 environmental tobacco smoke, I think, because you don't have  
17 the uncertainty of extrapolation from the very high to low  
18 levels.

19 To get back to the question, I think, yes, I still  
20 believe there are thresholds; I just don't know where it is.

21 MR. FURR: Thank you.

22 In the proposed rule, OSHA never actually  
23 addresses what level of ETS exposure, in any quantitative  
24 sense that it believes to pose a significant risk to health,  
25 does it?

1 DR. BAYARD: I still would say, when you're  
2 testing it, the answer is I think the same typical  
3 occupational levels, from my reading of it.

4 MR. FURR: But in a quantitative sense?

5 DR. BAYARD: In a quantitative sense, they're not  
6 giving a dose response slope, but they are giving  
7 quantitative estimates of bodies, estimated bodies for  
8 average, typical exposures.

9 MR. FURR: But, as you just stated, the proposed  
10 rule contains no effort by OSHA to derive a dose response  
11 relationship for ETS and either lung cancer or heart  
12 disease.

13 DR. BAYARD: I think that's correct.

14 MR. FURR: Along the same vein, there is no  
15 assumption in the proposed rule of the level of ETS exposure  
16 that poses a significant versus an insignificant risk, is  
17 there?

18 DR. BAYARD: I'm sorry, the term is -- and here's  
19 where we get back at the significant versus insignificant.

20 MR. FURR: Regardless of what it means, is there  
21 any assessment in the proposed rule of what level creates a  
22 significant versus an insignificant risk? There's none in  
23 there, is there?

24 DR. BAYARD: It's hard to answer the question  
25 because if you'll tell me what you mean by "significant", if

1 you mean 1 in 1,000, which I think, is that an OSHA  
2 definition of "significant"?

3 MR. FURR: Dr. Bayard, regardless of the  
4 definition of significant or insignificant, without a dose  
5 response relationship, they simply can't address that issue,  
6 can they?

7 DR. BAYARD: The answer to that question is no,  
8 because -- I can't address the issue either, because I don't  
9 know what "significant" means. If significant means a  
10 thousand bodies, then that's significant. If it means a  
11 hundred bodies, then it's significant. If it means one  
12 Board of Directors per microgram of RSP per cubic meter,  
13 that's significant.

14 If you'll give me a definition of "significant,"  
15 I'll do the best I can.

16 MR. FURR: Okay. Isn't it true that there is no  
17 assessment in the proposed rule of a level of ETS exposure  
18 tied to any particular risk level, whether that be 10 to the  
19 6th or any other risk?

20 DR. BAYARD: The level of ETS. I don't even think  
21 I'd go that far because they're assuming some typical  
22 exposure levels, and then they're giving you bodies for  
23 prevalence of exposure to these typical levels.

24 Assuming that they could provide a reasonable  
25 estimate of what these typical exposure levels are, then the

1 equation could be easily completed.

2 MR. FURR: But they don't have that information,  
3 so they haven't been able to do it yet, have they?

4 DR. BAYARD: I'd say they don't have the exact  
5 information that you're asking. At least it isn't written  
6 out chapter and verse in that way. I think all of the  
7 information is actually in there. I think it's just a  
8 matter of doing a little multiplication.

9 MR. FURR: Okay. While we're talking about  
10 thresholds, I want to move on to something else. That is,  
11 one of the points you make in your written statement and  
12 again here today is that for mainstream smoking there is no  
13 evidence of an exposure threshold for risk.

14 DR. BAYARD: That's correct.

15 MR. FURR: You're not saying that there is not an  
16 exposure threshold, are you? I know that's a double  
17 negative.

18 DR. BAYARD: I have trouble with it. Say it  
19 again.

20 MR. FURR: You're not saying that there is any  
21 evidence that there is no threshold for mainstream smoke and  
22 lung cancer risk, are you?

23 DR. BAYARD: Let me try and answer.

24 MR. FURR: Okay.

25 DR. BAYARD: I'm saying there is evidence that

1 there is no threshold.

2 MR. FURR: There is evidence that there is no  
3 threshold?

4 DR. BAYARD: Yes.

5 MR. FURR: What evidence is that?

6 DR. BAYARD: The dose response that you see in all  
7 of the studies. I can look it up on the books if you want.

8 MR. FURR: Isn't it true that there is not a  
9 single study that contains any information on the risk of  
10 lung cancer for smoking one cigarette or less per day?

11 DR. BAYARD: I don't think so, but if you wait a  
12 minute, I'd like to look.

13 (Pause)

14 DR. BAYARD: There's the ACS-9 study, for example,  
15 occasional smoking; occasional smoking may be --

16 MR. FURR: Dr. Bayard, let's get back to our  
17 precise question and precise answer. I asked a very precise  
18 question.

19 MR. MYERS: Your Honor, can he be allowed to  
20 answer the question?

21 JUDGE VITTON: Okay. He's going to try to  
22 clarify his question, I think.

23 MR. MYERS: He was in the middle of the sentence.

24 JUDGE VITTON: Mr. Myers, I'll take care of it,  
25 okay? All right.

1           Are you going to clarify the question, are you  
2 going to rephrase it, what?

3           MR. FURR: I'll try to repeat it, because I  
4 thought it was very clear.

5           JUDGE VITTONI: All right. Repeat the question.

6           MR. FURR: The question is: Do any of the studies  
7 contain any data with respect to whether the smoking of one  
8 cigarette per day poses an increase in risk.

9           JUDGE VITTONI: All right.

10          DR. BAYARD: There are a lot of studies, from what  
11 I'm reading in Table 4-6.

12          MR. FURR: One cigarette or less per day?

13          DR. BAYARD: There's less than 1, there's 1 to 4.  
14 No. Those seem to be the normal breakdowns.

15          MR. FURR: Dr. Bayard, let's move on.

16          DR. BAYARD: Thank you.

17          MR. FURR: Isn't there evidence emerging that, at  
18 least for some alleged carcinogens, there probably is a  
19 threshold of exposure for increases in risk?

20          DR. BAYARD: For some carcinogens, is there  
21 evidence of threshold. I don't know. If you'll tell me  
22 about specific cases.

23          MR. FURR: All right. What about radon? Isn't  
24 there evidence that there's a threshold for radon exposure  
25 and risk?

1 DR. BAYARD: I'm not an expert on radon.

2 MR. FURR: So you just simply don't know, as you  
3 sit here?

4 DR. BAYARD: I have read several radon studies. I  
5 would try to remember them, but I don't think I could give a  
6 very good answer.

7 MR. FURR: Have you read recent reports by Bernard  
8 Cohen of the University of Pittsburgh and Graham Covitz of  
9 the Harvard School of Public Health?

10 DR. BAYARD: What were they on, please?

11 MR. FURR: They were titled, "Test of the Linear  
12 No Threshold Theory for Lung Cancer Induced by Exposure to  
13 Radon," appeared in Environmental Research in 1994; and a  
14 "Test of the Linear No Threshold Model of Radiation and  
15 Carcinogenesis" that appeared in 1995 in "Advances in  
16 Chemistry".

17 DR. BAYARD: I have not read those.

18 MR. FURR: You're not familiar with them, and so  
19 you don't know whether those articles contain any  
20 information that would suggest that there is a threshold for  
21 radon exposure?

22 DR. BAYARD: That's correct.

23 MR. FURR: Thanks.

24 Dr. Bayard, you read a lot of testimony during  
25 your Direct Examination and the judge cut you off right when

1 I wanted you to keep reading, so we're going to have to go  
2 to that section now. That is the Hazard Identification  
3 section in your written comments.

4 DR. BAYARD: Why didn't you object?

5 MR. FURR: Well, although you wanted my input, I  
6 wasn't sure the Judge did.

7 DR. BAYARD: When does that stop you?

8 JUDGE VITTONI: All right. Mr. Furr, I think I'm  
9 having a hard time making you happy here today.

10 MR. FURR: You are. I'm not very happy.

11 (Laughter)

12 MR. FURR: Let's go to the Hazard Identification  
13 section that begins on page 10. I want to ask you some  
14 questions about that.

15 On that page, you state in your written submission  
16 that OSHA's hazard identification provides an outline and  
17 rough summary of the data but no analysis or reference to an  
18 OSHA analysis. Is that correct?

19 DR. BAYARD: That's correct.

20 MR. FURR: You found OSHA's discussion of the  
21 epidemiologic studies to be unconvincing or, as you describe  
22 it in your written comments on page 12, to be perplexing to  
23 even an experienced reader. That's correct, isn't it?

24 DR. BAYARD: Yes.

25 MR. FURR: By that, you mean that the proposed

1 rule really does not contain an explanation of how OSHA  
2 evaluated the epidemiologic studies, does it?

3 DR. BAYARD: (No response.)

4 MR. FURR: There's no identification of the  
5 criteria that were used to assign studies for various  
6 categories, for instance?

7 DR. BAYARD: For the most part, I was left  
8 somewhat unfulfilled.

9 MR. FURR: And you're an experienced reader of  
10 literature with respect to these epidemiologic studies?

11 DR. BAYARD: I sure know these data.

12 MR. FURR: As you put in your written comments,  
13 "OSHA's discussion was hardly explanatory."

14 DR. BAYARD: That's correct.

15 MR. FURR: And that had to do with their  
16 assignment of studies to one of three categories, either the  
17 positive equivocal with positive trend or equivocal?

18 DR. BAYARD: That's correct. May I just continue  
19 for just a minute?

20 MR. FURR: Sure.

21 DR. BAYARD: Without sounding too harsh on OSHA,  
22 EPA often has different rules about the way they do business  
23 than OSHA does. I can tell you I certainly understood where  
24 they were coming from and how they would purport to do an  
25 analysis, but I can't imagine most people who don't know

1 this data set --

2 MR. FURR: So you would agree that an interested  
3 member of the public without your experience would have a  
4 difficult time understanding how they assign these studies  
5 to various categories?

6 DR. BAYARD: Yes.

7 MR. FURR: OSHA also includes in their proposed  
8 rule that the relative risk for lung cancer from chronic ETS  
9 exposure ranges from 1.2 to 1.5. They really don't provide  
10 any explanation as to how they reached that conclusion,  
11 either, do they?

12 DR. BAYARD: Not to my satisfaction.

13 MR. FURR: Wouldn't you agree that members of the  
14 public without your experience in this area might have a  
15 difficult time actually meaningfully commenting on the  
16 information contained in the Proposed Rule?

17 DR. BAYARD: I can speak for myself. I don't  
18 think I can speak to members of the public.

19 MR. FURR: I was intrigued by another statement in  
20 that section of your written comments, and it's one that you  
21 did get to today. That is, you state that, quote: "I  
22 understand from discussions with OSHA staff, that a more  
23 complete analysis of the weight of evidence for ETS and lung  
24 cancer is forthcoming." That's what you said today, isn't  
25 it?

1 DR. BAYARD: Before the Judge cut me off.

2 MR. FURR: Okay. Well, I just correctly read a  
3 statement from your written comments, didn't I?

4 DR. BAYARD: Excuse me?

5 MR. FURR: I correctly read that statement, didn't  
6 I, as contained in your written comments at page 11? The  
7 last sentence before "quantitative risk assessment" on that  
8 page, the heading.

9 DR. BAYARD: Yes, sir.

10 MR. FURR: That's a statement that you wrote  
11 sometime prior to the submission of these comments in  
12 September of 1994?

13 DR. BAYARD: Yes. I can tell you when I wrote  
14 them.

15 MR. FURR: When did you write them?

16 DR. BAYARD: Because August 10th and August 12th.

17 MR. FURR: So the discussions you're referencing  
18 in that statement, then, must have occurred before all of  
19 August 10th or 12th?

20 DR. BAYARD: Yes.

21 MR. FURR: Tell us about those discussions. Who  
22 were they with?

23 DR. BAYARD: I know Ken Brown, and Ken Brown has  
24 submitted a draft of Hazard Identification Analysis to OSHA,  
25 and I have been asked to review it.

1 MR. FURR: You say that you understand from  
2 discussions with OSHA staff. Who on the OSHA staff did you  
3 have those discussions with?

4 DR. BAYARD: I speak mainly with Demetra Collia.

5 MR. FURR: Anyone else?

6 DR. BAYARD: I speak to some other people on the  
7 staff, but she's the main person that I talk to.

8 MR. FURR: Were there any other OSHA staff members  
9 that you were referencing when you made this statement in  
10 your written comments?

11 DR. BAYARD: Well, can I try and answer, and if it  
12 doesn't work, you can --

13 MR. FURR: Sure.

14 DR. BAYARD: I deal with Demetra Collia because  
15 she's the one who deals with the risk assessment, and when  
16 she gets the risk assessment material in and wants me to  
17 review it, she will call me and it's based on that, that I  
18 assume OSHA has contracted the work, because they are going  
19 to do a more complete analysis than the analysis that was  
20 presented as a preliminary analysis, and that seems  
21 reasonable to me.

22 Does that answer your question? Does that help at  
23 all?

24 MR. FURR: Well, we can work with that.

25 What did Demetra tell you about further analyses

1 that were to be done?

2 MR. SHEEHAN: Your Honor, I'm going to have to  
3 interject here. I think it's a proper question what  
4 contacts Dr. Bayard has had with OSHA, but the substance of  
5 those contacts I don't think is relevant for this hearing  
6 today.

7 MR. FURR: Of course it's relevant, Your Honor.  
8 I'm trying to find out what OSHA's frame of mind was when  
9 they issued this proposed rule. This statement suggests  
10 that they had already determined how to proceed before these  
11 hearings even began.

12 What I'd like to know is what OSHA was saying  
13 about their future plans even before this hearing began.

14 DR. BAYARD: May I say something? My feeling is  
15 that we've had many versions of risk assessments, that I've  
16 done and I've asked people to review as we've gone along.  
17 It doesn't mean you always start with your best shot, as we  
18 certainly found out on our own.

19 MR. FURR: What did Demetra tell you about OSHA's  
20 plans for further analyses on the hazard identification  
21 section?

22 MR. SHEEHAN: Your Honor, I don't think this is a  
23 proper line of questioning.

24 JUDGE VITTON: Mr. Furr, why do you want to  
25 explore this area? Tell me that. I'm not trying to --

1 MR. FURR: Because I'd like for the decision-maker  
2 to have the benefits of evaluating the objectivity with  
3 which OSHA has approached this rulemaking.

4 JUDGE VITTON: The obvious one?

5 MR. FURR: The objectivity with which OSHA has  
6 approached this rulemaking, and I think, whether or not they  
7 had already formulated a further plan of analysis prior to  
8 the receipt of 100,000 comments from the public and a six-  
9 month hearing is relevant to that.

10 DR. BAYARD: It's probably best to ask OSHA, I  
11 think.

12 MR. FURR: I don't have that opportunity,  
13 unfortunately.

14 JUDGE VITTON: I don't --

15 MR. SHEEHAN: Well, he's trying to get that  
16 opportunity from Dr. Bayard, and that's not proper to us.

17 MR. FURR: Is there a privilege here, Your Honor?  
18 It's clearly relevant; can't we have an answer? There is no  
19 privilege.

20 MR. SHEEHAN: Your Honor, the OSHA rules do state  
21 that Cross Examination is allowed on crucial matters. This,  
22 obviously, is getting into internal policy making and  
23 deliberations, and this is not a proper subject.

24 MR. FURR: Your Honor, surely I can Cross Examine  
25 on a statement included in his written statement and

1 repeated today in this hearing.

2 MS. SHERMAN: Your Honor, I would like to point  
3 out --

4 JUDGE VITTONI: Yes.

5 MS. SHERMAN: -- that Dr. Bayard is not under an  
6 compulsion to answer any questions, and I think that he is  
7 well aware of the rules of this hearing as is Mr. Furr.

8 MR. FURR: Of course, he's not under any  
9 compulsion, Your Honor, but when Reynolds testified, you  
10 exercised a great deal of control over what questions would  
11 and wouldn't be answered.

12 You overruled Mr. Grossman on many occasions when  
13 he posed an objection.

14 I'm not really asking for anything more than for  
15 you to continue to exercise your role in this hearing.

16 JUDGE VITTONI: All right. Look, he's raised the  
17 question in his written statement here and repeated here  
18 today. You have asked him a question on who he has talked  
19 to. He has told you who he has talked with.

20 I don't want to spend a lot of time on this.

21 MR. FURR: I don't either.

22 JUDGE VITTONI: Okay. So what's your next  
23 question? Let me hear it and then I'll decide whether he's  
24 going to answer it.

25 MR. FURR: My question is: What information did

1 the OSHA staff communicate to you at that time with respect  
2 to their plans for a more complete analysis of the weight of  
3 evidence for ETS and lung cancer?

4 JUDGE VITTON: All right. If you can answer it.  
5 Can you answer that question?

6 DR. BAYARD: I can try. I don't know where he's  
7 going. You know, Jeff, what happens is, once you get the  
8 material from Dr. Brown, she says, can I review this for  
9 her.

10 I review the material, and I tell her my opinions,  
11 and I don't ask what's going on. That's what I do.

12 Now, if you think there's some mysterious policy  
13 that I know about, I don't.

14 MR. FURR: Okay. Let me ask it this way. On what  
15 information did you draw your conclusion that the OSHA staff  
16 planned a more complete analysis of the weight of evidence  
17 for ETS and lung cancer? What was the basis for your  
18 statement here?

19 DR. BAYARD: When I get a ream of material, or  
20 when I get an amount of material that Demetra sends me that  
21 her contractor has supplied to her, then I assume that this  
22 material will at least form the basis of a more complete  
23 hazard identification other than the table that I saw and am  
24 somewhat perplexed about.

25 MR. FURR: So is your testimony that this was

1 merely an assumption on your part and you were never told by  
2 anyone on the OSHA staff that there was a more complete  
3 analysis forthcoming?

4 DR. BAYARD: You know, I guess it was an  
5 assumption, or it is an assumption.

6 MR. FURR: Okay. I wish you would have told me  
7 that 10 minutes ago.

8 DR. BAYARD: I, personally, can't imagine why  
9 someone would do an analysis and not plan to do something  
10 with it.

11 MR. FURR: Okay. Let's move on.

12 I want to talk about OSHA's quantitative risk  
13 assessment. You know what I mean by the phrase "risk  
14 assessment"?

15 DR. BAYARD: Yes, sir.

16 MR. FURR: You used another phrase today in your  
17 testimony, I think you called it "population risk estimates"  
18 or something like that. What was the phrase you used today?  
19 "Quantitative estimates of population risks," Slide 44.  
20 That's what I want to talk to you about.

21 Dr. Bayard, wouldn't you agree that for a risk  
22 assessor, the most difficult problem faced in any risk  
23 assessment is the problem created by uncertainty?

24 DR. BAYARD: That's a good place to start. Let's  
25 just say it's an uncertainty, and risk assessments can be

1 really difficult. A lot of that uncertainty is  
2 extrapolation from high to low doses, extrapolating from  
3 animals to humans, estimation of exposure.

4 But certainly the two that EPA has fastened on as  
5 measures of uncertainty are the animal to human and the high  
6 to low.

7 MR. FURR: But there are many sources --

8 DR. BAYARD: No, let me just finish, Jeff.

9 MR. FURR: Your Honor, this is unresponsive to my  
10 question.

11 JUDGE VITTON: Wait a minute. Just let him  
12 finish the answer. Go ahead.

13 DR. BAYARD: In that sense, that part of the  
14 uncertainty, the animal to human and the high to low dose  
15 extrapolation, is not present in either the OSHA or the EPA  
16 risk assessment, as I see it.

17 I'm sorry. Go ahead.

18 MR. FURR: You would agree that the proper  
19 identification treatment and discussion of uncertainty is  
20 crucial to a risk assessment, though, wouldn't you?

21 DR. BAYARD: Yes.

22 MR. FURR: And it's emphasized heavily in EPA's  
23 own risk assessment guideline?

24 DR. BAYARD: I don't know how heavily it is  
25 emphasized. It is discussed.

1 MR. FURR: It's acknowledged as a very important  
2 issue, isn't it?

3 DR. BAYARD: Can you point that out to me?

4 MR. FURR: Well, I don't want to take time to do  
5 that, but I'll state for the record that it's on page 111317  
6 and many other places in EPA's guidelines.

7 Let's talk about OSHA's risk assessment.

8 A form of uncertainty faced by risk assessors is  
9 simple statistical variability, isn't it?

10 DR. BAYARD: I'm sorry. I missed that. Please  
11 state that again.

12 MR. FURR: Okay. A form of the uncertainty  
13 problem is just simple statistical variability?

14 DR. BAYARD: Yes.

15 MR. FURR: And that's another way of saying how to  
16 address uncertainty created by chance variation and sampling  
17 problems.

18 DR. BAYARD: Yes.

19 MR. FURR: To arrive at its estimate of risk from  
20 occupational exposure to ETS, OSHA utilized a number of  
21 factors that are properly described as statistics, didn't  
22 it?

23 DR. BAYARD: Yes.

24 MR. FURR: Let me read a list of the factors to  
25 you and you tell me whether any of these are not statistics.

1           The relative risk estimates used by OSHA for lung  
2 cancer from occupational ETS; that's a statistic, isn't it?

3           DR. BAYARD: It's a statistical estimate.

4           MR. FURR: Okay. As is the relative risk estimate  
5 that OSHA used for cardiovascular disease?

6           DR. BAYARD: That's correct.

7           MR. FURR: OSHA also used a background or baseline  
8 incidence rate for lung cancer and cardiovascular disease  
9 that is also a parameter that's in the nature of a  
10 statistic, isn't it?

11          DR. BAYARD: That's correct.

12          MR. FURR: OSHA, as you mentioned in your oral  
13 testimony today, estimated the percentage of nonsmoking U.S.  
14 workers that are occupationally exposed to ETS. That's a  
15 statistic also, isn't it?

16          DR. BAYARD: That's a statistical estimate.  
17 That's correct.

18          MR. FURR: Like all statistics, the ones that we  
19 must mentioned have uncertainty in the sense of statistical  
20 variability that are associated with them.

21          DR. BAYARD: The answer is yes. I just want to  
22 make a very small comment on that.

23                 I think when you start getting into large  
24 population estimates, though, if you're estimating 74  
25 million, it seems to me, because you have a census count and

1 because you have such thorough labor statistics, it's an  
2 estimate. That statistic would probably be a less  
3 uncertainty than most. Even if it did have a little  
4 uncertainty, would not be a major sensitive member in your  
5 risk calculations.

6 MR. FURR: Let's explore that for some of the  
7 parameters that OSHA used.

8 DR. BAYARD: Sure.

9 MR. FURR: For the lung cancer risk assessment,  
10 OSHA used the Fontham, et al., risk number. Isn't that  
11 correct?

12 DR. BAYARD: Yes.

13 MR. FURR: That's a statistic that has uncertainty  
14 associated with it?

15 DR. BAYARD: Yes.

16 MR. FURR: That uncertainty can be described, in  
17 part, by the confidence intervals that surround the central  
18 estimate, can't it?

19 DR. BAYARD: Yes.

20 MR. FURR: For the risk assessment used by OSHA,  
21 the 95 percent confidence intervals surrounding the risk  
22 estimate are 1.03 to 1.73. That is a measure of the  
23 uncertainty around the central estimate, isn't it?

24 DR. BAYARD: Could you just tell me where that is,  
25 please?

1 MR. FURR: Well, it would be in Fontham -- excuse  
2 me. Let me rephrase that question.

3 OSHA used the Fontham 1991 study for its risk  
4 assessment for lung cancer of occupational ETS exposure. Do  
5 you have a copy of it or would you like me to hand you one?

6 DR. BAYARD: No, I have a copy.

7 MR. FURR: All right. If you take a look at the  
8 central estimate, I think you'll see that the 95 percent  
9 confidence intervals are from 1.02 to 1.73.

10 Your Honor, let me withdraw that and ask it a  
11 different way so we don't have to pull the study out.

12 JUDGE VITTONI: Okay.

13 MR. FURR: The confidence interval surrounding the  
14 central estimate are a measure of the uncertainty of the  
15 central estimate, aren't they?

16 DR. BAYARD: Yes.

17 MR. FURR: That would also be true for the Helsing  
18 study, of course. The central estimate used by OSHA in its  
19 quantitative risk assessment is a statistic whose  
20 uncertainty is measured in part by the confidence interval  
21 surrounding that number?

22 DR. BAYARD: Yes.

23 MR. FURR: Those confidence intervals reflect a  
24 specific kind of uncertainty, don't they? And that's the  
25 uncertainty due to random chance variation?

1 DR. BAYARD: It's probably more the sampling  
2 variability in this study.

3 MR. FURR: Sampling. Okay.

4 They don't reflect uncertainty due to uncontrolled  
5 sources of bias or confounding in any way, do they?

6 DR. BAYARD: Let me just try this and if you don't  
7 like it, we'll do it again, okay?

8 With respect to the Fontham study, she did control  
9 for a lot of the particular estimates so that her odds ratio  
10 of 1.34 was adjusted for several factors. So, in that  
11 sense, it controls for those factors. Both the odds ratio  
12 and the confidence intervals are adjusted for factors which  
13 were either matched into the study or adjusted for in the  
14 analysis.

15 It doesn't control for the ones that you didn't  
16 adjust for.

17 MR. FURR: Okay. Even with respect to the ones  
18 that you did adjust for, it controls for them only to the  
19 extent that he was to properly adjust for confounding or  
20 bias?

21 DR. BAYARD: That's correct.

22 MR. FURR: Okay. Now OSHA's quantitative risk  
23 assessment doesn't reflect the uncertainty surrounding the  
24 central risk assessments of Helsing and Fontham, does it?

25 DR. BAYARD: I think you're correct.

1 MR. FURR: All OSHA did was use the central risk  
2 estimate and treat it as though it were perfectly applicable  
3 to the current U.S. workplace without adjustment?

4 DR. BAYARD: I think the answer is yes, but I just  
5 want to make a small comment.

6 The business of risk assessment contains a lot of  
7 uncertainties, as I'm sure we'll be getting into. One of  
8 the estimates was the one related to the study itself. Then  
9 you're probably going to be dealing with the  
10 generalizability of that population to the --

11 MR. FURR: We are going to deal with that.

12 DR. BAYARD: Oh. Thanks. Go ahead.

13 MR. FURR: But the point is that OSHA did not deal  
14 with the uncertainty surrounding the central risk  
15 assessments from Fontham and Helsing, even as captured by  
16 the confidence intervals surrounding those estimates?

17 DR. BAYARD: I think that's correct.

18 MR. FURR: Let's talk a little more on OSHA's  
19 reliance on the Fontham study.

20 Now, Fontham, et al., did not even study males,  
21 did they; they only studied females?

22 DR. BAYARD: That's correct. Oh, wait a minute.  
23 They studied the male smoking habits.

24 MR. FURR: Subjects, cases, were female only?

25 DR. BAYARD: That's correct.

1 MR. FURR: Okay. So the risk estimate from  
2 Fontham, et al, is specific for females.

3 DR. BAYARD: That's correct.

4 MR. FURR: There is some uncertainty associated  
5 with applying a female specific risk assessment to perform a  
6 risk assessment for males, isn't there?

7 DR. BAYARD: That's correct. Although I would  
8 like to add that I think in the proposed rule -- I can look  
9 it up, but I don't want to take the time, unless you do --  
10 that the estimates that they projected from females to males  
11 were probably an undercount because the males have a higher  
12 background rate of lung cancer than nonsmoking, than do the  
13 nonsmoking females.

14 So, in that sense, I believe that while you are  
15 correct that OSHA did not display the uncertainty, they  
16 took the approach that they would prefer to underestimate  
17 the effect in males by using female rates.

18 MR. FURR: Dr. Bayard, OSHA's quantitative risk  
19 assessment does not address the uncertainty created by  
20 relying solely on the Fontham and Helsing studies as the  
21 source of the risk assessment as opposed to looking at all  
22 of the data, does it?

23 DR. BAYARD: That's correct, but I would like to  
24 add something there too. That is, what I tried to tell you  
25 before, and for the purposes of this discussion, I would

1 like to stay on the lung cancer.

2 The workplace exposure measures, unlike the  
3 spousal exposure measures, have a lot more problems with  
4 measuring exposure. Recall; we talked about proxy versus  
5 direct interview.

6 In that sense, I kind of think that if you take  
7 the best study and make your extrapolations from that, with  
8 respect to the workplace, you're better off than trying to  
9 take a whole bunch of studies that aren't of comparable  
10 quality.

11 I don't mean to stray far away from your question,  
12 but I think your question was, would I do better if I  
13 combined a whole bunch of studies rather than just took one;  
14 and I don't think you gain anything by taking studies that  
15 aren't good quality and adding them in just to get a smaller  
16 deviation.

17 MR. FURR: Dr. Bayard, I want to hand you a  
18 document that is a transcript of a meeting held at the Kato  
19 Institute, titled, "Tobacco Use, Politics and Safety," on  
20 October 4th, 1994.

21 DR. BAYARD: I remember that.

22 (Mr. Furr proffers document to Dr. Bayard.)

23 MR. FURR: Dr. Bayard, you appeared at that  
24 meeting, didn't you?

25 MR. SHEEHAN: Can I have this?

1 MR. FURR: I will send you one, but we're going to  
2 enter that one into the record today.

3 MR. SHEEHAN: Thank you.

4 MR. FURR: You appeared at that meeting as a  
5 speaker, didn't you?

6 DR. BAYARD: Yes.

7 MR. FURR: You discussed EPA's ETS risk  
8 assessment?

9 DR. BAYARD: Yes.

10 MR. FURR: Could you turn to page seven of the  
11 transcript? I would ask you to take a look at the  
12 penultimate paragraph, please.

13 Dr. Bayard, in your testimony at the Kato  
14 Institute, didn't you, in effect, make the point that the  
15 proper approach to analyzing a group of studies is to look  
16 at all of the studies, and that you should not just pick out  
17 one study and rely upon that individual study?

18 DR. BAYARD: The answer is, I was specifically  
19 referring to the spousal exposure studies because that's  
20 what we did. On the other hand, I think, if I can interpret  
21 your question, I don't mean that people shouldn't look at  
22 the other studies. I don't mean that they shouldn't analyze  
23 them, but just the opposite. One should analyze all of  
24 these studies.

25 One should have the perspective of what studies

1 are the best studies for which to make both your hazard  
2 identification argument, conclusion, and your population  
3 risk assessments.

4 MR. FURR: But you agree that all the studies  
5 should be examined?

6 DR. BAYARD: I certainly agree that all of the  
7 studies should be examined, but I don't agree that when you  
8 have, specifically for the OSHA workplace data that you're  
9 asking about and the recall of exposure for some of these  
10 studies, that they should be factored into the risk  
11 assessment blindly and just by rote formula.

12 MR. FURR: Let's talk about blindly using rote  
13 formula.

14 DR. BAYARD: Good.

15 MR. FURR: When EPA performed its ETS risk  
16 assessment, it used a technique frequently referred to as  
17 meta-analysis, didn't it?

18 DR. BAYARD: That was one of the techniques that I  
19 have explained today.

20 MR. FURR: You used that technique because you  
21 believe a benefit of that technique is that it helps you to  
22 analyze a whole group of studies and to take into account  
23 all of the data in arriving at your risk estimate?

24 DR. BAYARD: Yes. I just have one quick comment  
25 on that.

1           What we tried to do with ETS was to look at both  
2 positive and negative studies. It's something that EPA has  
3 not done very much of the way we've seen it, and we've tried  
4 to determine some objective measures of doing that. We  
5 didn't say that was the only analysis, it was one of the  
6 analyses that we did.

7           MR. FURR: Okay. Dr. Bayard, isn't it true that  
8 if a meta-analysis is performed upon all of the occupational  
9 data contained in the lung cancer, ETS epidemiologic  
10 studies, that that meta-analysis produces a summary risk  
11 estimate of 1.0?

12           DR. BAYARD: Let me try to help out. I think the  
13 answer is probably it's close. On the other hand, I am  
14 basing my answer on my remembrance of three meta-analyses  
15 that I've seen on the workplace exposure data. One is by  
16 Tweedi, one is by Max Layard, and one is by Peter Lee, I  
17 think.

18           All three of those analyses, as I remember, were  
19 wrong, for the reason that they combined studies from  
20 different countries.

21           When EPA was doing the risk assessment, one of the  
22 comments we got after our first draft, in fact from Max  
23 Layard, who is a consultant for the Tobacco Institute, was  
24 don't combine all countries. You should be looking at your  
25 data by country because there are so many different

1 conditions in the countries. When we looked at the data  
2 separately, we decided, our statistical tests said there are  
3 differences between countries.

4 Now if there are differences between countries for  
5 studies with spousal exposures, it seems to me reasonable to  
6 believe that there should be other factors which should be  
7 taken into account so that you don't combine studies for  
8 workplace exposures.

9 Therefore, while I agree that the meta-analyses  
10 that I have seen are probably around one total, I don't  
11 agree that they were properly done.

12 MR. FURR: What does a meta-analysis of the U.S.  
13 occupational data produce?

14 DR. BAYARD: I can only tell you what I have read.  
15 I have not done one. I agreed with your statement for the  
16 purposes of just moving this discussion along.

17 MR. FURR: I appreciate that.

18 Dr. Bayard you're aware, and in fact I think you  
19 mentioned today, the other large...

20 JUDGE VITTON: Excuse me a second. Let me make  
21 sure I understand what your answer is to that last question.

22 When you say you agree with his statement, you  
23 mean that the risk is one?

24 DR. BAYARD: His question was, when you combine  
25 all the workplace studies and do a meta-analysis, that the

1 odds ratio is one. I agreed that...

2 JUDGE VITTONI: For the United States.

3 DR. BAYARD: No, because no one has done an  
4 analysis that I've seen separately for the United States.

5 MR. FURR: You haven't performed one?

6 DR. BAYARD: No.

7 MR. FURR: You don't know whether it's 1.0 or not.  
8 Have you reviewed the R.J. Reynolds testimony to  
9 this hearing?

10 MR. FURR: The first question was... I'm sorry.  
11 Do you want to ask her? I'm sorry.

12 JUDGE VITTONI: Just a second.

13 MR. FURR: Have you reviewed...

14 DR. BAYARD: I'm trying to answer the first  
15 question, answer the first question, Your Honor.

16 The first question was something about whether I  
17 agreed that it was one. Can you read it back? I want to  
18 answer every question. He asked one question and didn't  
19 wait for an answer, and then you asked another question.

20 MR. FURR: Let's straighten it out.

21 The first question is this...

22 DR. BAYARD: Can we get back to the first  
23 question?

24 MR. FURR: Do you know what a meta-analysis of the  
25 U.S. occupational data produces?

1 DR. BAYARD: No.

2 MR. FURR: Have you reviewed the testimony  
3 submitted to this proceeding by R.J. Reynolds?

4 DR. BAYARD: Reviewed some of it. But I think I  
5 reviewed mostly Mike Ogden's, and a fellow named Steichen?

6 MR. FURR: Thomas Steichen, yes.

7 DR. BAYARD: Yeah.

8 MR. FURR: Dr. Bayard, aren't you also aware that  
9 the other large recent U.S. study by Brownson et al that you  
10 mentioned earlier found a relative risk of 1.0 for any  
11 occupational exposure to ETS?

12 DR. BAYARD: I think it's in my slide.

13 (Pause)

14 DR. BAYARD: Slide 43. Our estimate, which we  
15 took from Dr. Butler, was .98 for never-smoking women who  
16 worked outside the home for at least six months.

17 MR. FURR: I was just being generous when I said  
18 1.0.

19 DR. BAYARD: And it was less than that. It was  
20 0.67 if you include the all-never-smoking women.

21 MR. FURR: So Brownson et al found a risk  
22 estimate for any occupational exposure that's very different  
23 than that reported by Fontham et al, don't you agree?

24 DR. BAYARD: That's correct. Furthermore, ne has  
25 to look at the Brownson data versus the Fontham data,

1 whereas the Fontham data had a far lower proportion of white  
2 and a higher proportion of minorities. The Brownson data  
3 was 100 percent white women. Whether or not one is  
4 generalizable to the other without an adjustment is also a  
5 question.

6 MR. FURR: I agree. The point I want to get to is  
7 in OSHA's reliance upon the Fontham risk estimate, they  
8 don't acknowledge the uncertainty created by the differences  
9 between the Fontham risk estimates and the Brownson risk  
10 estimates, do they?

11 Let me ask it this way.

12 Did OSHA perform any type of sensitivity analysis  
13 on its risk assessment that would include the estimates from  
14 the other data sources such as Brownson?

15 DR. BAYARD: I haven't seen one.

16 MR. FURR: It's not included in the proposed rule  
17 is it?

18 DR. BAYARD: Not that I've seen.

19 MR. FURR: You believe that performing such a  
20 sensitivity analysis is an important effort, don't you?

21 DR. BAYARD: If you can. I think probably a  
22 worthwhile exercise. You kind of have to identify all your  
23 parameters that, even your constants that you're putting in  
24 and try to develop a distribution for them. You're trying  
25 to develop a distribution out of a best estimate number that

1 you usually don't have any idea of what the distribution is.  
2 It's probably a worthwhile exercise. In fact we've thought  
3 of trying to do it ourselves.

4 MR. FURR: Do you have the final EPA risk  
5 assessment up there? I assume you do.

6 DR. BAYARD: I have it everywhere.

7 (Laughter)

8 MR. FURR: Could you turn to page 631?

9 (Pause)

10 I want to read you some material that I believe  
11 appears on page 631.

12 "All of these figures are based on calculations in  
13 which unknown parameter values are replaced with numerical  
14 estimates that are subject to uncertainty and departures in  
15 either direction cannot be precluded as unrealistic  
16 probabilities for the correct population risk estimate."

17 DR. BAYARD: I'm sorry, I can't find it.

18 MR. FURR: That is the third sentence in the first  
19 full paragraph on the page.

20 (Pause)

21 MR. FURR: Rather than read it again, I'll let you  
22 read it to yourself and then I want to ask you a question  
23 about it.

24 (Pause)

25 DR. BAYARD: Just that one sentence?

1 MR. FURR: Yes.

2 (Pause)

3 MR. FURR: My question is, wouldn't you agree that  
4 that statement applies equally to OSHA's risk assessment?

5 (Pause)

6 DR. BAYARD: I'm sorry. Something is causing me  
7 to blank out here.

8 "All of these figures are based on calculations on  
9 which unknown parameter values are replaced with numerical  
10 estimates that are subject to uncertainty."

11 (Pause)

12 DR. BAYARD: It looks familiar, yes.

13 MR. FURR: Wouldn't you agree that that statement,  
14 the principle embodied in that statement applies equally  
15 well to OSHA's risk assessment?

16 DR. BAYARD: I think so.

17 MR. FURR: I want to talk to you about another  
18 source of uncertainty in OSHA's risk assessment. That's the  
19 uncertainty introduced by the assumptions that are made  
20 about exposure in the risk assessment.

21 When Drs. Ford and Samet were here, they told us  
22 that a relative risk estimate is meaningful only in the  
23 context of the exposures that are correlated with that risk  
24 estimate. You agree with that, don't you?

25 DR. BAYARD: Yes.

1 MR. FURR: That's really simply another way of  
2 saying that here's a dose response curve.

3 DR. BAYARD: What it means is, it means to me that  
4 the argument that a 1.19 for a relative risk for lung cancer  
5 is unrealistically small, and therefore, you can't make an  
6 identification out of it, has to be taken in the context of  
7 what's the exposure.

8 For example, a relative risk from active smoking  
9 might be 20 because an exposure to active smoking has such a  
10 much higher dose. Is that what you mean?

11 MR. FURR: Actually, I don't know what question  
12 you were answering, but...

13 DR. BAYARD: Sorry.

14 (Laughter)

15 MR. FURR: You already answered the first  
16 question. I wish I'd stopped there.

17 DR. BAYARD: Okay.

18 MR. FURR: You would further agree with Dr. Ford's  
19 testimony, wouldn't you, that to the extent that workplace  
20 ETS exposure is different than the exposures that were  
21 studied in the spousal smoking studies, that the workplace  
22 risks might be different?

23 DR. BAYARD: I think that's a general statement.

24 MR. FURR: On January 5th in these hearings, Dr.  
25 Roger Jenkins of the Oak Ridge National Laboratories

1 testified about a study that Oak Ridge recently completed.  
2 He testified that his data demonstrate that the potential  
3 exposure of ETS in the home is greater by a factor of four  
4 to six than the potential exposure in the occupational  
5 setting.

6 If Dr. Jenkins is correct, then OSHA's reliance on  
7 the spousal smoking studies in general and on the Helsing  
8 study in particular where they rely on the Helsing risk  
9 estimate for the quantitative risk assessment, would tend to  
10 overestimate the risk from occupational exposure, wouldn't  
11 it?

12 DR. BAYARD: Do you mean Helsing or do you mean  
13 Fontham?

14 JUDGE VITTON: Jenkins.

15 (Laughter)

16 MR. FURR: I mean the Helsing risk estimate,  
17 because the Helsing risk estimate came from a spousal  
18 smoking study.

19 DR. BAYARD: Oh, I see.

20 What you have to do is extrapolate time activity  
21 patterns. I mean say your workplace levels and your home  
22 levels are the same. For a minute, we're going to allow the  
23 first clause of your statement to stand, and that is that  
24 assuming Jenkins is right.

25 MR. FURR: That they're not the same.

1 DR. BAYARD: Maybe we'd better back up and let you  
2 ask the question again.

3 MR. FURR: Dr. Jenkins testified that his data  
4 showed that potential ETS exposures in the home were  
5 probably four to six times greater than potential ETS  
6 exposures in the workplace.

7 If he's correct, and I'm not asking you whether  
8 you think he's correct.

9 DR. BAYARD: Okay.

10 MR. FURR: If he's correct, wouldn't OSHA's  
11 reliance on a risk estimate from a spousal smoking study  
12 which is a home exposure study, tend to over-estimate the  
13 risks from occupational exposure?

14 DR. BAYARD: The reason I'm having trouble  
15 answering your question is because I don't remember how OSHA  
16 took the Helsing data and extrapolated it to the workplace.  
17 That's the only reason I'm having trouble.

18 MR. FURR: This question assumed that OSHA used  
19 the central risk estimate from Helsing's, ever exposed from  
20 the spousal smoking study, without any adjustment  
21 whatsoever.

22 DR. BAYARD: Okay.

23 MR. FURR: If they did that, wouldn't that result  
24 in an over-estimation of risk from the occupational setting  
25 if Jenkins is correct?

1 DR. BAYARD: The answer is, as I see it, yes.  
2 But, you're assuming that the Jenkins sample was a  
3 representative sample. You're assuming the Helsing sample  
4 was a representative sample. And that those two  
5 representative samples somehow encompassed what home  
6 exposures are and what work exposures are, and that the  
7 Helsing study would dovetail with the Jenkins study. The  
8 Helsing people who were exposed at home would actually be  
9 the same, be representative of the Jenkins people who were  
10 exposed at home. So making all those assumptions, I agree  
11 with you 100 percent.

12 MR. FURR: You've hit on a very important point.  
13 That is, this usage of the spousal smoking studies to  
14 predict occupational risk depends on a number of  
15 assumptions, and those include the assumption that the  
16 exposures in the spousal smoking studies were the same as  
17 the exposures in the current U.S. work force.

18 DR. BAYARD: It's harder for me to remember  
19 exactly what OSHA did. If you're just taking average  
20 exposures, that's one thing. If you're taking exposures at  
21 different levels, then you can make proper extrapolations.

22 Let me just try one more thing, and I'll quickly  
23 try to leave.

24 Part of my testimony was that the exposure levels  
25 in the workplace and the exposure levels in home tend to

1 average about the same. But depending on the sample that  
2 you take, you can get people who are highly exposed or  
3 people who are very low exposed. So if you see a study like  
4 Jenkins where he doesn't have representative samples, I  
5 think he testified that he wasn't talking about his  
6 representativeness of the sample, I mean he could be pulling  
7 in people who were very likely exposed.

8 What did he say, that 12 percent of his work group  
9 had about, worked in places with no smoking restrictions?  
10 I'm starting to lose it.

11 The argument I'm making is that it appeared that  
12 Jenkins' sample was not representative of the workplace  
13 sample.

14 Now I don't know how representative... Of the  
15 workplace distribution of smokers, or smoking exposure.

16 I don't know how representative Helsing is because  
17 I just don't know that database.

18 MR. FURR: And because we don't know how  
19 representative Helsing is, that's yet another source of  
20 uncertainty, isn't it?

21 DR. BAYARD: Because I don't know how  
22 representative it is doesn't mean that OSHA doesn't.

23 MR. FURR: Unless it's 100 percent perfectly  
24 statistically representative of the current U.S. work force,  
25 then reliance on that risk estimate introduces some

1 uncertainty into the risk assessment, doesn't it?

2 DR. BAYARD: The answer is yes, but you have to  
3 start some place.

4 JUDGE VITTON: Mr. Furr, let me ask you a  
5 question.

6 Is there a good point within the next few minutes  
7 here to break for lunch?

8 MR. FURR: In 10 to 15 minutes I would be at a  
9 breaking point.

10 JUDGE VITTON: All right.

11 How are you doing?

12 DR. BAYARD: I would like to break fairly soon  
13 myself. Could we go another five or ten, is that all right?

14 MS. SHERMAN: Do you want to break now, before  
15 lunch?

16 DR. BAYARD: I find that my mind isn't working  
17 very well right now.

18 MR. FURR: I won't comment, Your Honor.

19 (Laughter)

20 MR. FURR: Let's have lunch, if his mind's not  
21 working well.

22 JUDGE VITTON: One hour. 1:15.

23 (Whereupon, at 12:15 p.m. the hearing was  
24 recessed, to reconvene at 1:15 p.m. this same day, Friday,  
25 March 10, 1995.)

AFTERNOON SESSION

1

JUDGE VITTON: On the record.

2

3

We resume our questioning of Dr. Bayard with Mr.

4

Furr.

5

MR. FURR: Good afternoon, Dr. Bayard.

6

DR. BAYARD: Thank you.

7

MR. FURR: Is your mind working better?

8

DR. BAYARD: I hope so.

9

(Laughter)

10

MR. FURR: Let's turn to a new topic. That's the

11

topic of the occupational studies versus the home studies,

12

and which are preferable for purposes of conducting an

13

occupational risk assessment.

14

You're aware, aren't you, that OSHA's policy is

15

that when occupational data is available, that OSHA prefers

16

to use occupational data to perform risk assessments for the

17

workplace.

18

DR. BAYARD: I didn't know it was a policy, but I

19

would imagine it would be. Furthermore, occupational levels

20

are so much higher than typical environmental levels that

21

it's probably the reasonable thing to do, to make an

22

extrapolation.

23

First of all, ordinarily on occupational

24

chemicals, you never get testing in the homes; but

25

environmental tobacco smoke really isn't an occupational

1 chemical, it just happens to be there in the workplace.

2 MR. FURR: You've put your finger right on it.  
3 That policy makes sense because it eliminates the  
4 uncertainty associated with extrapolating risk from one set  
5 of exposure conditions to another set of exposure  
6 conditions. That's why we use occupational data, isn't it?

7 DR. BAYARD: I think you asked two questions. Let  
8 me try the first one first.

9 The first one was it eliminates uncertainty from  
10 going from one setting to an occupational setting. That's  
11 true. I believe that's correct in the occupational  
12 settings, that's fine because you have high levels of  
13 exposure to occupational compounds. But I don't see the  
14 major problem with going from spousal exposure studies to...

15 MR. FURR: I haven't asked you about that.

16 DR. BAYARD: I'm sorry.

17 MR. FURR: I appreciate your attempt to explain  
18 your answer. The only thing I would ask is that you try to  
19 keep those explanations relevant to the question that I ask  
20 instead of some other point.

21 DR. BAYARD: Maybe my mind isn't working yet.

22 MR. FURR: You've taken the position today that  
23 the spousal studies are more reliable for estimating  
24 occupational risks than are the occupational data. Correct?

25 DR. BAYARD: No. I don't think I've taken that

1 position. I think what I've taken is the position that  
2 spousal studies are more reliable for hazard identification.

3 I suggested... Than are studies for occupational,  
4 for the reasons that I've presented, and I won't go into  
5 them right now.

6 What I haven't said, at least I don't think I've  
7 said definitively, is that the spousal studies should be  
8 used to make estimates of population, of workplace... I'm  
9 sorry.

10 What I haven't said is that spousal studies should  
11 not be used for occupational population estimates of risk  
12 because what I thought I presented in one of my first slides  
13 is that typical studies in the home... I'm sorry. Typical  
14 exposure levels in the home are about the same as typical  
15 exposure levels where smoking is permitted in a workplace,  
16 but the workplace varies much more.

17 MR. FURR: Let me make sure I understand this. I  
18 think we're shortcutting some of this examination maybe.

19 So your position is that the spousal studies are  
20 not preferable to the occupational studies for the  
21 quantitative estimate of risk in the workplace.

22 DR. BAYARD: Not necessarily. That is what I've  
23 said. One has to look at the database and try to get the  
24 best studies they can. I think in the best of all worlds  
25 what I would like to do is get an estimate of risk from

1 bars, say, and taverns, because their exposure levels are so  
2 high; then I'd like to get one from places where you don't  
3 see much...

4 MR. FURR: Having those type of estimates would  
5 eliminate some of the uncertainty in the current risk  
6 assessment, wouldn't it?

7 DR. BAYARD: I think to the extent that you're  
8 getting better estimates of exposure in these places. But  
9 that doesn't mean that you can't take risk estimates from  
10 spousal studies and extrapolate them to risk estimates in  
11 the workplace.

12 For example, if I have spousal studies, which I  
13 feel are probably the best I can do in terms of getting  
14 exposure levels in the home. Then I should be able to  
15 extrapolate to comparable exposure levels, to workplaces of  
16 comparable exposure levels.

17 I think you're right in the sense that there's  
18 always some uncertainty. Where you have exposure levels  
19 which are roughly the same, then it becomes a matter of how  
20 much time you're spending in one place versus another place.  
21 That's all.

22 MR. FURR: I want to talk for just a moment or two  
23 about OSHA's reliance on the Helsing study for the risk  
24 estimate for the cardiovascular portion of the risk  
25 assessment.

1 I take it you would agree that if OSHA is going to  
2 rely on the spousal smoking study for the risk estimate,  
3 that they at least need to make a smoking status  
4 misclassification adjustment to that risk estimate. Just  
5 like you did in the EPA report.

6 DR. BAYARD: Can I think about that for just a  
7 minute. It sounds right, can I just think about it?

8 MR. FURR: Sure.

9 (Pause)

10 DR. BAYARD: In general, I would agree, except  
11 that the Helsing study, from what I remember of it, it was  
12 just part of the MR. FIT study, and that was a clinical  
13 trial where status was a lot more accurately determined.

14 While I agree with you in general, I don't know if  
15 I agree with you in the Helsing study because I haven't read  
16 the study close enough myself.

17 MR. FURR: You know that OSHA did not make any  
18 adjustment to the Helsing risk estimate for smoking status  
19 misclassification in the proposed rule, don't you?

20 DR. BAYARD: I didn't know that, but I take your  
21 work for it.

22 MR. FURR: I want to focus on a different type of  
23 exposure issue in the quantitative risk assessment. That's  
24 the issue of whether exposures have changed over time.

25 Could you look at page 511 of your EPA report?

1 (Pause)

2 DR. BAYARD: Can I make a correction before that?

3 The MR. FIT study was the Svensen and it wasn't  
4 the Helsing study.

5 MR. FURR: We know that.

6 DR. BAYARD: Why didn't you tell me?

7 (Laughter)

8 MR. FURR: It's your evidence, Dr. Bayard. Not  
9 mine.

10 JUDGE VITTON: What is the page number, Mr. Furr?

11 MR. FURR: 511.

12 (Pause)

13 MR. FURR: On that page, while discussing the  
14 utility of various studies for use in the risk assessment,  
15 EPA states that the earlier study results are more uncertain  
16 for projection of current risk. Do you see that?

17 DR. BAYARD: No.

18 MR. SHEEHAN: Which paragraph?

19 DR. BAYARD: Yes, I do.

20 MR. FURR: The middle sentence, middle paragraph.  
21 Do you see that statement?

22 DR. BAYARD: Yes, sir.

23 MR. FURR: You agree with that, don't you?

24 (Pause)

25 MR. FURR: Are there parts of the report that you

1 don't agree with?

2 DR. BAYARD: Just want to look at this. I'll  
3 answer the first question.

4 MR. FURR: The only question is, you agree with  
5 that, don't you?

6 DR. BAYARD: I'm sorry. I have to agree with it  
7 in a certain context. The earlier studies are more...

8 MR. FURR: How about in the context it's written?

9 DR. BAYARD: That's why I'm reading it, thanks.

10 MR. FURR: That's because exposure conditions,  
11 among other things, may have changed since the time the  
12 earlier study were conducted.

13 MR. SHEEHAN: Mr. Furr. I'm not sure he's  
14 answered the question yet. He's still reading.

15 (Pause)

16 DR. BAYARD: I'm sorry.

17 (Pause)

18 MR. FURR: Is this counting as my time?

19 JUDGE VITTON: Dr. Bayard, do you understand the  
20 question? The question is relatively simple.

21 DR. BAYARD: I'm trying to understand the context  
22 of it. I think we're understanding the context in terms of  
23 the cohort studies rather than the case control studies.

24 MR. FURR: You would agree that that principle  
25 applies to the case control studies equally, wouldn't you?

1 DR. BAYARD: No. That's why I'd like to stay in  
2 one and then we'll get on to the other.

3 I think what it's saying, and it just doesn't make  
4 any sense to me.

5 MR. FURR: Let me ask you a question about what  
6 it's saying, okay?

7 Isn't it that earlier studies may have been  
8 conducted under different exposure conditions than currently  
9 exist, and therefore are less reliable for the projection of  
10 current risk?

11 DR. BAYARD: If we can interpret it that way, then  
12 that's okay.

13 MR. FURR: Great.

14 When Dr. Jenkins testified, he told us that in  
15 general, the levels of ETS to which individuals are exposed  
16 today are substantially lower than the marker studies  
17 indicate that they were exposed to in the past.

18 Now if that's correct, relying on the studies of  
19 risk under historical exposure conditions would tend to  
20 over-estimate the risk posed by current exposure conditions,  
21 wouldn't it?

22 DR. BAYARD: Yes, unless you do the proper  
23 extrapolation. Yes.

24 MR. FURR: Pulling the central risk estimate from  
25 studies conducted under historical exposure conditions and

1 using it as though it applies perfectly to the current  
2 workplace, would tend to over-estimate the risk if exposures  
3 have changed.

4 DR. BAYARD: Yes, but I think you've got to talk  
5 about prevalence versus intensity, but why don't I let you  
6 go on.

7 MR. FURR: EPA submitted drafts of the risk  
8 assessment to its Scientific Advisory Board for review,  
9 didn't it?

10 DR. BAYARD: Yes.

11 MR. FURR: A subcommittee of the SAB held public  
12 hearings in 1990 and 1992.

13 DR. BAYARD: Yes, sir.

14 MR. FURR: You're aware that transcripts were  
15 generated from those public hearings?

16 DR. BAYARD: Yes, sir.

17 There were also reports generated from those  
18 hearings.

19 MR. FURR: Dr. Bayard, I want to hand you a copy  
20 of the transcript generated from the 1992 public hearing of  
21 the SAB and ask you to look at page 2-29.

22 (Document was handed to Dr. Bayard)

23 (Pause)

24 MR. FURR: Have you found that page?

25 DR. BAYARD: Yes, sir.

1 MR. FURR: Isn't it true that during the SAB's  
2 1992 hearing, Dr. Hammond who was OSHA's witness on exposure  
3 in this hearing testified that, "I think there have been  
4 significant changes over the last decade in ETS exposure in  
5 general."

6 DR. BAYARD: Yes.

7 MR. FURR: Didn't she go on to say that to "look  
8 at workplace exposures today would be quite misleading in  
9 terms of interpreting workplace exposures of ten years ago"?

10 DR. BAYARD: Yes.

11 MR. FURR: Thank you.

12 Let's sum this point up, Dr. Bayard. Even if one  
13 believes that the Fontham and Helsing central estimates were  
14 perfectly valid estimates of the risk in those studies, the  
15 use of those risk estimates to conduct a risk assessment for  
16 the current U.S. work force depends upon a number of  
17 assumptions about the similarity of exposure.

18 DR. BAYARD: The answer is yes, and I think it's  
19 just a matter of extrapolating.

20 MR. FURR: I want to talk about another source of  
21 uncertainty in OSHA's risk assessment, and that's the  
22 background population incidence rates...

23 DR. BAYARD: Yes.

24 MR. FURR: ...for lung cancer and hear disease  
25 that OSHA used in the risk assessment.

1           The incidence rates for both lung cancer and heart  
2 disease are highly dependent on age, race, and sex, aren't  
3 they?

4           DR. BAYARD: Yes.

5           MR. FURR: If the age, race, and sex of the  
6 distributions of the populations from which OSHA withdrew  
7 its background incidence rates is different than the age,  
8 sex, and race distributions of the current U.S. workplace,  
9 then that is yet another source of uncertainty in the risk  
10 assessment, isn't it?

11           DR. BAYARD: I think with respect to the way OSHA  
12 drew, OSHA's formula is on page 15995, I believe. The  
13 background lung cancer rates are lung cancer rates in the  
14 population. Then they're extrapolated to workers by a  
15 relative risk from the workplace transformed to attributable  
16 risk and then apportioned by prevalence.

17           The answer I would give to you is that the rates  
18 from the Garfinkel study which are the female lung cancer  
19 rates are properly used in that circumstance, I believe.  
20 Because the formula doesn't call directly for workplace lung  
21 cancer rates. The workplace lung cancer rates are derived  
22 by use of the overall non-smoking lung cancer rates and the  
23 relative risk derived from the Fontham study.

24           MR. FURR: So the population risk estimates that  
25 OSHA generated may not really have been specific to the

1 workplace then.

2 DR. BAYARD: Oh, yes they were. They were  
3 specific to the workplace in this case because the formula  
4 extrapolated them to that.

5 MR. FURR: Is it your testimony that OSHA made any  
6 adjustment to the background incidence rates for heart  
7 disease and lung cancer other than just pulling it from the  
8 general population studies?

9 DR. BAYARD: Can we talk about lung cancer first?

10 MR. FURR: Sure.

11 DR. BAYARD: What OSHA did, as I see it, they got  
12 the women's nonsmoking lung cancer rates from a separate  
13 study. They then used a relative risk from a workplace  
14 exposure study which was the Fontham study so they could say  
15 well, if the background is such and such, then we can  
16 determine the effect of lung cancer, the lung cancer rates  
17 for workers based on the fact that the workplace risk is 1.3  
18 times the general population risk.

19 MR. FURR: Dr. Bayard, are you familiar with a  
20 report titled "Choices in Risk Assessment, The Role of  
21 Science Policy in the Environmental Risk Management  
22 Process," that was prepared by Sandia National Laboratories?

23 DR. BAYARD: I am familiar with that report, but I  
24 didn't think it was prepared by the laboratories. I thought  
25 it was prepared by a contractor. Am I right on that?

1 MR. FURR: Well, I guess you are if that's how you  
2 want to describe it. It says it was prepared for Sandia  
3 National Laboratories.

4 DR. BAYARD: Steve Malloy is certainly not a  
5 contractor for Sandia Laboratories.

6 MR. FURR: Do you have a copy of that report with  
7 you?

8 DR. BAYARD: No, I don't.

9 MR. FURR: Let me hand you one.

10 (Report handed to Dr. Bayard)

11 MR. FURR: I've handed you only Chapter 10 of that  
12 report. I'd ask you to look at page 195.

13 (Pause)

14 MR. FURR: Chapter 10 of that report is titled  
15 "Workplace Indoor Air Quality" isn't it?

16 DR. BAYARD: Yes.

17 MR. FURR: Page 195, the report concludes that the  
18 estimated risk of lung cancer from occupational exposure  
19 depends on, and I want to read a quotation to you...

20 DR. BAYARD: Mr. Furr, just steer me in the  
21 general direction of the page when you read it.

22 MR. FURR: I'm going to read you the burger dot  
23 portions on page 195, and the language introducing the  
24 burger dots.

25 MR. SHEEHAN: Would you give him a minute to look

1 at it first?

2 MR. FURR: Why doesn't he look at while I read it  
3 so we can keep moving? Then if he needs to think about it,  
4 we'll think about it.

5 Doesn't that report state that "multiple and  
6 compounded science policy decisions concerning 1, the  
7 characterization of the available epidemiology..."

8 DR. BAYARD: I'm sorry. Just tell me where this  
9 is so I can follow with you.

10 JUDGE VITTON: Isn't he just reading it?

11 MR. FURR: Yes.

12 DR. BAYARD: Can't see it.

13 MR. FURR: Begin with the sentence with the single  
14 burger dot.

15 DR. BAYARD: The estimated benefits?

16 MR. FURR: Yes. Let me back up so there's no  
17 confusion on the record.

18 You agree that this is a chapter titled "Workplace  
19 Indoor Air Quality."

20 DR. BAYARD: Yes.

21 MR. FURR: It's addressing the OSHA quantitative  
22 risk assessment on ETS.

23 DR. BAYARD: It's more than the quantitative risk  
24 assessment. Isn't it addressing the whole proposed rule?

25 MR. FURR: It is, but the portion I'm focusing on

1 has to do with the quantitative risk assessment.

2 Let's read that together.

3 "The estimated benefits depend entirely on  
4 multiple and compounded science policy decisions concerning  
5 the characterization of the available epidemiology for ETS  
6 exposure; the estimated increase in risk attributable to ETS  
7 exposure; and point estimates for occupational risk of lung  
8 cancer attributable to ETS exposure; and the estimated  
9 background rate of lung cancer, number of nonsmoking U.S.  
10 workers exposed, and number of nonsmoking U.S. workers  
11 exposed to ETS."

12 These are the parameters that we've been  
13 discussing this morning with respect to the uncertainty that  
14 those parameters introduce into the work assessment, aren't  
15 they?

16 DR. BAYARD: That's correct.

17 MR. FURR: Let's go to the next paragraph.

18 Doesn't the report state that, "Depending on the  
19 science policy decisions made, the estimated risk of lung  
20 cancer from occupational exposure to ETS ranges from zero to  
21 OSHA's estimates. Based on the science policy decisions  
22 made by OSHA, OSHA's risk estimates are not more plausible  
23 than an estimate of zero risk."

24 That's what the report states, isn't it?

25 DR. BAYARD: Yes. Basically, though. It

1 basically looks like junk.

2 MR. FURR: Excuse me?

3 DR. BAYARD: It basically looks like junk.

4 MR. FURR: I have no idea what that means. But my  
5 question is, you don't disagree with that summary, do you?

6 DR. BAYARD: This summary?

7 MR. FURR: Yes.

8 DR. BAYARD: Yes, I do disagree with it.

9 MR. FURR: Dr. Bayard, when EPA submitted its  
10 draft risk assessments to the Scientific Advisory Board, it  
11 also submitted to the SAB specific questions that it wanted  
12 the SAB to answer, didn't it?

13 DR. BAYARD: Yes, sir.

14 MR. FURR: In 1992, wasn't one of those questions  
15 posed to the SAB by EPA, whether the assumptions used to  
16 derive the lung cancer population estimates and  
17 uncertainties involved were characterized adequately. That  
18 was one of the questions posed, wasn't it?

19 DR. BAYARD: I actually have that here.

20 MR. FURR: Doesn't that sound familiar to you?

21 DR. BAYARD: It sounds familiar, but I'd rather be  
22 exact because I want to be prepared and see what they  
23 responded to us.

24 MR. FURR: We're going to get to that right now.  
25 You have with you, don't you, the November 20,

1 1992 report of the SAB?

2 You submitted it to the record, I believe.

3 DR. BAYARD: Yeah, and I brought a copy with me.

4 (Pause)

5 MR. FURR: I'd ask you to turn to page...

6 JUDGE VITTONI: Mr. Furr, is he going to need any  
7 of these other papers?

8 MR. FURR: We may be going back to some of them.  
9 That's why I asked him to attempt to keep a stack as we go.

10 DR. BAYARD: I purposely did bring a copy in, and  
11 I didn't bring that much, so I know I have it.

12 (Pause)

13 MR. FURR: Let me hand you one. I'd ask you to  
14 turn to page four.

15 (Document handed to Dr. Bayard.)

16 MR. FURR: In a subsection of that report titled  
17 "Lung Cancer Population Impact," at page four, letter I, the  
18 question posed to the SAB was, "Is the degree of confidence  
19 in these estimates as stated appropriately characterized?"  
20 Isn't it?

21 DR. BAYARD: Yes.

22 MR. FURR: Wasn't the SAB's response, "No."

23 DR. BAYARD: Yes.

24 MR. FURR: "The confidence in these estimates  
25 represented by the range of 2500 to 3300 deaths due to ETS

1 understates the uncertainties associated with each of the  
2 assumptions that went into the risk estimate. It indicates  
3 a much higher degree of precision than the 90 percent  
4 confidence interval surrounding the summary relative risk  
5 for spousal smoking in the U.S., the 1.19. There are other  
6 assumptions used in the risk assessment that increase the  
7 uncertainty."

8 That was the SAB's response.

9 DR. BAYARD: Yes.

10 MR. FURR: The SAB was fairly critical of the  
11 EPA's quantitative risk assessment, wasn't it?

12 DR. BAYARD: That's one statement that the SAB  
13 made. On other statements that they made, we don't see that  
14 critique at all. On page six they say, "Is the presented  
15 population impact of ETS on lower respiratory infections and  
16 asthma in children scientifically defensible."

17 MR. FURR: Dr. Bayard, that's not my question. My  
18 question has to do with lung cancer.

19 Your Honor?

20 DR. BAYARD: I'm sorry. I'm sorry, Jeff. Mr.  
21 Furr.

22 MR. FURR: You can call me Jeff.

23 JUDGE VITTON: For the record, let's call him Mr.  
24 Furr. After you're out of here you can call him Jeff.

25 Why don't you repeat the question again so we know

1 where we are.

2 MR. FURR: Dr. Bayard, the SAB was fairly critical  
3 of EPA's lung cancer quantitative risk assessment, weren't  
4 they?

5 DR. BAYARD: I would disagree with that. The  
6 reason I disagree is because you have taken out of context  
7 question number I on page four. But on page three, where  
8 there was a question on lung cancer population impact, the  
9 question was, F, "Is the approach used to derive estimates  
10 of U.S. female never-smoker lung cancer risks scientifically  
11 defensible?"

12 The answer was an unqualified, "Yes. The  
13 combination of U.S. epidemiologic studies of nonsmoking  
14 women married to smokers provide an appropriate and sound  
15 basis for estimating the relative risk of lung cancer  
16 associated with ETS among American women who have never  
17 smoked cigarettes."

18 Then...

19 MR. FURR: Dr. Bayard...

20 DR. BAYARD: I'm sorry.

21 Then on page H, they say, "Are the assumptions  
22 used to derive these lung cancer population estimates and  
23 uncertainties involved characterized adequately?"

24 "Yes," is the answer. "While the overall point  
25 estimate of approximately 3,000 total lung cancer deaths due

1 to ETS exposure annually in the United States is based on  
2 reasonable assumptions, the citation of a range of 2500 to  
3 3300 ETS related LCDs based on varying only one of the  
4 parameters involved in the estimation is misleading and  
5 implies a greater degree of precision in the estimation than  
6 is warranted. The document would be strengthened by  
7 additional acknowledgement and characterization of these  
8 uncertainties."

9 So my response to you is they agreed with our  
10 estimate of central tendency, which was 3,000. They  
11 disagreed with our characterization of the uncertainty.

12 Thank you.

13 MR. FURR: Dr. Bayard, the SAB subcommittee that  
14 reviewed the ETS risk assessment included Dr. Blot at the  
15 National Cancer Institute, didn't it?

16 DR. BAYARD: That's correct.

17 MR. FURR: Dr. Blot is an epidemiologist?

18 DR. BAYARD: I characterize him more as a  
19 biostatistician, but he may be an epidemiologist also.

20 MR. FURR: You're aware that he has been the  
21 principal investigator of at least one of the ETS  
22 epidemiologic studies?

23 DR. BAYARD: That's correct.

24 MR. FURR: You believe him to be an expert in  
25 epidemiology and risk assessment, don't you?

1 DR. BAYARD: Yes.

2 MR. FURR: And you believe that he made a valuable  
3 contribution to the SAB's review of the EPA's risk  
4 assessment, don't you?

5 DR. BAYARD: Yes.

6 MR. FURR: Do you still have with you a copy of  
7 the 1992 SAB transcript?

8 MR. SHEEHAN: You mean report?

9 MR. FURR: Transcript. Have I not handed you the  
10 transcript yet?

11 DR. BAYARD: Yes.

12 MR. FURR: Yes, I have.

13 I ask you to turn to page 2-99, the bottom of the  
14 page.

15 (Pause)

16 DR. BAYARD: "While the overall estimate of  
17 approximately 3,000"?

18 MR. FURR: No, actually I'm going to focus on a  
19 slightly different part.

20 Isn't it true there that Dr. Blot stated during  
21 the hearing that EPA's quantitative risk assessment "tends  
22 to ignore the sampling variation inherent in the estimate of  
23 the relative risk of lung cancer among nonsmoking women  
24 married to smokers versus nonsmokers. Taking this variation  
25 alone into account would result in a confidence interval

1 based range for the annual ETS induced LCDs as low as a few  
2 hundred or less."

3 DR. BAYARD: That's correct.

4 MR. FURR: Then Dr. Blot...

5 DR. BAYARD: I'm sorry. Could we just finish  
6 that? "To as high as 5,000 or more."

7 MR. FURR: Didn't he go on to state that, "Other  
8 sources of uncertainty associated with each assumption  
9 employed would tend to widen the potential range even  
10 further?"

11 DR. BAYARD: I guess that wasn't Dr. Blot. That  
12 was Dr. Littman.

13 MR. FURR: Who chaired the SAB committee.

14 DR. BAYARD: Yes, but you were talking about Dr.  
15 Blot.

16 MR. FURR: I apologize. You're right. That was  
17 the chairman of the SAB subcommittee that reviewed the risk  
18 assessment, wasn't it.

19 DR. BAYARD: Okay.

20 MR. FURR: Dr. Blot and Dr. Littman were not the  
21 only SAB members to be critical of EPA's quantitative risk  
22 assessment, were they?

23 Can you turn to page 2-109 of the transcript?

24 DR. BAYARD: The answer to the first question was,  
25 the concept that you're presenting to me is the critique of

1 the way EPA characterized uncertainty in its draft. The  
2 concept of the central tendency of the estimate of 3,000, we  
3 see to see universal agreement here. But the measures of  
4 central tendency, the measures of uncertainty they said were  
5 improperly characterized and the EPA agreed with them. The  
6 EPA went back and revised the estimates based on the SAB  
7 report to us.

8 MR. FURR: My question is, weren't there other SAB  
9 members that were critical of EPA's treatment of uncertainty  
10 in the quantitative risk assessment? You can turn to page  
11 2-109, if you would.

12 (Pause)

13 MR. FURR: Dr. Kabat was also a member of the SAB  
14 subcommittee that reviewed the risk assessment wasn't he?

15 DR. BAYARD: Yes.

16 MR. FURR: He's an epidemiologist...

17 DR. BAYARD: Yes.

18 MR. FURR: ...who has been a principal  
19 investigator in at least two epidemiologic studies of ETS.

20 DR. BAYARD: Yes.

21 MR. FURR: You view him as an expert in  
22 epidemiology and risk assessment, don't you?

23 DR. BAYARD: Yes.

24 MR. FURR: And you believe that he made a valuable  
25 contribution to the SAB's review of the EPA's risk

1 assessment, don't you?

2 DR. BAYARD: Yes.

3 MR. FURR: Isn't it true that Dr. Kabat cautioned  
4 EPA about the extension of the female estimates to males and  
5 to former smokers. And in Dr. Kabat's words, "EPA's piling  
6 of assumption on top of assumption."

7 DR. BAYARD: That's correct.

8 MR. FURR: Thank you.

9 DR. BAYARD: I haven't finished that answer,  
10 please. I won't take long.

11 I think Dr. Kabat in this instance failed to  
12 realize that the only, that the male, we used female risk  
13 estimates to extrapolate to...

14 MR. FURR: Dr. Bayard, I'm really not asking you  
15 whether Dr. Kabat was right or wrong. I just want to probe  
16 whether or not there was any unanimity among the  
17 subcommittee as to whether EPA had properly handled  
18 uncertainty.

19 DR. BAYARD: Under...

20 MR. SHEEHAN: I think the witness...

21 JUDGE VITTON: Look, that's in the record. If  
22 we're going to get into an argument about what he  
23 misconstrued or...

24 MR. FURR: I'm not asking about that.

25 DR. BAYARD: I think he was asking about

1 unanimity, and as long as he's asking about unanimity, I  
2 want to say what there was unanimity on.

3 JUDGE VITTON: That's.. I told you you would  
4 have...

5 DR. BAYARD: I thought that was the question he  
6 asked me.

7 JUDGE VITTON: Doctor, you had an opportunity for  
8 an hour and forty minutes, I think, in your Direct  
9 Examination. I told you I'd give you an opportunity  
10 afterwards if you wanted to. But in order to keep the  
11 examination moving along, I would like you to keep your  
12 answers as directed as possible to the question, okay?

13 DR. BAYARD: Uh huh.

14 MR. FURR: Dr. Bayard, do you have a copy of the  
15 risk assessment guidelines of 1986?

16 DR. BAYARD: Yes, I do.

17 MR. FURR: I want to ask you a few questions about  
18 them, please.

19 EPA's designation of ETS as a Group A carcinogen  
20 was done pursuant to the scheme set forth in those  
21 guidelines, wasn't it?

22 DR. BAYARD: Yes, it was.

23 MR. FURR: I want to focus on that scheme, and in  
24 particular the differences in that scheme for classifying a  
25 potential carcinogen as either Group A or Group B-1.

1                   Can you turn to page 1-12 of the guidelines? It's  
2 51 Fed Reg 34000.

3                   (Pause)

4                   MR. FURR: Isn't it true that EPA classifies a  
5 substance as a Group A carcinogen only if there is  
6 "sufficient evidence from epidemiologic studies to support a  
7 causal association between exposure to the agent and  
8 cancer"?

9                   DR. BAYARD: That's correct.

10                  MR. FURR: The operative phrase there is  
11 sufficient evidence, correct?

12                  DR. BAYARD: That's correct.

13                  MR. FURR: In contrast, a substance is classified  
14 as a Group B-1 carcinogen if there is "limited evidence,"  
15 isn't that correct?

16                  DR. BAYARD: That's correct.

17                  MR. FURR: Don't the guidelines provide some  
18 guidance on how EPA is to evaluate whether the epidemiologic  
19 data should be considered sufficient versus limited?

20                  DR. BAYARD: That's correct.

21                  MR. FURR: That guidance includes three criteria,  
22 doesn't it? those being that before a causal association  
23 can be inferred between an exposure and cancer based on  
24 human studies, that is a Class A designation, Group A  
25 designation made that there must be no unidentified bias

1 that could explain the association; that the possibility of  
2 confounding has been considered and ruled out as explaining  
3 the association; and that the association is unlikely to be  
4 due to chance.

5 Is that correct?

6 DR. BAYARD: That's correct.

7 MR. FURR: In contrast, the guidelines provide  
8 that data should be considered to provide only limited  
9 evidence of carcinogenicity if alternative explanations such  
10 as chance, bias, and confounding cannot adequately be ruled  
11 out. Is that correct?

12 DR. BAYARD: That's correct.

13 MR. FURR: During his testimony, Dr. Ford told us  
14 that in Western culture, the scientific method places the  
15 burden on the investigator to disprove the null hypothesis  
16 and show that any differences in outcomes among the groups  
17 being compared are not due to random chance, bias, or  
18 confounding.

19 This same approach is reflected in EPA's  
20 guidelines, isn't it?

21 (Pause)

22 DR. BAYARD: No, I don't think so.

23 MR. FURR: Don't the guidelines place the burden  
24 on the investigator with respect to ruling out the  
25 likelihood of chance, bias, or confounding being an

1 explanation for the observed association?

2 DR. BAYARD: The guidelines, that's correct, but  
3 the guidelines also focus on a total weight of the evidence  
4 and not any one individual study.

5 MR. FURR: I agree.

6 Dr. Ford also testified that for epidemiologic  
7 studies, the burden in eliminating chance, bias and  
8 confounding increases as the relative risk being studied  
9 decreases. You agree with that also, don't you?

10 DR. BAYARD: In general, yes. That's not  
11 universally true because as you said before, the relative  
12 risk is a function of exposure. For example, we will bring  
13 back the example of active smoking. Active smoking has very  
14 high relative risk to it, but there's quite a large dose,  
15 and we then have, so that when we have lower doses, or lower  
16 exposures to the substance of tobacco smoke, we would expect  
17 a lower risk. For example, with...

18 MR. FURR: I don't know what question you're...

19 I must have asked a question that...

20 JUDGE VITTON: Doctor, I think we understand your  
21 answer. In general you agree with it, but there are  
22 situations where it doesn't always apply.

23 DR. BAYARD: The question was...

24 MR. FURR: Let me ask the question so we can move  
25 on.

1 JUDGE VITTON: I think you've answered the  
2 question, really.

3 Go ahead.

4 MR. FURR: In general, the smaller an association  
5 is, the more difficult it is to rule out bias, confounding,  
6 and chance as an explanation for that association.

7 DR. BAYARD: On any one study, right. I'd agree  
8 with that.

9 MR. FURR: Let's turn to another topic.

10 I want to ask you a few questions about whether or  
11 not there was ever any consensus, even within the  
12 Environmental Protection Agency with respect to the validity  
13 of EPA's risk assessment.

14 MR. SHEEHAN: Your Honor, I'm sorry. I think  
15 we're going to have to object to this line of questioning.

16 Dr. Bayard is here to testify about the report and  
17 what he did, what his role was in that report. He's not  
18 here to testify about the consensus of the agency or what  
19 other people in the agency thought. I don't think that's a  
20 proper line of questioning.

21 MR. FURR: Your Honor, that's a spurious argument.

22 Dr. Bayard is here to present the Environmental  
23 Protection Agency's evidence. His notice of appearance was  
24 given by someone else within the Environmental Protection  
25 Agency for the Environmental Protection Agency. Dr. Bayard

1 has arrived as that spokesperson.

2 I'm not interested at all in examining Dr. Bayard  
3 personally. I'm hoping to probe the EPA's evidence through  
4 Dr. Bayard, which is his identity in this hearing.

5 MS. NEUWIRTH: He is not here to answer anything  
6 about the internal deliberations of the agency, though.

7 JUDGE VITTON: What do you want to ask him?

8 MR. FURR: Dr. Bayard has presented the EPA's...

9 On numerous occasions, Dr. Bayard has attempted to  
10 deflect attacks on the Environmental Protection Agency's  
11 report by arguing that this report is just one more report.  
12 The Surgeon General, the NRC, IARC, and many others are in  
13 agreement with us. We have an agreement by consensus, and  
14 that adds weight to our findings somehow.

15 I don't have the Surgeon General, NRC, or IARC up  
16 here. I also don't have time to disassemble that complete  
17 argument. What I would like is the opportunity to probe  
18 whether there was even any consensus within the  
19 Environmental Protection Agency about the report.

20 There's a great deal of documentary evidence that  
21 there was no such consensus, and I would like the  
22 opportunity to ask Dr. Bayard about it.

23 JUDGE VITTON: It seems to me, Mr. Furr, you're  
24 dealing with the report itself. If the report is issued by  
25 the Environmental Protection Agency, that is the position of

1 the agency.

2 MR. FURR: What I'd like to do, Your Honor, is  
3 expose for the decisionmaker that there is a great deal of  
4 evidence that there is considerable disagreement within the  
5 agency about the scientific validity of the report so that  
6 the decisionmaker can consider the question of whether or  
7 not this report was released because of its scientific  
8 statements or as a policy statement by the agency.

9 JUDGE VITTON: Well, I think you can ask Dr.  
10 Bayard that final specific question, but I don't think, I  
11 know I'm not going to allow you to explore whether he agrees  
12 with another scientist in the agency or there was internal  
13 disputes. The report speaks for the agency, and I think  
14 it's for another forum to determine if that report is, how  
15 should I put it...

16 MR. FURR: Your Honor, let me make one further  
17 point on this that I think really should be decisive of this  
18 question.

19 In the statement that Dr. Bayard submitted today,  
20 on page two, in the section titled, "Background." The last  
21 sentence of the second paragraph, Dr. Bayard states that  
22 "The report was prepared by ORD's Office of Health and  
23 Environmental Assessment, OSHA, and was written with both  
24 in-house staff and outside contracting assistance. Before  
25 being released in draft form for public review, the passive

1 smoking report received many internal reviews, mostly within  
2 ORD."

3 Now the implication of that statement, and the  
4 only reason to make that statement, is that it somehow adds  
5 weight to the credibility and validity of the report. It  
6 also suggests that everyone who reviewed the report within  
7 the EPA signed off on the conclusions and analysis. I would  
8 like the opportunity, which Dr. Bayard has opened the door  
9 to, to probe that issue.

10 JUDGE VITTON: I don't think he's opened the door  
11 to that. I think the internal decisionmaking process, the  
12 discussions among the people involved in the report, I don't  
13 think are relevant to this proceeding. I'm going to ask you  
14 to move on.

15 MR. FURR: Your Honor, could I give you a one  
16 minute preview of where I'd like to go, because this is  
17 going to be no surprise to Dr. Bayard.

18 JUDGE VITTON: You can state it for the record,  
19 but you're using up your time. Go ahead.

20 MR. FURR: I think this is important enough to  
21 state for the record.

22 JUDGE VITTON: Okay.

23 MR. FURR: During the internal reviews, drafts of  
24 the Environmental Protection Agency's report were submitted  
25 to various offices outside of Washington within the EPA,

1 including offices in Cincinnati and Raleigh.

2 Written critiques of the reports were generated  
3 subsequently, by those offices. Those critiques are highly  
4 critical of the analysis and conclusions contained within  
5 the report, and include refusals to sign off on the report  
6 by those offices.

7 Those critiques also make a number of  
8 recommendations for changes to the report which EPA,  
9 Washington, or whoever was responsible for this report did  
10 not do.

11 I would also probe, if given the opportunity,  
12 whether or not EPA Washington, that being Dr. Bayard's  
13 group, even attempted to respond on the merits to the  
14 outside offices, or whether instead, it challenged the  
15 audacity of those offices to criticize its work.

16 Now all that seems highly relevant to me.

17 JUDGE VITTON: I don't agree. I don't think it's  
18 relevant to this proceeding. I think that's an issue for  
19 another forum.

20 MR. FURR: I'll move on.

21 JUDGE VITTON: Okay.

22 MR. FURR: Dr. Bayard, I want to talk to you about  
23 the issue of dose response that you spent some time on  
24 today.

25 EPA's position is that the identification of a

1 dose response relationship within the ETS data was a  
2 critical finding in determining whether or not ETS is a  
3 cause of lung cancer, isn't it?

4 DR. BAYARD: It was certainly an important one. I  
5 wouldn't say it's critical. There are a lot of analyses  
6 which I presented today.

7 MR. FURR: Let me hand you a copy of the  
8 transcript of a hearing held on July 21, 1993 before the  
9 Subcommittee on Specialty Crops and Natural Resources. You  
10 testified in that hearing, didn't you?

11 DR. BAYARD: Yes, sir.

12 MR. FURR: If I refer to that as Congressman  
13 Rose's hearing, you'll know what I'm referring to?

14 DR. BAYARD: Yes.

15 (The document was handed to Dr. Bayard.)

16 MR. FURR: Could I ask you to turn to page 22 of  
17 that transcript, please?

18 Did you find it?

19 DR. BAYARD: Yes.

20 MR. FURR: I'd ask you to focus on the question  
21 from Mr. Baselor in the middle of the page.

22 Mr. Baselor, after a series of questions about how  
23 you reached your decision, Mr. Baselor began asking you a  
24 question that reads, "You said you started in 1988," and  
25 before he finished, you began responding, and in part wasn't

1 your response, "So my answer was no. When I first started I  
2 didn't believe it at all. It was only when I saw the  
3 evidence on dose response trends and the epidemiology  
4 studies that I couldn't explain any other way."

5 Wasn't that your response?

6 DR. BAYARD: Yes.

7 MR. FURR: You'd agree that the dose response  
8 trend information was critical to your decisionmaking,  
9 wasn't it?

10 DR. BAYARD: It was critical to the way I...  
11 I'm sorry.

12 JUDGE VITTON: Go ahead.

13 DR. BAYARD: I'm just...

14 MR. FURR: Dr. Bayard...

15 DR. BAYARD: I'm sorry, I...

16 JUDGE VITTON: He didn't finish the answer yet.

17 DR. BAYARD: I'm just...

18 I think it changed the way I thought about the  
19 data because I couldn't explain that away. Before I saw the  
20 dose response data, it looked to me that there was very  
21 little evidence of an effect in epidemiologic studies, but I  
22 just couldn't explain that away.

23 I don't think it was critical in the final agency  
24 decision or the final SAB decision on unanimously endorsing  
25 the Group A carcinogenicity, but it did change the way I

1 started looking at the data.

2 MR. FURR: Dr. Bayard, didn't you tell the Rose  
3 subcommittee that you became convinced only when you saw the  
4 evidence on dose response? Isn't that what we just read?

5 DR. BAYARD: What we just read was, "So my answer  
6 was no. When I first started I didn't believe it at all.  
7 It was only when I saw the evidence and dose response trends  
8 in the epidemiology studies that I couldn't explain any  
9 other way." I certainly...

10 MR. FURR: Dr. Bayard, I'd like to hand you an EPA  
11 1994 publication called "Setting the Record Straight.  
12 Secondhand Smoke is a Preventable Health Risk," and ask you  
13 to turn to page three.

14 (Document handed to Dr. Bayard)

15 DR. BAYARD: Thank you.

16 (Pause)

17 MR. FURR: Dr. Bayard, that's an EPA publication  
18 isn't it?

19 DR. BAYARD: Yes.

20 MR. FURR: I'd ask you to look at the bottom  
21 paragraph, first column on page three under a heading, "The  
22 Epidemiology Studies."

23 DR. BAYARD: Yes.

24 MR. FURR: Doesn't that bottom paragraph state  
25 that, "Probably the most important finding for a causal

1 relationship is one of increasing response with increasing  
2 exposures since such associations cannot be explained by  
3 other factors."

4 DR. BAYARD: That was a general statement, and  
5 then I get more specific. Then the statement becomes more  
6 specific. The statement itself doesn't refer to the ETS  
7 studies, it refers to in general that when you see causal  
8 relationships they provide very strong criteria for the  
9 causality.

10 MR. FURR: You're not telling us that that  
11 statement was just thrown into that document without  
12 reference to the ETS epidemiologic studies, are you?

13 DR. BAYARD: It was a topical sentence, and the  
14 sentence said, it related to analysis of epidemiology  
15 studies. It says when you see dose response relationships,  
16 unless you can explain them away, they become very serious  
17 indicators that there's a causal effect.

18 MR. FURR: But this wasn't a document issued on  
19 general epidemiologic principles or a thought piece on the  
20 criteria for drawing causal inferences, was it? This was a  
21 document on the EPA studies. That statement appears in a  
22 column titled "Epidemiology," doesn't it?

23 DR. BAYARD: I don't think we have an argument  
24 here. All I'm saying is that the principle holds regardless  
25 of the ETS studies.

1 MR. FURR: Okay. In the ETS risk assessment, EPA  
2 performed a number of assessments to determine whether or  
3 not there were statistically significant exposure response  
4 trends, right? That's what you've talked about some today.

5 DR. BAYARD: That's correct.

6 MR. FURR: The trend test that EPA used is known  
7 as the Cochran-Armitage or Mantel-Haensel extension test,  
8 right?

9 DR. BAYARD: Some of them were. Some of them are  
10 based on logistic regression.

11 MR. FURR: Those trend tests were conducted on  
12 unadjusted data, weren't they?

13 DR. BAYARD: The Cochran-Armitage are. The  
14 Mantel-Haensel test was a Mantel-Haensel test. They were  
15 adjusted on the unadjusted data. The...

16 MR. FURR: In fact they can't be applied to  
17 adjusted data, can they?

18 DR. BAYARD: I'm sorry. The others were based on  
19 adjusted analyses.

20 MR. FURR: The Mantel-Haensel method cannot even  
21 applied to adjusted data, can it?

22 DR. BAYARD: It all depends. The Mantel-Haensel  
23 method is actually a method of adjusting the data. The  
24 adjusted data... I'm sorry, I'll stop there.

25 MR. FURR: Dr. Bayard, isn't it true that a number

1 of publications have reported that many potential  
2 confounders have reported spousal smoking lung cancer  
3 associations, are correlated with spousal smoking in a dose  
4 response fashion.

5 DR. BAYARD: Pleas repeat that. I'm sorry.

6 MR. FURR: Today when you were testifying you  
7 stated that EPA could find no evidence of confounding being  
8 correlated with spousal smoking exposure in a dose response  
9 fashion, didn't you?

10 DR. BAYARD: Intensity, that's correct.

11 MR. FURR: In fact there are several sources of  
12 that evidence aren't there?

13 DR. BAYARD: Please.

14 (Pause)

15 MR. FURR: Dr. Bayard, I want to hand you first,  
16 maybe I can make only one trip.

17 I want to hand you copies of two studies. One by  
18 Koo, published in 1988 titled "Life History Correlates of  
19 Environmental Tobacco Smoke, A Study of Nonsmoking Hong Kong  
20 Chinese Wives With Smoking Versus Nonsmoking Husbands."

21 And a copy of a paper by LaMarchand et al  
22 entitled, "Dietary Patterns of Female of Nonsmokers With and  
23 Without Exposure to Environmental Tobacco Smoke."

24 You've seen these papers before, haven't you?

25 DR. BAYARD: Yes, but it's been awhile.

1 (Documents handed to Dr. Bayard.)

2 MR. FURR: I'd ask you to look at the Koo paper  
3 first. Take a look at the abstract, if you would.

4 Didn't Koo report in 1988 that a number of disease  
5 risk factors were correlated with spousal smoke exposure  
6 with increasing intensity of risk factors among heavier  
7 smoking spouses?

8 (Pause)

9 MR. FURR: You see that in the abstract, don't  
10 you?

11 MR. SHEEHAN: He's reading.

12 (Pause)

13 MR. FURR: I think I highlighted it for you.

14 (Pause)

15 DR. BAYARD: This appears to be a study that seems  
16 to show some relationship of...

17 I'm sorry, let me read it again.

18 (Pause)

19 JUDGE VITTON: Let me hear the question again,  
20 Mr. Furr.

21 MR. FURR: Doesn't Koo report that a number of  
22 disease risk factors were correlated with spousal smoke  
23 exposure with increasing intensity of risk factors among  
24 heavier smoking spouses?

25 DR. BAYARD: It says "The former were better off

1 in terms of socioeconomic status, more conscientious  
2 housewives, ate better diets, and had better indices of  
3 family cohesiveness."

4 MR. FURR: Then it says "The differences were  
5 usually largest when comparing wives of never smoked versus  
6 heavily smoking husbands."

7 DR. BAYARD: Yes.

8 MR. FURR: Isn't that the type of evidence that  
9 one would look for to determine whether confounding may be  
10 correlated in a dose response fashion with the intensity of  
11 spousal smoking?

12 DR. BAYARD: That's correct.

13 MR. FURR: EPA did not consider that data in the  
14 risk assessment, did they?

15 DR. BAYARD: I think we actually do have it in our  
16 risk assessment. It's in our dietary information, I think.

17 MR. FURR: No, they did not consider that finding  
18 in the Koo study.

19 DR. BAYARD: We did not.

20 MR. FURR: It's not addressed anywhere in the EPA  
21 report, is it?

22 DR. BAYARD: I think it is.

23 MR. FURR: Well, let me ask the question  
24 differently because I don't want to get back into the  
25 report.

1           This morning you testified that no such evidence  
2           existed. Are you now ready to change that testimony?

3           DR. BAYARD: I'm going to read the paper again and  
4           see if I can agree with it.

5           MR. FURR: You know Linda Koo by reputation at  
6           least, don't you?

7           DR. BAYARD: I know very little about her. I've  
8           just read a couple of her papers.

9           MR. FURR: You believe that she's a reputable  
10          epidemiologist, don't you?

11          DR. BAYARD: Her papers are published.

12          MR. FURR: You don't have any question in your  
13          mind that the data in her report support that statement, do  
14          you?

15          DR. BAYARD: It seems reasonable to me, Mr. Furr.

16          MR. FURR: You don't have any question in your  
17          mind that the methods employed by Koo in obtaining and  
18          analyzing that data were correct, do you?

19          DR. BAYARD: I haven't read the paper closely  
20          enough.

21          MR. FURR: As you sit here today, do you have any  
22          reason to question Linda Koo's scientific abilities?

23          DR. BAYARD: I have no reason to question them  
24          because I have to go through the paper.

25          MR. FURR: I'd ask you to take a look at the

1 LaMarchand paper at page 14.

2 (Pause)

3 MR. FURR: I hope you got the highlighted copy  
4 again, did you?

5 DR. BAYARD: Yes, sir.

6 MR. FURR: Page 14, doesn't LaMarchand report that  
7 beta carotene intake was inversely related to ETS exposure  
8 among female nonsmokers in a linear fashion?

9 DR. BAYARD: I think that's right. Where was  
10 that? Page 14?

11 MR. FURR: Page 14.

12 DR. BAYARD: That's correct.

13 MR. FURR: Isn't that further evidence that  
14 confounding may be correlated with the intensity of spousal  
15 exposure in a dose response fashion?

16 DR. BAYARD: It's a question that it could be  
17 looked at. On the other hand...

18 (Pause)

19 MR. FURR: Your Honor, is EPA testifying as a  
20 panel today?

21 MR. SHEEHAN: Your Honor, I would suggest if Mr.  
22 Furr wants to ask him questions about what's in these  
23 documents, and he's just going to confirm what it says,  
24 that's one thing. But if he's going to testify about the  
25 document he needs to be given a little bit of time to at

1 least familiarize himself with it.

2 JUDGE VITTON: I'm giving him the time.

3 MR. FURR: I want to raise... I haven't objected.  
4 I've tried to not object to the behavior on the panel today.  
5 But my question is, is EPA testifying as a panel? If not,  
6 I'd prefer that Dr. Bayard not consult with other members of  
7 the panel in between questions.

8 JUDGE VITTON: Mr. Furr, they're just passing him  
9 documents back and forth. Other people have testified and  
10 have had people there and they've consulted with some of  
11 their other members of the panel sitting there, even if the  
12 other people have not testified.

13 MR. SHEEHAN: If you don't want us to pass  
14 documents back and forth it's going to take a little bit  
15 longer. We're trying to help Mr. Bayard get documents.

16 MR. FURR: My question s very clear. You're just  
17 taking up time. I don't mind documents.

18 MR. SHEEHAN: No, we're not taking up time.  
19 We're...

20 MR. FURR: I'm asking that Dr. Bayard not consult  
21 with other people unless I have a chance...

22 JUDGE VITTON: All right.

23 Dr. Bayard, do you have an answer?

24 DR. BAYARD: The answer is, I think the question  
25 was do I agree with the statement on page 14?

1 MR. FURR: No. Is that finding reported on page  
2 14?

3 DR. BAYARD: That finding is reported on page 14.

4 MR. FURR: That's a finding that was not taken  
5 into account in EPA's analysis of the studies, isn't it?

6 DR. BAYARD: Yes, it was.

7 MR. FURR: Can you point to me where in the risk  
8 assessment EPA addresses that finding?

9 Actually, I'll ask you to do that in a post-  
10 hearing comment, because you and I both know it can't be  
11 done, and I don't want to stand here for ten minutes while  
12 you leaf through the report.

13 DR. BAYARD: Well,...

14 MR. SHEEHAN: If he's going to ask the question, I  
15 think he has to give him a chance to...

16 JUDGE VITTON: Mr. Furr, you asked him the  
17 question. Give him the opportunity to respond to it.

18 DR. BAYARD: A finding of whether or not beta  
19 carotene intake determined by questionnaire on what appears  
20 by the diet of current beta carotene intake... Wait.

21 The question of whether or not...

22 MR. FURR: Dr. Bayard, I have to keep working  
23 while you're talking. Please proceed.

24 DR. BAYARD: Current beta carotene intake is  
25 linearly related to passive smoke exposure, makes it a

1 potential confounder if beta carotene intake is also  
2 determined to be a preventive factor for lung cancer in a  
3 dose dependent way.

4 That doesn't make it a confounder. It gives it a  
5 possibility of being a potential confounder.

6 There were several studies that did look at beta  
7 carotene intake. One of them was Kalandid, if I'm not  
8 mistaken. They found no interaction effect, or no  
9 confounding effect between beta carotene and passive  
10 smoking. So while it may be discussed in the LaMarchand  
11 paper as having some relationship to exposure to  
12 environmental tobacco smoke, the studies indicate, the  
13 actual environmental tobacco smoke lung cancer studies  
14 indicate that it is not a confounder.

15 The strongest study in that, I believe, is the  
16 Fontham 1994 paper which also looked at diet, cholesterol,  
17 and I believe beta carotene intake, and found no confounding  
18 effect between environmental tobacco smoke and lung cancer.

19 MR. FURR: Dr. Bayard, you would agree that the  
20 LaMarchand finding provides some evidence of confounding  
21 factors being correlated in a dose response fashion with ETS  
22 exposure, wouldn't you?

23 DR. BAYARD: A confounding factor is only a  
24 confounding factor in this study...

25 MR. FURR: A potential confounding factor.

1 DR. BAYARD: But that's why, a potential  
2 confounding factor, we did examine it on page 556. So we  
3 have discussed it.

4 MR. FURR: You're familiar with LaMarchand by  
5 reputation, at least, I take it.

6 DR. BAYARD: I'm only familiar with that paper.

7 MR. FURR: Do you have any reason as you sit here  
8 today to doubt that the data in that paper support the  
9 contentions being advanced by LaMarchand?

10 DR. BAYARD: I feel that's fine.

11 MR. FURR: Do you have any reason as you sit here  
12 to question the methodology employed in that paper?

13 DR. BAYARD: No.

14 MR. FURR: Dr. Bayard, you're familiar with a  
15 reference book on statistics published by Breslow and Day,  
16 aren't you?

17 DR. BAYARD: Yes.

18 MR. FURR: As a statistician you hold the work of  
19 Drs. Breslow and Day in high esteem?

20 DR. BAYARD: Quite competent.

21 MR. FURR: It's an authoritative reference in the  
22 field, isn't it?

23 DR. BAYARD: Yes.

24 MR. FURR: It is a reference that is quoted at  
25 length in the EPA risk assessment, isn't it?

1 DR. BAYARD: Yes.

2 MR. FURR: I want to hand you a copy of at least a  
3 part to that reference titled "Statistical Methods in Cancer  
4 Research," and ask you to turn to page 97.

5 Let me make sure I give you the highlighted copy  
6 to try to speed these up.

7 (Document handed to Dr. Bayard)

8 (Pause)

9 MR. FURR: Do you see the material that's been  
10 highlighted on page 97, Dr. Bayard?

11 (Pause)

12 JUDGE VITTON: Do you see it there, Doctor?

13 DR. BAYARD: Yes.

14 MR. FURR: Let me read that and you tell me, I'm  
15 going to try to excerpt some of the material. You tell me  
16 if I've captured the gist of that paragraph, okay?

17 DR. BAYARD: Yes.

18 MR. FURR: The object of a dose response analysis  
19 is to demonstrate a continuously increasing risk with  
20 increasing exposure. A trend statistic may sometimes give a  
21 significant result even if the relative risks are not  
22 continuously increasing. One may wish to restrict the trend  
23 statistic to a comparison of positive dose or duration  
24 levels, and exclude the baseline non-exposed category when  
25 testing specifically for a dose response effect.

1 I read that correctly didn't I, Dr.  
2 Bayard? (Inaudible)

3 DR. BAYARD: That's correct.

4 I would like to add the next paragraph, too.

5 MR. FURR: Doctor, I don't want to ask about the  
6 next paragraph.

7 JUDGE VITTON: There's no question, Doctor. Wait  
8 until we get a question, okay?

9 MR. FURR: In conducting its trend analyses, EPA  
10 did not restrict the analysis to the positive dose or  
11 duration levels, did it?

12 DR. BAYARD: That's correct. I would add that  
13 this argument was also brought up to our Science Advisory  
14 Board. It was brought up by Dr. Switzer and it was brought  
15 up I believe, also by Dr. LaVois.

16 MR. FURR: I'm just asking you what EPA did.

17 DR. BAYARD: Both consultants for the...

18 And Judge, I'm stating that the issue has been  
19 brought up before and that there are other opinions on it,  
20 and that there are reasons why EPA did it.

21 JUDGE VITTON: Okay, fine. Thank you.

22 DR. BAYARD: So let me finish?

23 MR. FURR: So in conducting...

24 JUDGE VITTON: Wait a minute.

25 I will give you an opportunity to explain your

1 answers, but we cannot take the time to wander through the  
2 entire SAB report here and pick up what everybody questioned  
3 on this particular area.

4 He's asking you a specific question, I think it's  
5 fairly specific. Please respond to it, but I'll give you  
6 time to explain your answer, but we cannot wander through  
7 the document and pick up everybody's comment on this  
8 particular subject.

9 MR. MYERS: Your Honor, I have to object.

10 His answer was extraordinarily responsive. There  
11 was an implication that EPA didn't consider something in  
12 violation of a standard method. First Dr...

13 MR. FURR: Your Honor, he's got three lawyers up  
14 here.

15 MR. MYERS: Yes, but first he tried to give the  
16 second half of what was really involved and he was cut off.  
17 Then he tried to give an explanation of what EPA did so it  
18 could be put in the proper scientific context.

19 Coming back to it at 5:00 o'clock tonight is not  
20 the same thing. There is a coherent answer that he was  
21 giving that fit. If he's really interested in the answer as  
22 opposed to making a record for a deposition in another case,  
23 then he'll let him finish the answer. That's what we're  
24 here for, finding some scientific truth. He's trying to  
25 give it to us.

1 JUDGE VITTON: Thank you, Mr. Myers.

2 I think he's completed his answer with respect to  
3 that specific question.

4 Let's go on to the next question so we can keep  
5 moving.

6 MR. FURR: In contract, Dr. Bayard, isn't it true  
7 that EPA included the baseline non-exposed category in  
8 conducting its trend analyses?

9 DR. BAYARD: EPA included the baseline trend or  
10 the baseline category in conducting its trend analysis. The  
11 reason they did is because we believe it's the suitable  
12 analysis. In fact, the Mantel-Haensel test describes that  
13 analysis.

14 Dr. Mantel was a consultant for the Tobacco  
15 Institute and commented on our report. Nowhere in his  
16 comments to our report did he say we shouldn't use the  
17 baseline study, the baseline... I'm sorry, the non-exposed  
18 category. And nowhere did he say that his test was an  
19 improper test to use.

20 MR. FURR: Your Honor, I'm coming to a problem  
21 here because Dr. Bayard phrased his opinion, or phrased his  
22 answer in what we, the EPA, considered to be proper. What  
23 I'm running into is, I again have documentary evidence that  
24 there were scientists who were asked to review the report  
25 because of the value placed on their opinions that disagree

1 whether that was a proper approach. This is the line of  
2 questioning that you cut me off before, and I'm just raising  
3 it to illustrate for you the problem that it's creating.

4 JUDGE VITTON: I understand what you're doing,  
5 Mr. Furr, but I told you we're not going to explore the  
6 internal decisionmaking with respect to the opinions on this  
7 report.

8 MR. FURR: Thank you.

9 Dr. Bayard, did EPA actually conduct any trend  
10 tests without the baseline non-exposed category?

11 DR. BAYARD: No.

12 MR. FURR: You do know that if such tests are  
13 conducted, that not a single study exhibits a statistically  
14 significant trend, don't you?

15 DR. BAYARD: I do not know that. We did not  
16 conduct it. But I would not conduct it because I don't  
17 think it's a proper test. I also would say that we inquire  
18 of our Science Advisory Board if they believed our  
19 methodology was correct, and they responded to us that they  
20 believed our methodology was correct. Given the comments to  
21 the Science Advisory Board, they had the opportunity to  
22 examine the issue twice, and they came back and they said  
23 no, they believed our methodology was correct.

24 MR. FURR: Dr. Bayard, you're not testifying, are  
25 you, that the Scientific Advisory Board approved of every

1 single technique, analysis, and point made in the EPA  
2 report, are you?

3 DR. BAYARD: I am not testifying to that, but I am  
4 testifying that that issue was already brought up twice by  
5 two very well respected people. Dr. Switzer is the chairman  
6 of the Department at Stanford, and they rejected him.

7 MR. FURR: So you respect the views expressed by  
8 Dr. Switzer, I take it, then?

9 DR. BAYARD: Certainly not on this issue.

10 MR. FURR: You are aware, Dr. Bayard, that it has  
11 been reported by Layard that if the baseline non-exposed  
12 group is left out of the analysis, that none of the trends  
13 are statistically significant?

14 DR. BAYARD: I'm not aware of that.

15 MR. FURR: You're simply not familiar with that  
16 report?

17 DR. BAYARD: I just haven't seen that. I'm sorry.

18 MR. FURR: You don't have any basis then for  
19 questioning the accuracy of Dr. Layard's findings, do you?

20 MR. MYERS: Your Honor, he hasn't seen it. How  
21 can he base one way or the other?

22 JUDGE VITTON: Mr. Myers, please sit down.

23 What's your question?

24 DR. BAYARD: I haven't seen it. How can I base it  
25 one way or the other?

1 (Laughter)

2 MR. FURR: Dr. Bayard, are you familiar with two  
3 epidemiologists named Malcolm McClure of the Harvard School  
4 of Public Health, and Sander Greenland of the UCLA School of  
5 Public Health?

6 DR. BAYARD: I've met Dr. Greenland. I haven't  
7 met Dr. McClure.

8 MR. FURR: You've met Dr. Greenland, but not Dr.  
9 McClure?

10 DR. BAYARD: That's correct.

11 MR. FURR: You're familiar with Dr. McClure by  
12 reputation, aren't you?

13 DR. BAYARD: Not really.

14 MR. FURR: Let me hand you a paper titled, "Tests  
15 for Trend and Dose Response, Misinterpretations and  
16 Alternatives" by Drs. McClure and Greenland, published in  
17 the American Journal of Epidemiology in 1992.

18 (Document handed to Dr. Bayard)

19 DR. BAYARD: Thank you.

20 (Pause)

21 MR. FURR: Have you ever seen that paper, Dr.  
22 Bayard?

23 DR. BAYARD: No, I haven't.

24 MR. FURR: Have you ever read it?

25 (Laughter)

1 MR. FURR: I'm sorry. Of course you haven't.

2 Actually, that might be a good question.

3 Have you ever heard of it?

4 DR. BAYARD: I'm not familiar with this paper. I  
5 have never heard of it until you mentioned it.

6 MR. FURR: Wasn't that paper pointed out to you by  
7 a member of EPA's Cincinnati office as one that you should  
8 read in reevaluating the approach taken by EPA with respect  
9 to the dose response trend?

10 MR. SHEEHAN: Your Honor, there he goes again.  
11 He's trying to get into the internal...

12 MR. FURR: No, I'm just asking him, wasn't it  
13 pointed out to him.

14 MR. SHEEHAN: This is an internal deliberation,  
15 Your Honor.

16 JUDGE VITTON: Mr. Furr, I'm going to disallow  
17 the question.

18 MR. FURR: Your Honor, I have a question. I'm not  
19 quite sure how to proceed.

20 I have a number of questions like that that I feel  
21 the record will be incomplete if I don't at least ask those  
22 questions.

23 Now you may get very frustrated with...

24 JUDGE VITTON: Are these public documents or  
25 what?

1 MR. FURR: These are public documents because they  
2 were part of a public record that resulted from the Rose  
3 subcommittee hearing that I referred to earlier.

4 JUDGE VITTON: Okay. You're going to ask more  
5 questions like that one you just asked?

6 MR. FURR: I have a lot of questions like that  
7 one.

8 MR. SHEEHAN: Your Honor, I would suggest he put  
9 the documents in the record.

10 JUDGE VITTON: We've received a lot of documents  
11 into this record that people have offered and I'm sure we'll  
12 be getting a lot more between now and Monday, but...

13 MS. SHERMAN: Your Honor, could I ask a clarifying  
14 question at this point rather than waiting for my turn? To  
15 help me understand the nature of this...

16 JUDGE VITTON: You mean of Mr. Furr or Dr.  
17 Bayard?

18 MS. SHERMAN: Actually, I believe of Dr. Bayard's  
19 lawyer.

20 JUDGE VITTON: What's your clarifying question?

21 MR. FURR: Does this count against my time?

22 JUDGE VITTON: No. I turned Mickey off.

23 (Laughter)

24 MS. SHERMAN: Mr. Sheehan, the documents that Mr.  
25 Furr is referring to, were they made part of the EPA docket

1 and record in terms of the EPA report, or were they merely  
2 turned over on request to Congressman Rose as part of his  
3 inquiry into some such thing?

4 MR. SHEEHAN: I think Ms. Neuwirth would be better  
5 able to respond.

6 MS. NEUWIRTH: The documents that Mr. Furr is  
7 referring to were documents that were either sent to  
8 Congressman Rose or were sent to Mr. Furr in response to a  
9 FOIA request. They were not part of the administrative  
10 record in the ETS report.

11 MS. SHERMAN: Thank you.

12 MR. FURR: Your Honor, this really does point  
13 to...

14 JUDGE VITTON: I'm sorry, I can't hear you, Mr.  
15 Furr.

16 MR. FURR: This really does put me on the horns of  
17 a dilemma now, because whether or not there in fact is an  
18 administrative record, what that record is comprised of,  
19 whether it was properly constructed, and when it was  
20 constructed is an issue in litigation which I have stayed  
21 clear of all day. I have not asked a single question  
22 unrelated to the opinions of Dr. Bayard expressed here in  
23 this hearing or related to the workplace smoking rule.

24 Now this latest point really introduces the  
25 litigation into this hearing. I can't let stand the

1 representation by EPA's counsel unchallenged with respect to  
2 the administrative record surrounding that document.

3 JUDGE VITTON: What do you want to say about it?

4 MR. FURR: What I really want is that statement on  
5 the record.

6 JUDGE VITTON: Okay.

7 But again, to go back to my original ruling, I do  
8 not believe that this is the proper forum for us to get into  
9 a deliberation, get involved on the deliberations that went  
10 within EPA internally before the report was issued.

11 MR. FURR: I've accepted Your Honor's ruling. I  
12 only want to submit that this was really a different  
13 attempt. I was, in effect, trying to impeach Dr. Bayard's  
14 statement that he had never had the document brought to his  
15 attention before.

16 JUDGE VITTON: The document being what?

17 MR. FURR: The report by Drs. McClure and  
18 Greenland.

19 JUDGE VITTON: All right. For the limited  
20 purpose of exploring whether he's seen the report, I'll  
21 permit you to ask that question.

22 MR. FURR: May I show him the document, Your  
23 Honor?

24 JUDGE VITTON: Yes.

25 (Pause)

1 MR. FURR: Dr. Bayard, I want to hand you an EPA  
2 Memorandum dated March 23, 1992 from Patricia A. Murphy,  
3 Epidemiologist, Meds, to Lynn Pabba, Acting Chief of Meds,  
4 and ask you to turn to page four.

5 JUDGE VITTONI: You're asking him just on the  
6 report, on the report from whoever that doctor was, I can't  
7 remember.

8 DR. BAYARD: Dr. McClure and Dr. Greenland.

9 JUDGE VITTONI: Yeah. Your question is has he  
10 ever see that before.

11 MR. FURR: That's right.

12 JUDGE VITTONI: Your answer was that you have not  
13 seen it, is that right?

14 DR. BAYARD: I have no memory of reading this  
15 report at all. In fact I don't even have any memory of ever  
16 having seen a reference to it.

17 MR. FURR: My question was whether the report had  
18 ever been brought to his attention before.

19 MS. SHERMAN: I think he just answered that  
20 question.

21 MR. SHEEHAN: He hasn't seen it, he hasn't heard  
22 of it, he doesn't have any memory of it.

23 JUDGE VITTONI: I thought his answer was I don't  
24 have a recollection.

25 Is that your answer?

1 DR. BAYARD: Yes. I know I haven't read this  
2 before. I don't remember anyone bringing it to my attention  
3 before.

4 JUDGE VITTONI: If you want to try to refresh his  
5 recollection, I'll permit you to do that. For the limited  
6 purpose of that.

7 (Document handed to Dr. Bayard)

8 DR. BAYARD: Thank you.

9 MR. FURR: Dr. Bayard, can you take a look at the  
10 cover page of that memorandum? You've seen that memo  
11 before, haven't you?

12 DR. BAYARD: Yes.

13 MR. FURR: Can you turn to page four please, the  
14 bottom of the page?

15 Let me back up.

16 That's a memorandum from an epidemiologist in the  
17 Cincinnati office that was generated as part of the  
18 Cincinnati office's review of a draft of the risk  
19 assessment, isn't it?

20 DR. BAYARD: Yes.

21 MR. FURR: I want to read to you, in an effort to  
22 refresh your memory, some language that appears at the  
23 bottom of that page. It's the sentence beginning with, "I  
24 feel."

25 DR. BAYARD: Yes.

1 MR. FURR: Doesn't that memorandum state, "I feel  
2 the importance of the trend test and its associated P-Value  
3 is overstated. Misclassification and measurement error can  
4 mask a dose response trend but it can also sometimes create  
5 one. In some cases, there is a significant P-Value that  
6 examination of the data shows that there is not a consistent  
7 upward trend in the odds ratios. See M. McClure and S.  
8 Greenland, 1992, "Tests For Trend and Dose Response,  
9 Misinterpretations and Alternatives," American Journal of  
10 Epidemiology, 135.

11 DR. BAYARD: That's correct.

12 MR. FURR: You've seen that before then, haven't  
13 you?

14 DR. BAYARD: I've seen it, but I don't remember  
15 ever having... It is gone from my memory.

16 MR. FURR: But it was brought to your attention?

17 DR. BAYARD: I remember seeing the memo, but I  
18 certainly didn't remember that part of it.

19 MR. SHEEHAN: For the record, Your Honor, this  
20 memo was written by Patricia Murphy to Lynn Pabba. It's not  
21 written by Dr. Bayard.

22 MR. FURR: He says he's seen it.

23 JUDGE VITTON: Thank you for the identification.

24 MR. SHEEHAN: And Your Honor, it does not attack  
25 the report that Mr. Furr is referring to here.

1 MR. FURR: Did you ever follow that suggestion and  
2 take a look at the paper?

3 MR. SHEEHAN: Your Honor, here we go again.

4 JUDGE VITTON: I think he's already answered he  
5 has no recollection of seeing this thing. We've got it on  
6 several times now.

7 Let's move on, Mr. Furr.

8 MR. FURR: Let's look at the paper now, Dr.  
9 Bayard. Do you still have it up there?

10 Can you take a look at the abstract? Tell me if I  
11 read this correctly.

12 "Tests for overall trend such as the Mantel  
13 extension tests are not tests for monotonic dose response.  
14 A survey of epidemiologic articles shows widespread  
15 misinterpretation of the Mantel extension test, and  
16 overstatement of evidence for monotonic dose response when  
17 there are few exposed subjects. To properly evaluate the  
18 hypothesis that risk continues to increase with further  
19 increases in exposure, one must examine several statistics  
20 and estimates. Given sufficient data, parametric or  
21 polynomial regression analyses can provide more detailed  
22 dose response."

23 Did I read that correctly?

24 DR. BAYARD: Except for one word. Where you said,  
25 "Given sufficient data," you said "parametric" instead of

1 "non-parametric."

2 MR. FURR: Thank you.

3 That abstract refers to the Mantel extension test  
4 that EPA utilized in performing its dose response trend  
5 analysis, doesn't it?

6 DR. BAYARD: Actually we didn't need the extension  
7 of it, we just needed the trend test. The extension test  
8 refers to combining trend tests by strata, so we didn't use  
9 the extension test, we just used the basic Mantel-Haensel  
10 trend test.

11 MR. FURR: You agree with Drs. McClure and  
12 Greenland that a trend test determines only that there is a  
13 linear component in the risk observed for various exposure  
14 levels, don't you?

15 DR. BAYARD: I would like to agree with you, but I  
16 think I'd like you to ask the question again.

17 MR. FURR: You agree with Drs. McClure and  
18 Greenland that a trend test determines only that there is a  
19 linear component in the risk observed for various exposures  
20 levels.

21 DR. BAYARD: I can't agree with the statement, but  
22 I'd like to try to help out. Because the trend tests will  
23 determine whether or not there is a linear component, it  
24 doesn't determine that there is one. It determines whether  
25 or not there is a linear component.

1 MR. FURR: Let me ask the question differently.  
2 Maybe that will help.

3 Wouldn't you agree that a test for trend is not a  
4 test for dose response in the sense of demonstrating a  
5 continuously increasing or monotonic increasing risk with  
6 increasing exposure?

7 DR. BAYARD: Within the bounds of statistical  
8 variation, I'd say yes, it does.

9 MR. FURR: Dr. Bayard, I want to try to look at an  
10 example with you of how a test for trend can be misconstrued  
11 as a dose response. Could you look at your EPA report at  
12 page 542?

13 (Pause)

14 DR. BAYARD: Yes, I introduced these as slides  
15 this morning.

16 MR. FURR: That's Table 511, titled "Exposure  
17 Response Trends for Females" correct?

18 DR. BAYARD: That's correct.

19 MR. FURR: I'd ask you to look on page 542 at the  
20 information in that table for the Lamb-T study on cigarettes  
21 per day.

22 DR. BAYARD: Yes.

23 MR. FURR: That table shows that the exposure  
24 categories were zero, one to ten, 11 to 20, and greater than  
25 21 cigarettes per day, doesn't it?

1 DR. BAYARD: That's correct.

2 MR. FURR: The relative risk reported for those  
3 categories are one, 2.18, 1.85, and 2.07, correct?

4 DR. BAYARD: That's correct.

5 MR. FURR: EPA calculated a P-for trend of 0.01.

6 DR. BAYARD: That's correct.

7 MR. FURR: In other words, this Lamb-T study is  
8 one of those studies that EPA would count as showing a  
9 statistically significant trend, isn't it, Dr. Bayard?

10 DR. BAYARD: That's correct.

11 MR. FURR: Do you have an estimate of what the  
12 P-Value would be in that study if one were to exclude the  
13 unexposed group?

14 DR. BAYARD: It's probably one, probably .5 on  
15 a...

16 MR. FURR: Good guess. Let me represent to you it  
17 would be 0.45. That wouldn't surprise you at all, would it?

18 DR. BAYARD: No.

19 MR. FURR: If that type of analysis had been done,  
20 that study would not be counted as a statistically  
21 significant study, would it?

22 DR. BAYARD: Yes, it would.

23 MR. FURR: With a P of .45?

24 DR. BAYARD: It wouldn't have a statistically  
25 significant trend test.

1 MR. FURR: That's what I meant to ask.

2 DR. BAYARD: It has three dose groups, three  
3 exposure levels, all of which are statistically significant  
4 independently.

5 MR. FURR: That study would not have been included  
6 in EPA's tally of studies showing a statistically  
7 significant trend...

8 DR. BAYARD: I'm sorry, you interrupted me,  
9 because the question was, would that have been counted as a  
10 significant study.

11 MR. FURR: I'll withdraw that. I misspoke.  
12 That's not my question.

13 DR. BAYARD: But the answer to that question..

14 MR. FURR: Your Honor, this is easy. That wasn't  
15 my question. I misspoke.

16 JUDGE VITTON: He did change his question. You  
17 corrected it...

18 DR. BAYARD: He only changed his questions when I  
19 started answering it.

20 (Laughter)

21 JUDGE VITTON: He realized...

22 DR. BAYARD: At what point can you say I don't  
23 like that answer, I'm going to take my question back. You  
24 know.

25 (Laughter)

1 JUDGE VITTON: I'm giving you an opportunity, but  
2 go ahead. He did change his question.

3 MR. FURR: Dr. Bayard, that's one of the studies  
4 that EPA included in its list of studies showing a  
5 statistically significant trend, isn't it?

6 DR. BAYARD: Yes, it is.

7 MR. FURR: You don't contend that that study shows  
8 a dose response do you?

9 (Pause)

10 DR. BAYARD: Yes, I do. I don't think it's  
11 particularly linear. It may saturate really quickly, but it  
12 varies. You don't have perfect data here, Mr. Furr. The  
13 best you can try to do is try to get different categories,  
14 and you try to get some differential between the categories.

15 That may be an aberration, that is an aberration,  
16 number one, because if you look at most of the other data,  
17 most of...

18 MR. FURR: Your Honor, my question was whether  
19 this study shows a dose response.

20 JUDGE VITTON: All right.

21 Let's take five minutes. We've been going for an  
22 hour and a half.

23 (Whereupon, a brief recess was taken.)

24 JUDGE VITTON: We're back on the record.

25 Dr. Bayard, we resume with Mr. Furr.

1 MR. FURR: Thank you, Your Honor.

2 Dr. Bayard, I want to go back to right where we  
3 left off.

4 My question is, you don't contend that the Lamb-T  
5 data show a dose response, do you?

6 (Pause)

7 DR. BAYARD: I would go back and look at the data,  
8 but I think I do. I'd like to explain just quickly why I  
9 feel I do.

10 Because it's so difficult when you get down to  
11 small sample sizes to try to distinguish between exposure  
12 categories. We never expected to see dose response to begin  
13 with. The fact that we don't see a wonderful monotonic dose  
14 response in the Lamb-T study shouldn't be evidence that  
15 there is no effect or dose response. Because if you look at  
16 the relative risks, they're quite high. They're higher than  
17 most of the other studies, in fact. So if you want to take  
18 off 183 plus 84 people, you will succeed in lowering the  
19 power. You will succeed in throwing away all those  
20 unexposed data. To say that there is no dose response  
21 merely because you can manipulate the data and get a  
22 straight line by excluding the unexposed group is not  
23 substantive to me.

24 MR. FURR: Dr. Bayard, as the EPA reported the  
25 Lamb-T data in that chart, the data show that the two

1 highest levels of exposure pose less risk than the lowest  
2 level of exposure, don't they?

3 DR. BAYARD: I'd say that's not statistically  
4 significant at all, and it's not a meaningful statement...

5 MR. FURR: Answer my question first, and then you  
6 can explain it.

7 Don't they show a lower level of risk for the two  
8 highest levels of exposure than the lowest level?

9 DR. BAYARD: No.

10 MR. FURR: No?

11 DR. BAYARD: No.

12 MR. FURR: What is the risk for the category of  
13 one to ten cigarettes per day?

14 DR. BAYARD: 2.18 of 2.46.

15 MR. FURR: What is the risk for the category of 11  
16 to 20 cigarettes per day?

17 DR. BAYARD: I'm sorry. I was looking at the last  
18 one. I'll answer the question.

19 1.85 or 2.29.

20 MR. FURR: And doesn't that show that the two  
21 highest levels of exposure show a lower risk than the lowest  
22 level of exposure?

23 DR. BAYARD: In one of the groups it does.

24 MR. FURR: And you believe that that data supports  
25 the contention that the Lamb-T study shows a dose response?

1 DR. BAYARD: Yes. And I'll tell you why.

2 MR. FURR: You've already told us why. I just  
3 wanted to make sure that...

4 DR. BAYARD: Because I believe that...

5 JUDGE VITTON: Let him finish, Mr. Furr, quickly.

6 DR. BAYARD: I believe that the risks have to be  
7 compared with a suitable control. If you remove the control  
8 group then you're removing the matching. I mean if you  
9 remove the unexposed group then you're removing the  
10 matching. The comparison doesn't make sense.

11 I think if you take, under certain circumstances  
12 when you have... I'm sorry. That's enough. Go ahead.

13 MR. FURR: New topic.

14 EPA reported that ten, and you emphasized this  
15 morning, that ten of the 14 studies with sufficient exposure  
16 response data show statistically significant trends for one  
17 or more measures, right?

18 DR. BAYARD: Yes.

19 MR. FURR: In other words, some of the studies  
20 that EPA reviewed contain more than one analysis of trend  
21 information.

22 DR. BAYARD: That's correct. That information is  
23 on Table 512.

24 MR. FURR: Let's do this a little differently. Do  
25 you have a copy of the Fontham 1991 study with you?

1 DR. BAYARD: Yes, I do.

2 MR. FURR: Could you turn to Table 6 on page 41?

3 (Pause)

4 MR. FURR: I've got an extra if you need it.

5 DR. BAYARD: I'm sorry, I do.

6 (Document handed to Dr. Bayard.)

7 MR. FURR: Page 41, Table 6, please.

8 (Pause)

9 MR. FURR: In EPA's risk assessment, EPA reported  
10 that Fonham, 1991, showed a statistically significant  
11 trend, didn't it?

12 DR. BAYARD: Yes. I think so.

13 MR. FURR: But the value included in the EPA's  
14 report was the trend, the P-for trend for adenocarcinoma  
15 only, wasn't it?

16 (Pause)

17 DR. BAYARD: I don't think so. I'm going to have  
18 to check that.

19 Do you want to help me out on this?

20 MR. FURR: Is it Table 511 that contains the trend  
21 information in the EPA report?

22 DR. BAYARD: That's correct.

23 MR. FURR: Will you look at Fonham there? What  
24 P-for trend does EPA report?

25 DR. BAYARD: .04, .01, and .02 and .07. I'll try

1 to check and see which ones we did.

2 (Pause)

3 DR. BAYARD: It should have been all histologies,  
4 number six, and it should have been pack years, number six,  
5 so it should have been .04 which would have been the middle  
6 of the page, 1.96, 1.13, 1.25, 1.33.

7 MR. FURR: Fontham et al had more trend analyses  
8 than reported in the EPA report, didn't it? In fact Table 6  
9 shows us eight trend analyses, doesn't it?

10 DR. BAYARD: That's correct. However, the  
11 decision on which analyses we would do was made long before  
12 we even heard of the Fontham study. The decision was to try  
13 to determine the effects from all lung cancers and from  
14 spousal exposure, and to do the adjusted analyses when that  
15 adjusted analysis was presented.

16 So all these eight tests that you see in Table 6  
17 weren't considered because the preferable test had already  
18 been done. The reason we did that was so that we could  
19 compare like statistics and like studies.

20 MR. FURR: Dr. Bayard, where in the EPA report  
21 does EPA acknowledge the role of multiple comparisons in  
22 producing the statistically significant P-for trend?

23 DR. BAYARD: I don't think we do.

24 MR. FURR: I don't think you do either.

25 That is an important issue though, isn't it?

1 DR. BAYARD: I don't think it's an important issue  
2 with this. I think in general it's an important issue, and  
3 I think Dr. Roth brought that up also. The theory of  
4 multiple comparisons being that if you take 20 tests, even  
5 if there's no significant difference, you're going to find  
6 one by chance at the five percent level. So I thought about  
7 that, and so here on Table 512... I'm sorry. We actually  
8 have three comparisons. However, they're not independent.

9 The theory of multiple comparison deals with  
10 independent tests. In lots of these papers, as you see,  
11 there are no data for some of these tests. We only had,  
12 for example in Correa, he only presented one digit, and  
13 that's one test, and that's all we could take from him. So  
14 in that sense we didn't have any multiple comparisons.

15 With the Fontham study we actually did have  
16 multiple comparisons because as pointed out on Table 511,  
17 there were four tests. One dealt with all lung cancers, one  
18 dealt with only adenocarcinomas, and they were for spousal  
19 smoking. So there were four trend tests. One for pack  
20 years and one for just years. All of them were at least  
21 very, very close to statistical significance if not  
22 statistically significant, so they weren't very independent.

23 When you have years, whether you do a comparison  
24 by years or whether you do a comparison by pack years or  
25 whether you do a comparison by cigarettes per day, they're

1 not independent comparisons because the three measures are  
2 related.

3 MR. FURR: Dr. Bayard, the consequence of multiple  
4 comparisons is that the outcome appears to be more  
5 statistically significant than it should be, isn't that  
6 correct?

7 DR. BAYARD: That's a general statement. You'd  
8 have to be more specific.

9 MR. FURR: You agree with that as a general  
10 principle?

11 DR. BAYARD: No. When you've got multiple  
12 comparisons you may have some significant and some not. You  
13 just have to look at your data.

14 MR. FURR: Can you turn to page 551 of the EPA  
15 report, please?

16 (Pause)

17 MR. FURR: In that section, EPA is evaluating the  
18 potential role of confounding in the epidemiologic studies.

19 Can you look at the last sentence before Section  
20 542 on page 551?

21 (Pause)

22 DR. BAYARD: Yes.

23 MR. FURR: Doesn't that state that "Multiple tests  
24 on the same data increase the chance of a false positive,  
25 i.e., outcomes appear to be more significant than warranted

1 due to the multiple comparisons being made on the same  
2 data."

3 DR. BAYARD: That's correct.

4 MR. FURR: Are you familiar with the Bonferoni  
5 adjustment technique?

6 DR. BAYARD: Yes.

7 MR. FURR: That's a technique for adjusting the  
8 influence of multiple comparisons and statistical  
9 significance testing, isn't it?

10 DR. BAYARD: That's correct.

11 MR. FURR: Did EPA perform a Bonferoni adjustment  
12 on its analysis of trend in the spousal smoking studies?

13 DR. BAYARD: No, it did not. They did not because  
14 of the reason I just told you. The Bonferoni test is  
15 designed for independent comparisons. These are not  
16 independent comparisons.

17 MR. FURR: Isn't it true that if a Bonferoni  
18 adjustment had been performed, that none of the studies  
19 would have shown a statistically significant trend?

20 DR. BAYARD: That's true. If we threw the data  
21 away there would also be no significant trend. You don't  
22 perform a Bonferoni test when it's not called for.

23 MR. FURR: Dr. Bayard, epidemiologic textbooks do  
24 not use the term dose response interchangeably with the term  
25 trend, do they?

1 DR. BAYARD: I imagine some do and some don't.

2 MR. FURR: You wouldn't agree with that, with the  
3 use of those terms interchangeably, would you?

4 DR. BAYARD: Well, trend, they can mean the same  
5 things, they can mean different things. You can talk about  
6 trend in the weather. Cyclical trends.

7 MR. FURR: If we use the phrase "dose response" to  
8 refer to a continuously increasing monotonic dose response,  
9 using the term in that context, wouldn't you agree that if  
10 OSHA wishes to test the statistical significance of the dose  
11 response information in the epidemiologic studies, that it  
12 cannot rely on EPA's trend test?

13 DR. BAYARD: You expect me to agree to that?

14 MR. FURR: Yes.

15 DR. BAYARD: No.

16 MR. FURR: Why?

17 DR. BAYARD: Because I think we did the proper  
18 test.

19 MR. FURR: That's not my question. My question  
20 is, is EPA's trend test equivalent to a test for a  
21 monotonically increasing dose response test?

22 Isn't the answer obvious?

23 JUDGE VITTON: Let him think about it.

24 (Pause)

25 DR. BAYARD: It's one of the ways of doing a dose

1 response test, but there are different ways you can test for  
2 monotonicity. This is only one of them.

3 MR. FURR: Did the Lamb-T data we just looked at  
4 show a monotonic dose response?

5 DR. BAYARD: No.

6 MR. FURR: But it was identified by EPA as a study  
7 with a statistically significant P-for trend.

8 DR. BAYARD: That's correct.

9 MR. FURR: I'll talk to you about a different  
10 topic.

11 I want to ask you some questions about the Fontham  
12 study.

13 (Pause)

14 MR. FURR: An aspect of that study that you have  
15 discussed in your comments is the fact that Fontham used a  
16 second control group of patients with colon cancer during  
17 the first three years of the study. Correct?

18 DR. BAYARD: That's correct.

19 MR. FURR: You suggest that the results of that  
20 analysis indicate that recall bias did not play a role in  
21 producing the risk observed in the Fontham study.

22 DR. BAYARD: That's correct.

23 MR. FURR: Dr. Bayard, isn't it true that the risk  
24 estimates generated using the colon cancer controls were  
25 uniformly lower than those generated using the population

1 controls?

2 You've got the study, you can turn to Table 5,  
3 pages 40.

4 DR. BAYARD: I'm looking for it right now.

5 (Pause)

6 DR. BAYARD: Okay.

7 JUDGE VITTON: What is the question, Mr. Furr?

8 MR. FURR: The question was, doesn't that table  
9 show that the risk estimates generated using the colon  
10 cancer controls were uniformly lower than the risk estimates  
11 generated using the population controls?

12 DR. BAYARD: The differences are indistinguishable  
13 to my eye. I think you could say .01, 1.28 versus 1.29 is  
14 meaningful? 1.17 versus 1.20 is meaningful? 1.14 versus  
15 1.26 is meaningful? 1.17 versus 1.21 is meaningful? They're  
16 so small as to be indistinguishable, so I wouldn't say that  
17 there's difference enough to matter.

18 MR. FURR: That's not my question.

19 DR. BAYARD: I'm sorry.

20 MR. FURR: My question is simply, are they lower  
21 in every single case reported in Table 5?

22 DR. BAYARD: My answer is simply yes, but it's  
23 meaningless.

24 MR. FURR: No. Fontham did not promulgate the use  
25 of the colon cancer controls all the way through the full

1 five years of their study and into the final report, did  
2 they?

3 DR. BAYARD: That's correct.

4 MR. FURR: So we really don't have any way of  
5 knowing whether this pattern of the colon cancer controls  
6 producing a uniformly lower risk estimate than the  
7 population controls being lower, held up, do we? We simply  
8 can't tell from the Fontham study.

9 DR. BAYARD: I'm just not going to agree with your  
10 premise that these things are uniformly lower. To me those  
11 differences are meaningless. Don't forget I'm a  
12 statistician. To me a number one, one little, doesn't mean  
13 a whole bunch of difference.

14 MR. FURR: Let's talk about the numbers. Show me  
15 an instance in which they're not lower.

16 (Pause)

17 DR. BAYARD: I'll be right there.

18 MR. FURR: I'm asking you about Table 5.

19 (Pause)

20 DR. BAYARD: Look at the adjusted odds ratios on  
21 Table 7.

22 MR. FURR: Dr. Bayard, I asked you a question  
23 about Table 5.

24 DR. BAYARD: You asked me a question about Table  
25 5, but what you said to me was show me an instance where

1 it's not lower, and I'm showing you several instances where  
2 it's not lower in Table 7.

3 MR. FURR: Dr. Bayard, my questions are in the  
4 context of Table 5. If you want to submit some comments  
5 about Table 7 at some other point, I'm sure you're free to  
6 do so.

7 DR. BAYARD: My answer is going to be that with  
8 respect to Table 5, you've pointed out some meaningless  
9 differences on adjusted odds ratio. If you then go to Table  
10 7, you're going to find out that your answers are going to  
11 be slightly different.

12 MR. FURR: Your Honor...

13 DR. BAYARD: There's just not a whole bunch of  
14 difference in these two control groups. And because of  
15 that, she did the statistical tests and she said there's not  
16 a whole bunch of difference in these control groups, so for  
17 the rest of the analysis I'm going to combine the control  
18 groups.

19 MR. FURR: Dr. Bayard, on Table 5, show me where  
20 the colon cancer control groups don't generate a lower risk  
21 than the population controls.

22 DR. BAYARD: I think I've answered that question.

23 JUDGE VITTON: Mr. Furr, the numbers are the  
24 numbers. Getting him to repeat them, I'm not sure that adds  
25 much to this record.

1 MR. FURR: He said he couldn't distinguish them by  
2 his eye. I'm just wondering which numbers he can't tell the  
3 difference between.

4 DR. BAYARD: Mr. Furr, it's just a distinction  
5 without a difference here.

6 JUDGE VITTON: Okay, let's not...

7 MR. FURR: Dr. Bayard, EPA did not make any  
8 adjustment in its analysis of the epidemiologic studies to  
9 account for any recall bias, did it?

10 DR. BAYARD: That's correct. I'm just trying to  
11 think of whether or not we used the adjusted odds ratios  
12 that the authors might have used to see whether any of the  
13 authors themselves might have adjusted for any recall bias.  
14 I think Fontham would have been the only one to do an  
15 analysis of recall bias.

16 MR. FURR: And EPA made no effort to make any  
17 adjustment for recall bias.

18 DR. BAYARD: That's correct.

19 MR. FURR: I want to talk to you about another  
20 topic with respect to the Fontham study. You have mentioned  
21 several times today the importance of the concept of the  
22 generalizability of one study for purposes of its use in  
23 risk assessment. I may use the term representativeness, but  
24 I mean the same thing there.

25 One of the features that you have emphasized with

1 respect to the Fontham study is the generalizability or  
2 representativeness of the study, isn't that correct?

3 DR. BAYARD: I think so.

4 MR. FURR: Isn't it a fact that in the Fontham  
5 study the data are primarily from two larger urban areas in  
6 California, being Los Angeles and San Francisco?

7 DR. BAYARD: I think yes, and I think the second,  
8 the 1994 study was more weighted with those two locations,  
9 if I'm not mistaken.

10 MR. FURR: That's because those are the only two  
11 areas in which Fontham continued collecting cases isn't it?

12 DR. BAYARD: I didn't know that.

13 MR. FURR: In fact more than 80 percent of the  
14 cases and 85 percent of the controls are from Los Angeles or  
15 California, aren't they?

16 DR. BAYARD: In the '94 study.

17 MR. FURR: That's correct. You agree with that,  
18 don't you?

19 (Pause)

20 DR. BAYARD: Yes.

21 MR. FURR: The other cases and controls in the  
22 study are from three other urban areas, that is Houston, New  
23 Orleans, and Atlanta?

24 DR. BAYARD: That's correct.

25 MR. FURR: There's a statement in your written

1 comments that I'd like for you to explain. On page six you  
2 state that the Fontham study is representative at least of  
3 the southern part of the United States. Can you explain how  
4 a study that consists of 80 percent urban Californians is  
5 representative of the southern part of the United States?

6 DR. BAYARD: You know when I was writing that, I'm  
7 thinking, I'm from New England, and...

8 (Laughter)

9 MR. FURR: It all blends together if you move out.

10 DR. BAYARD: Anything south of New England is  
11 south. That's a real parochial northerner for you.

12 JUDGE VITTON: To make sure we understand this...

13 (Laughter)

14 MR. FURR: I don't think there is any  
15 understanding this.

16 JUDGE VITTON: New York and New Jersey, in your  
17 opinion, are part of the South?

18 DR. BAYARD: New York is not part of this country,  
19 Your Honor.

20 (Laughter)

21 JUDGE VITTON: We won't get into that.

22 DR. BAYARD: That was a New Yorker speaking.

23 MR. FURR: Dr. Bayard, when you look at Table 4 on  
24 page 39 in the Fontham '91 study, according to Table 4, only  
25 7.90 percent of the cases, six percent of the colon cancer

1 controls, and three percent of the population controls had a  
2 usual adult residence in a rural area, correct?

3 DR. BAYARD: I'm sorry, Mr. Furr. Please direct  
4 me which side of the table. Usual childhood resident?

5 MR. FURR: No, usual adult. Bottom row.

6 (Pause)

7 DR. BAYARD: Yes, sir.

8 MR. FURR: Isn't it true that about 24.8 percent  
9 of the U.S. population lives in a rural area?

10 DR. BAYARD: I don't know. But I don't want to  
11 argue with you on it. If you say it's okay, yeah.

12 MR. FURR: You don't challenge that.

13 DR. BAYARD: Absolutely not.

14 MR. FURR: Take another look at the table, if you  
15 will. Doesn't that table show that between 41 and 42  
16 percent of the study subjects were minorities?

17 (Pause)

18 DR. BAYARD: Aren't women all minorities? I'm  
19 just kidding.

20 (Pause)

21 DR. BAYARD: Of the cases or the controls?

22 MR. FURR: I think I said all subjects.

23 DR. BAYARD: Oh. I'd have to add them up.

24 MR. FURR: You don't disagree that...

25 DR. BAYARD: I'm sorry. Just subtract them from

1 the 63 percent and 68 percent and 64 percent, is that right?  
2 You're talking about ethnic group, right? You're saying  
3 whatever isn't white is considered a minority?

4 MR. FURR: I think that's correct.

5 DR. BAYARD: So you're saying somewhere between  
6 37, 32, and 35.

7 MR. FURR: Isn't it true that about 19.7 percent  
8 of the U.S. population are minorities?

9 DR. BAYARD: I didn't know. I thought it was a  
10 little more than that. But I guess if you have the figures  
11 there that's okay.

12 MR. FURR: Doesn't that table also show that about  
13 19 percent of the cases in the study were Asians?

14 DR. BAYARD: Uh hmm.

15 MR. FURR: Isn't it true that...

16 JUDGE VITTON: Yes, or no? Yes? Is that a yes?

17 DR. BAYARD: I was trying to agree...

18 JUDGE VITTON: You said uh hmm...

19 DR. BAYARD: I was trying to agree with him, and  
20 then all of a sudden I said maybe I'd better look at this.

21 Around 19 percent are Asians?

22 MR. FURR: Right.

23 DR. BAYARD: It looks like 16 and 10 for the colon  
24 cancer. For the lung cancer control, it's 16; for the colon  
25 cancer controls it's 10; for the population controls it's

1 14.5.

2 MR. FURR: We're looking at '91. I was really  
3 asking about '94, but that's okay.

4 Isn't it true that only about three percent of the  
5 U.S. population is Asian?

6 DR. BAYARD: I don't know.

7 MR. FURR: You don't contend that the study  
8 subjects in the Fontham et al study are representative of  
9 the U.S. population in any statistically valid sense, do  
10 you?

11 DR. BAYARD: I think, I can either say, I can say  
12 no but I don't think that's the relevant factor here. The  
13 relevant factor here is whether or not they're controls  
14 for...

15 MR. FURR: He can tell us the relevant factor, but  
16 is the answer no?

17 JUDGE VITTON: I'm not sure. Do you agree or not  
18 agree with what he just said?

19 DR. BAYARD: That cases are representative of the  
20 U.S. population? Based on the figures he told me, they're  
21 probably undersubscribed of whites.

22 JUDGE VITTON: On what? I'm sorry I didn't hear  
23 you.

24 DR. BAYARD: Undersubscribed of whites. There are  
25 probably a lower percentage of whites that are in the

1 general population.

2 JUDGE VITTON: In the Fontham study.

3 DR. BAYARD: In the '91 that I'm reading now

4 MR. FURR: I understand.

5 JUDGE VITTON: Okay.

6 Is there something else you wanted to say about  
7 that or what?

8 DR. BAYARD: I wanted to say that, just that as  
9 long as this is controlled for race, which Fontham did. She  
10 adjusted for race where she couldn't match them in the  
11 analysis, then I think the analysis is still a consistent,  
12 legitimate analysis and it doesn't buy us the odds ratio.

13 MR. FURR: Fontham did not match for race though,  
14 did she?

15 DR. BAYARD: I know she did in the '94 study.  
16 Whether she did in the '91 study, I don't know.

17 DR. DAVIS: Your Honor, I hate to interrupt, I  
18 apologize, but Mr. Furr has now exceeded three hours by my  
19 clock. I'd appreciate if we'd have the opportunity to ask  
20 questions of Dr. Bayard, and I myself have a flight to catch  
21 in a little while.

22 JUDGE VITTON: Well, Dr. Davis, by my clock he  
23 has not exceeded the three hours, but he's coming up within  
24 about one minute.

25 MR. FURR: Your Honor, I'm not even halfway done.

1 JUDGE VITTON: I understand that, Mr. Furr, but  
2 we are coming up on three hours. I would like to wrap it up  
3 at this point right now for you, and I'd like to give Dr.  
4 Davis a chance to get his questions in, then we will come  
5 back to the other people who have asked for time to ask  
6 questions also.

7 MR. FURR: If some of the other members will cede  
8 their time to me, does that mean I will get a further  
9 opportunity?

10 JUDGE VITTON: I'd like to give Dr. Davis time,  
11 and then we'll come back to what the other people want and  
12 we'll see where we are. He tells me he's got a plane to  
13 catch. I'd like to try to accommodate him if we can.

14 MR. FURR: Only so it's on the record, Your Honor,  
15 I just want to object to being cut off now. Given the  
16 importance of Dr. Bayard's testimony and the fact that we  
17 have been directed to put our questions to him by OSHA and  
18 its experts when they were unable to answer our questions, I  
19 don't think we're being given an opportunity required to  
20 fully probe Dr. Bayard's opinions, and I'd like that  
21 objection on the record.

22 JUDGE VITTON: We are not finished questioning  
23 Dr. Bayard yet today so time is not done yet.

24 MR. FURR: Thank you.

25 DR. DAVIS: Thank you, Your Honor. I appreciate

1 it.

2 Dr. Bayard, I would first like to ask you some  
3 questions that relate to the testimony that you've given  
4 here so far today and some of the answers that you've given  
5 to questions posed to you by Mr. Furr, and then I'd like to  
6 come back and ask some more general questions.

7 I wanted to just pick up on a comment that you  
8 made at the very beginning of your testimony. I think you  
9 were asked about ETS exposure in restaurants or other  
10 locations and you made a comment about how you'd be  
11 particularly concerned about the waiters and waitresses,  
12 those who were in restaurants for a prolonged period of time  
13 because that's their workplace, but that you might not worry  
14 so much about somebody who was passing through a restaurant  
15 for 20 minutes or eating there for a shorter period of time.

16 I just wanted to ask you, isn't it true that  
17 generally speaking cancer risk would depend on total  
18 cumulative exposure to the carcinogen in question? So that  
19 for example, if somebody worked in a smoky environment for  
20 five or six hours a day, but then had an hour of exposure to  
21 ETS in a restaurant and another two hours of exposure to ETS  
22 in the home and another hour of ETS exposure in some other  
23 location, that you sort of have to consider all of those  
24 exposures in determining cancer risk. Would you agree with  
25 that?

1 DR. BAYARD: I would agree with it, but in terms  
2 of studying it, it makes it a little bit more difficult.  
3 The Fontham '94 study was particularly strong in studying  
4 that, I thought, because she actually got estimates of total  
5 exposure and found that the highest levels, the highest  
6 indices of exposure actually averaged about an 80 percent  
7 increase with very strong dose response, and most  
8 significant dose response trends.

9 So the statement that you make, the total  
10 cumulative exposure, adds to the risk, the risk is additive  
11 over exposure, it seems to be well supported by her data.

12 DR. DAVIS: My point is that wouldn't it be  
13 inappropriate to look at any specific isolated risk such as  
14 an hour in a restaurant and say that that's not important  
15 because we don't have studies to show that that's not  
16 important, because actually you have to consider from a  
17 public policy standpoint, you have to consider that exposure  
18 added to other exposures.

19 DR. BAYARD: I don't do public policy, so it's  
20 hard for me to do. When I see risks in a restaurant for an  
21 hour, what they mean to me is dirty control groups.

22 DR. DAVIS: Let me rephrase it.

23 If you identify a Group A carcinogen, wouldn't it  
24 be best to try and reduce exposure to the absolute minimum  
25 or eliminate exposure all together if that's feasible?

1 DR. BAYARD: In general, yes, although I think  
2 arsenic has some elements of being an essential element,  
3 doesn't it? But I think in general, that's true.

4 DR. DAVIS: If you had your choice between being  
5 exposed to tobacco smoke in a restaurant or not being  
6 exposed, what would be your preference?

7 DR. BAYARD: When I go to lunch or dinner with  
8 Chris Coggins we sit in a no-smoking section.

9 DR. DAVIS: Thank you.

10 (Laughter)

11 Let me move on to the issue of threshold.

12 JUDGE VITTON: I don't know how relevant that is  
13 to anything, to tell you the truth.

14 (Laughter)

15 DR. DAVIS: You mentioned that you believe that  
16 there may in fact be a threshold below which exposure may  
17 not be harmful, is that correct?

18 DR. BAYARD: I think that's true for almost  
19 anything. I...

20 DR. DAVIS: But you are not aware...

21 DR. BAYARD: Can I please finish?

22 DR. DAVIS: Go ahead.

23 DR. BAYARD: There comes a problem as to  
24 sensitivity. Certain people are going to be highly  
25 sensitive and you can never tell which it does the damage.

1 As a statistician you look at probabilities and you say gee,  
2 I'm 20 minutes here, there isn't going to kill me. You're  
3 probably right.

4 DR. DAVIS: You're not aware that anybody has  
5 defined what the threshold may be for ETS and lung cancer  
6 risk, is that correct?

7 DR. BAYARD: No. I...

8 DR. DAVIS: Is there a consensus...

9 DR. BAYARD: I'm sorry. Let me finish.

10 DR. DAVIS: Go ahead.

11 DR. BAYARD: We didn't particularly look for  
12 thresholds for environmental tobacco smoke because we  
13 examined the data at typical environmental levels so there's  
14 no real desire to extrapolate downward. As far as we were  
15 concerned, pardon the expression, that was the beauty of the  
16 data. We didn't have to use low dose extrapolation models.

17 DR. DAVIS: Are you aware of any consensus in the  
18 scientific or medical community that there is a threshold  
19 below which exposure to ETS is safe?

20 DR. BAYARD: No.

21 DR. DAVIS: Are you aware of any specific levels  
22 of ETS no matter how you define that, whether it's nicotine  
23 levels or RSP or anything, are you aware that there's nay  
24 specific level of ETS that any scientific or medical  
25 organization has identified as a threshold below which

1 exposure is safe?

2 DR. BAYARD: I'm going to ask you the same  
3 question that I asked Mr. Furr. When you say safe, are you  
4 defining a level of risk? Are you defining a deminimus  
5 risk? Are you saying no risk?

6 DR. DAVIS: For the sake of argument, let's say  
7 deminimus risk.

8 DR. BAYARD: I think that's calculable. I think  
9 one could make an estimate. Mr. Repace has certainly  
10 developed a model which provides estimates of, dose response  
11 estimates of deminimus risk.

12 DR. DAVIS: Re there any organizations that have,  
13 or agencies that have come forth with thresholds to your  
14 knowledge, based on reputable science that identify a  
15 threshold?

16 DR. BAYARD: For environmental tobacco smoke?

17 DR. DAVIS: Yes.

18 DR. BAYARD: No, but I'd also add that there  
19 aren't... There aren't may agencies that would be doing it  
20 anyway. So the answer is no, I'm not aware of any, and no I  
21 suspect that there aren't any. Unless you can probably...  
22 Unless you would make the jump of faith to policy, that...  
23 I'm sorry. It's beyond where I have any...

24 DR. DAVIS: Let me put it this way. If there isn't  
25 any threshold out there that the scientific community has

1 agreed is appropriate, would it be wise public policy to try  
2 and eliminate exposure or to reduce it to the absolute  
3 minimum?

4 DR. BAYARD: Again, I don't do public policy. I  
5 believe that people shouldn't have to be involuntarily  
6 exposed to something that they don't want to be exposed to,  
7 but...

8 DR. DAVIS: Let me put it in personal terms then.  
9 If we take a hypothetical toxin and there's no threshold  
10 where exposure is safe, would you yourself want to be  
11 exposed to it?

12 DR. BAYARD: No, I sit in the no smoking section.

13 DR. DAVIS: In terms of active smoking there was  
14 some discussion earlier today about thresholds for active  
15 smoking. I think Mr. Furr asked the question are there any  
16 studies that look at risk of disease in those who smoke one  
17 cigarette a day. Would you agree with the statement that in  
18 epidemiologic studies there's probably a problem in  
19 estimating risk of disease in those who smoke one cigarette  
20 a day because not that many people smoke one cigarette a  
21 day? Would that make sense to you?

22 DR. BAYARD: Yes. The studies that we presented in  
23 our Chapter 4 on active smoking found dose response linear  
24 relationships, a lot of them at low doses. But again, there  
25 are very few people who smoke that little a day.

1 DR. DAVIS: Would it surprise you to hear a  
2 statistic that about 90 percent of smokers smoke five or  
3 more cigarettes a day?

4 DR. BAYARD: No. Our estimates were not at the 90  
5 percent levels. We were at the, our estimates were that the  
6 average smoker smoked around 19 cigarettes a day.

7 DR. DAVIS: I'm not talking about the average.  
8 I'm talking about the percentage of all smokers, statistics  
9 from the national health interview survey show that about 90  
10 percent of smokers smoke five or more cigarettes a day. In  
11 other words, only ten percent smoke less than five  
12 cigarettes a day. Do those numbers sound reasonable to you?

13 DR. BAYARD: It sounds reasonable when you  
14 consider the distribution, but it was not within the view of  
15 what we really needed to develop our data.

16 DR. DAVIS: Would you agree with the point that  
17 because there are so few smokers smoking only one cigarette  
18 a day, therefore, most of the studies that look at active  
19 smoking would look at a range of cigarettes smoked per days  
20 such as one to four or one to nine?

21 DR. BAYARD: That's exactly what we present in  
22 Table 4, yes.

23 DR. DAVIS: Is it your understanding that most of  
24 the scientific literature that has looked at smoking risk  
25 among that lowest dose have found increased risk of cancer

1 and other diseases?

2 DR. BAYARD: Yes. I would stick to lung cancer.  
3 I'm not familiar with the data on other diseases.

4 DR. DAVIS: So the main point is that when you  
5 look at the lowest exposure level for active smoking, where  
6 there's a sample size that allows you to generate a valid,  
7 relative risk estimate, you will see an increased risk of  
8 lung cancer?

9 DR. BAYARD: Yes. Our statement was that there  
10 was no evidence of a threshold.

11 DR. DAVIS: Thank you.

12 There was a question posed to you about whether a  
13 member of the lay public could understand the quantitative  
14 risk estimate that OSHA has offered for public comment and I  
15 think your answer to that was that typical members of the  
16 lay public would probably have a hard time understanding  
17 OSHA's quantitative risk assessment. Aren't most risk  
18 assessments, if not all risk assessments, very technical  
19 type of analyses that would be beyond most members of the  
20 general public?

21 DR. BAYARD: Yes. I can further say that there  
22 are very few people that have read our report.

23 DR. DAVIS: You think most members of the general  
24 public would be able to well understand the EPA risk  
25 assessment or various other risk assessments from the EPA?

1 DR. BAYARD: I can speak probably more  
2 authoritatively about the ETS risk assessment and I suspect  
3 that most members of the general public wouldn't even delve  
4 into it.

5 DR. DAVIS: There was some suggestion that  
6 Mr. Furr made, I believe, that OSHA had some sort of a  
7 priori opinion about ETS and lung cancer risk before it  
8 began its work but would you agree that OSHA was not in any  
9 sort of scientific vacuum when it began its work on ETS,  
10 that in fact a great deal of work had been done on ETS  
11 before OSHA began the work that we're talking about now?

12 DR. BAYARD: I think -- I don't know if I  
13 completely agree with that because I think OSHA began this  
14 work actually in 1987 and that would have come directly  
15 after the National Academy of Sciences --

16 DR. DAVIS: Well --

17 DR. BAYARD: I'm sorry. Let me finish, please.  
18 And the Surgeon General's report. And so if they did that  
19 in 1987, they certainly would have had two fine reports from  
20 which to make an estimate.

21 DR. DAVIS: Well, let me put the point in time as  
22 the date on which it released material for public comment.  
23 I don't have the exact date on that.

24 DR. BAYARD: I hope they would have read and  
25 agreed with the EPA report.

1 DR. DAVIS: That was more like 1993 or so? The  
2 first OSHA material on ETS made available for public  
3 comment?

4 DR. BAYARD: 1991, wasn't it?

5 MS. SHERMAN: The proposal was published in 1994,  
6 in March.

7 MS. JANES: No, it was April.

8 MS. SHERMAN: Excuse me.

9 MS. JANES: 1994.

10 DR. DAVIS: April 1994. My question is when OSHA  
11 released that material for public comment in April of 1994,  
12 was it operating in any sort of science vacuum at that  
13 point?

14 DR. BAYARD: I hope not.

15 DR. DAVIS: So it would be unreasonable to expect  
16 OSHA not to have any opinion at all about ETS and lung  
17 cancer risk at that point in time. Would you agree with  
18 that?

19 DR. BAYARD: What was the question?

20 DR. DAVIS: In April of '94 when OSHA released  
21 material for public comment on ETS, would you have expected  
22 them to be absolutely neutral on the issue? I mean, let me  
23 rephrase that. Would you expect them to have formed no  
24 scientific opinion at all about ETS in April of '94?

25 DR. BAYARD: Would I have expected them to have --

1 JUDGE VITTON: Excuse me. I really don't see the  
2 value in this line of questioning right here, considering  
3 the discussion we had previously. I refuse to allow the  
4 exploration as part of this. You're getting him to state  
5 something that probably is obvious to everybody in this  
6 room.

7 DR. DAVIS: That's fine, Judge. I can move on.  
8 The reason why I wanted to explore this was because Mr. Furr  
9 was implying that OSHA had some sort of bias when it first  
10 started looking into this issue and the point that I wanted  
11 to make is that OSHA didn't enter this area before any work  
12 had been done.

13 JUDGE VITTON: Mr. Furr was not testifying.  
14 What's important here is the testimony we get from the  
15 witnesses.

16 DR. DAVIS: Right. And that's what I was  
17 attempting to clarify but I'll move on, Your Honor. Thank  
18 you.

19 You were asked earlier to comment on issues  
20 relating to uncertainty and statistical variability and so  
21 on. Is uncertainty present in most epidemiologic studies,  
22 in most risk assessments?

23 DR. BAYARD: Yes.

24 DR. DAVIS: Are you aware of any epidemiologic  
25 study or risk assessment that has no uncertainty at all?

1 DR. BAYARD: No.

2 DR. DAVIS: Do you think that the level of  
3 uncertainty or statistical variability in the EPA risk  
4 assessment is average or more or less than the typical risk  
5 assessment that EPA does?

6 DR. BAYARD: I stated that we have medium to high  
7 confidence. The answer is less. We stated that we have  
8 medium to high confidence in the estimate, not only in the  
9 hazard identification of the qualitative evaluation of Group  
10 A or known human carcinogens but in the population risk  
11 estimate also because of the low amount of extrapolation  
12 required to extrapolate from typical environmental exposures  
13 to other conditions. There is some degree of uncertainty  
14 because, again, we extrapolated from females to males and  
15 from -- and we made some estimates about the prevalence of  
16 spousal exposure and we did the best we could and some of  
17 that has to be uncertain. I don't think they account for  
18 great variability but there's some uncertainty in that.

19 DR. DAVIS: But the findings are fairly robust  
20 compared to findings that you would derive from a typical  
21 risk assessment?

22 DR. BAYARD: Well, I'd say certainly. You know,  
23 when you're going from animals to humans and saying, gee,  
24 we've got two animal studies, guilty, next, let's do a risk  
25 assessment and extrapolate downwards from animals to humans,

1 from 10,000 down to one, I'd say that's a fairly high degree  
2 of uncertainty compared to having the accumulated evidence  
3 from 30 epidemiology studies all at typical environmental  
4 levels.

5 DR. DAVIS: Dr. Bayard, you talked about the issue  
6 of applying female risk data to males.

7 DR. BAYARD: Mmm-hmm.

8 DR. DAVIS: Is there any reason to believe that  
9 males and females would differ in any substantive way in  
10 their susceptibility to the effects of smoking or ETS  
11 exposure?

12 DR. BAYARD: The answer is that not substantively.  
13 However, the male background rate for lung cancer is higher  
14 for male non-smokers than it is for female non-smokers.  
15 It's not a whole bunch higher but it is a little higher.  
16 However, we did not -- we didn't do what OSHA did. We  
17 didn't use the male background rate and multiply it by the  
18 relative risk to the females. We did the extra risk to  
19 males based on the extra risk to females. We used an  
20 additive model. Using an additive model, we would have  
21 underestimated the risk to males if the true risk to males  
22 is relative. If the true risk to males is additive, we  
23 probably hit it right on the nose.

24 DR. DAVIS: All other things being equal, say, in  
25 terms of level of exposure and duration of exposure, there's

1 no biologic reason why males and females should react any  
2 differently to ETS exposure.

3 DR. BAYARD: You're probably more of an expert on  
4 that than I am. I don't know if there's a hormonal  
5 difference and why males or females ought to have different  
6 lung cancer rates. I know animals of the species can often  
7 vary in lung cancer rates.

8 DR. DAVIS: Well, in humans you mentioned the  
9 relative risk of lung cancer from active smoking and I don't  
10 remember the exact figure that you threw out but the current  
11 population survey number two showed a relative risk of lung  
12 cancer of 22 in males for active smoking and 12 for females,  
13 so that probably relates to duration of exposure, females  
14 have a lower relative risk because they typically haven't  
15 been smoking for as many years compared to males, maybe they  
16 smoke fewer cigarettes a day as well. But generally  
17 speaking, there is a biologic response to smoking in terms  
18 of lung cancer risk in both males and females, is that  
19 correct? Both have an increased risk of lung cancer if they  
20 smoke.

21 DR. BAYARD: The answer is yes but I don't know if  
22 I'd characterize it the same way you do. I think you  
23 characterized it as saying that females smoke fewer years  
24 than males and therefore the female relative risk should be  
25 lower than males. I think the female lung cancer rate may

1 be lower than males because females have smoked fewer years  
2 but when you do relative risk estimates, you're usually  
3 looking at specific females and so you would go back and  
4 assume -- and look at their history. In general, the  
5 female's history, if people are going to start smoking,  
6 they'll probably start smoking before they're age 18 or 19.  
7 And so I don't think I can agree with the statement on  
8 comparing the relative risk the way you did but I think I  
9 would agree on rates.

10 DR. DAVIS: Well, I'll move on. But the point is  
11 that women did take longer to take up smoking than men  
12 because men took up smoking in the 1920s and '30s in large  
13 numbers and women typically waited until after World War II  
14 and that's why in many of these epidemiological -- that's a  
15 major reason why many of these epidemiologic studies --

16 JUDGE VITTON: Dr. Davis --

17 MR. FURR: Your Honor, I object --

18 JUDGE VITTON: Dr. Davis --

19 DR. DAVIS: I'll move on. I'll move on. Thank  
20 you.

21 JUDGE VITTON: You're not testifying today, sir.

22 DR. DAVIS: Dr. Bayard, there was some discussion  
23 earlier about meta-analysis.

24 DR. BAYARD: Yes, sir.

25 DR. DAVIS: And also you were asked whether there

1 has been -- you were asked about meta-analyses and work site  
2 studies on ETS?

3 DR. BAYARD: Yes.

4 DR. DAVIS: Let me pose this question to you. We  
5 have many meta-analyses on ETS exposure in the home, the  
6 spousal studies, is that correct?

7 DR. BAYARD: Yes.

8 DR. DAVIS: And risk assessments as well.

9 DR. BAYARD: Yes.

10 DR. DAVIS: Do you think it's necessary once that  
11 meta-analysis is done showing that ETS is a cause of lung  
12 cancer, once a meta-analysis is done on that for exposure in  
13 the home that one needs to repeat those same sites of  
14 studies and then follow them up with meta-analyses for every  
15 different site where a person may be exposed to ETS?

16 DR. BAYARD: I'm going to break that answer into  
17 two, if I could. The answer is no but I thought the  
18 question, I don't know whether your question is referring to  
19 a qualitative analysis or to a quantitative analysis because  
20 if you're talking about population risk estimates, under  
21 certain circumstances a meta-analysis can provide you better  
22 risk estimates because you're pooling data from different  
23 types of different groups, just like the Fontham study had  
24 one type of group and the Brownson study might have had  
25 another. Some of that might work for spousal studies. I

1 think that the workplace studies have a peculiar set of  
2 problems that one has to be more careful with and I tried to  
3 relate that today in my direct testimony in saying look at  
4 the proxy response because the recall with workplace  
5 exposure is fuzzy anyway if people give their own direct  
6 response. Look at the proxy response and see how that plays  
7 into effect. I think the reliability falls off dramatically  
8 and that's before you can even establish the exposure  
9 patent. The nice thing about the spousal studies is  
10 exposure would tend to be constant if you can rationalize  
11 and say they're married to the same man for a lot of years,  
12 he smokes the same for a lot of years, they probably live in  
13 the same house for a lot of years, so they're probably  
14 getting the same type of smoker density and exposure levels  
15 at home. I don't think you can make anything like that type  
16 of conclusion about exposure patterns for the workplace.

17 For example, you know, a lot of the argument about  
18 smoking is that the earlier you start smoking, the more you  
19 smoke, the higher your risks. But that's establishing a  
20 consistent pattern in general. When you do workplace  
21 studies, I think that pattern is if not violated has to be  
22 very carefully watched.

23 DR. DAVIS: Dr. Bayard, is asbestos a Group A  
24 carcinogen?

25 DR. BAYARD: Yes. Actually, it's considered Group

1 A by inhalation. I don't think it's considered -- I think  
2 it's considered a Group C by ingesting.

3 DR. DAVIS: Okay. Let me refer to inhalation and  
4 draw an analogy between asbestos and ETS. Would it be true  
5 to say that most of the early evidence on the relationship  
6 between asbestos and lung cancer came out of studies of  
7 shipyard workers?

8 DR. BAYARD: Shipyard and insulation workers.  
9 Yes.

10 DR. DAVIS: Okay. Once it was determined that  
11 shipyard workers were at a greatly increased risk of lung  
12 cancer and mesothelioma, I suppose you could add, do you  
13 think it's necessary to do separate studies for asbestos  
14 exposure in other settings, for example, schools or work  
15 sites besides shipyards before we make a pronouncement from  
16 the EPA or OSHA or anywhere else that you should try and  
17 avoid exposure to asbestos?

18 DR. BAYARD: I'd say you should try to avoid  
19 exposure to asbestos but -- it's all right.

20 DR. DAVIS: In order to determine that children in  
21 schools with asbestos have an increased risk of getting lung  
22 cancer later in life, wouldn't it be necessary to probably  
23 do some sort of prospective study, follow these children  
24 over many years, see if the ones in schools with asbestos  
25 were more likely to get lung cancer compared to kids in

1 schools without asbestos?

2 DR. BAYARD: I think at this age, at this time,  
3 since asbestos has probably been around for 40, 50 years in  
4 schools, you can probably do somewhat of a retrospective  
5 study.

6 DR. DAVIS: If we go back 20 years or so, would it  
7 be prudent to say, okay, let's set up a study and follow  
8 children for 20 or 30 years and see if those who are exposed  
9 to asbestos in the schools have a higher risk of lung cancer  
10 than students who are not? Would it have been appropriate  
11 to set up a study like that and wait 20 years before trying  
12 to get asbestos out of schools?

13 MR. FURR: Your Honor, I object. He's talking  
14 about what we should or shouldn't do about asbestos and has  
15 asked no questions about the rulemaking.

16 DR. DAVIS: Your Honor, the analogy here is  
17 comparing -- is going from the spousal studies to the work  
18 site studies and Mr. Furr and others have constantly made  
19 the point that we have to wait and do all the same sorts of  
20 studies in the workplace like we've had for the spousal  
21 studies and we can't take action until that happens. And I  
22 think this analogy is a very appropriate one to undermine  
23 the point that the critics are making.

24 JUDGE VITTON: I can see the analogy somewhat  
25 but --

1 DR. DAVIS: I'll move on, Your Honor.

2 DR. BAYARD: I think -- if I can --

3 DR. DAVIS: I'll move on. I think the point's  
4 been made.

5 Dr. Bayard, you talked about the level of exposure  
6 to ETS in the workplace compared to levels of exposure in  
7 the home.

8 DR. BAYARD: Yes.

9 DR. DAVIS: If you assume -- I think your  
10 testimony has been that typical levels of exposure in many  
11 work sites are comparable to the levels in the home that  
12 were found in the spousal studies. Is that correct?

13 DR. BAYARD: Based on the studies that we  
14 evaluated, that was true.

15 DR. DAVIS: And that being the case, is there any  
16 reason to believe that ETS exposure in the workplace is any  
17 less hazardous than ETS exposure would be in the home?

18 DR. BAYARD: No. I'm sorry. I assume that  
19 cumulative dose over a day, say, if they're the same in the  
20 workplace as they are in the home they should produce the  
21 same effect.

22 DR. DAVIS: Thank you. You acknowledged earlier  
23 today that you had some concerns about some aspects of  
24 OSHA's quantitative risk assessment. Is that correct?

25 DR. BAYARD: Mmm-hmm.

1 DR. DAVIS: Is it your understanding that the  
2 quantitative risk assessment that OSHA made available for  
3 public comment is to be considered a preliminary document  
4 and is certainly not a final document by any means, is that  
5 your understanding?

6 DR. BAYARD: That's what I thought we were here  
7 for, to make comments.

8 DR. DAVIS: And that any criticisms that you might  
9 have made or that anybody else might have made would  
10 certainly be considered seriously by OSHA and perhaps it  
11 might make modifications to its own risk assessment, just as  
12 EPA did to its. Is that your understanding?

13 DR. BAYARD: Yes. We made a lot of modifications  
14 to our risk assessment.

15 DR. DAVIS: And so you would expect that OSHA  
16 would consider testimony such as yours, public comments,  
17 feedback from its experts and consultants and then make any  
18 appropriate revisions to its risk assessment and its  
19 recommended policy before we could consider anything final  
20 by any means?

21 DR. BAYARD: Yes.

22 DR. DAVIS: A point was brought out earlier that  
23 exposure levels of ETS in the workplace now may be different  
24 from levels 10 years ago or 15 years ago. Do you think that  
25 ETS exposure still occurs at significant levels in work

1 sites now in the United States?

2 DR. BAYARD: Yes.

3 DR. DAVIS: I think you testified that there's  
4 probably a variety of exposure levels in different work  
5 sites. Is that correct?

6 DR. BAYARD: Yes.

7 DR. DAVIS: So that even if average levels of  
8 exposure have come down in U.S. work sites over the last ten  
9 years, say, certainly there would be some work sites where  
10 exposure would be occurring at levels of concern, would you  
11 agree with that?

12 DR. BAYARD: Yes.

13 DR. DAVIS: And let me give you a number and see  
14 if this seems reasonable to you. According to the Bureau of  
15 National Affairs in a 1991 study, only about a third of work  
16 sites had bans on smoking at the work site. Does that sound  
17 about right to you? Would you have any reason to question  
18 that figure?

19 DR. BAYARD: I have no reason to question it. I  
20 just don't know it.

21 DR. DAVIS: So that means that about two-thirds of  
22 work sites as of 1991 would have allowed smoking and there  
23 most probably would have been significant exposure in those  
24 work sites of their employees, would you agree with that?

25 DR. BAYARD: I have no reason to agree or disagree

1 with it. I can give you some personal experiences, I can  
2 give you the data that we have on specific work sites but I  
3 can't give you the exact prevalence as to who's banning  
4 what.

5 DR. DAVIS: No, I'm not asking for an exact  
6 prevalence but just your sort of general feeling. EPA has  
7 recommended that smoking in the workplace be banned or  
8 limited to specially ventilated smoking areas. Is that  
9 correct?

10 DR. BAYARD: Yes, it has.

11 DR. DAVIS: Now, in those two-thirds of companies  
12 in 1991 that were allowing smoking, how many of them do you  
13 think -- do you think a significant number of them were  
14 allowing smoking without having special ventilation such as  
15 what EPA has recommended?

16 DR. BAYARD: That's beyond my expertise.

17 DR. DAVIS: Okay. Mr. Furr asked you some  
18 questions about the opinions of members of the Science  
19 Advisory Board on the EPA risk assessment.

20 DR. BAYARD: Yes.

21 DR. DAVIS: Is it correct to say that the SAB did  
22 endorse the overall conclusion that ETS is a Group A  
23 carcinogen?

24 DR. BAYARD: Yes. Unanimously. That was 18  
25 people and it was a unanimous endorsement and I have

1 submitted that report, November 20, 1992, to OSHA.

2 DR. DAVIS: Thank you.

3 DR. BAYARD: Are you aware of any scientific or  
4 medical organization that has endorsed the findings of the  
5 EPA or come to the conclusion that ETS is a cause of cancer  
6 in humans?

7 DR. DAVIS: Yes.

8 DR. BAYARD: Are you aware that the AMA has  
9 endorsed this finding?

10 DR. DAVIS: The AMA has, the National Cancer  
11 Institute endorsed it and in fact thought so much of our  
12 report that they republished it under their own monograph  
13 series and distributed it. I think there were about 10,000  
14 copies they distributed. The American Public Health  
15 Association. Public Citizen. There were several more but I  
16 just don't remember them right now.

17 DR. BAYARD: EPA wasn't the first federal agency  
18 to conclude that passive smoking or ETS exposure is a cause  
19 of lung cancer, was it?

20 DR. DAVIS: No, that was the Surgeon General in  
21 1986.

22 DR. BAYARD: And that was a report that was  
23 released by the U.S. Department of Health and Human  
24 Services? Is that correct?

25 DR. DAVIS: That's correct.

1 DR. BAYARD: And that report represented the  
2 conclusion of that department as a whole, is that correct?

3 DR. DAVIS: I think so.

4 DR. BAYARD: And has the National Institute for  
5 Occupational Safety and Health had anything to say about  
6 ETS?

7 JUDGE VITTON: Dr. Davis, your questions are  
8 relevant but this record, we're in the 76th day. We have  
9 had NIOSH, we have had the AMA, we have had all of these  
10 reports discussed at an earlier date.

11 DR. DAVIS: I'll move on. Thank you.

12 Dr. Bayard, when an agency issues a report it goes  
13 through a very careful peer review process. You testified  
14 to that, I think, didn't you, about your report?

15 DR. BAYARD: Yes.

16 DR. DAVIS: Internal and external review?

17 DR. BAYARD: Yes.

18 DR. DAVIS: And in your testimony earlier today,  
19 in your written statement, you pointed out that the report  
20 received many internal reviews, mostly from within ORD.  
21 Various parts of it were also reviewed by outside experts,  
22 both from other federal agencies and from academic  
23 institutions, revisions incorporated the reviewers' comments  
24 wherever possible. Does that sound accurate? I was just  
25 reading from your written statement.

1 DR. BAYARD: Yes.

2 DR. DAVIS: And so if there had been any critique  
3 of the draft report internally then you would have responded  
4 to those as appropriate, is that correct?

5 DR. BAYARD: Whenever we could. There are often,  
6 as you must know --

7 JUDGE VITTONI: Gentlemen, I wouldn't let Mr. Furr  
8 get into this and I'm not going to let you get into it.

9 DR. DAVIS: Your Honor, here's my point. Mr. Furr  
10 asked a number of questions on this and also made statements  
11 on this about allegations that there was dissension in the  
12 EPA about the risk assessment. To my knowledge, even though  
13 Dr. Bayard was not permitted to answer those questions, the  
14 questions and the statements by Mr. Furr are part of the  
15 record and if you would be willing to strike those from the  
16 record, I'd like to move on.

17 JUDGE VITTONI: No, I'm not willing to strike them  
18 and I'm not willing to explore the area and now give him an  
19 opportunity to explain or to go into it at all. Mr. Furr's  
20 questions are questions. They are not evidence. I can't  
21 believe that they will ever be considered to be evidence.

22 DR. DAVIS: I'll move on, Your Honor, but I would  
23 state for the record that Mr. Furr made some statements and  
24 made some allegations and I think that was inappropriate  
25 that those cannot be answered.

1 JUDGE VITTON: I'm sure Mr. Furr is going to  
2 stand up and agree with you but --

3 DR. DAVIS: Only to the extent that he's made the  
4 allegations, they should be refuted to the same level of  
5 detail.

6 JUDGE VITTON: Okay. But it is not this  
7 forum's --

8 DR. DAVIS: I'll move on, Your Honor.

9 Dr. Bayard, you were asked about the issue of  
10 diet, possible confounding. And the EPA report does talk  
11 about the attempts to address possible confounding by diet.  
12 I think you mentioned page 5-56 in the report.

13 DR. BAYARD: Yes.

14 DR. DAVIS: And I would like to just draw your  
15 attention to a statement at the top of page 5-56, starting  
16 on the first line, "However, for diet to explain fully the  
17 significant association of ETS exposure in Greece, Hong  
18 Kong, Japan and the United States which differ by diet as  
19 well as other lifestyle characteristics, it would need to be  
20 shown that in each country there is a diet protective  
21 against lung cancer from ETS exposure, diet is inversely  
22 associated with ETS exposure and, three, the association is  
23 strong enough to produce the observed relationship between  
24 ETS and lung cancer." You would agree, I would imagine,  
25 with that statement.

1 DR. BAYARD: Yes.

2 DR. DAVIS: Okay. Now, Mr. Furr was asserting  
3 that beta carotene intake, I believe, or some sort of  
4 measure of diet is linearly related to ETS exposure, so let  
5 me put this question to you. If you assume that beta  
6 carotene reduces lung cancer risk, if you assume that, I  
7 don't think the EPA risk assessment is willing to make that  
8 assumption because lower on this page it says there may be a  
9 protective effect of beta carotene, but if you assume that  
10 beta carotene reduces lung cancer risk, okay? And if you  
11 assume that beta carotene consumption is inversely related  
12 to ETS exposure, then you assume that beta carotene  
13 consumption is not only inversely related to ETS exposure  
14 but there's a linear inverse relationship and then you have  
15 to assume, do you not, one more thing and that is that all  
16 of that is true in Greece, Hong Kong and the United States  
17 to fully account for the association between ETS and lung  
18 cancer risk. That was a long question and you may not have  
19 followed it but the point I'm trying to raise is you have to  
20 go through a series of assumptions in order to explain away  
21 this association by beta carotene or diet, is that correct?

22 DR. BAYARD: The answer is correct but I would  
23 like to go further. As I suggested to Mr. Furr that the  
24 Fontham study actually did control for this. I understand  
25 that the Kalandidi study adjusted for diet and found no

1 interaction effect. Furthermore, when I presented the  
2 highest exposure data, the highest exposure data was still  
3 showing 80 to 100 percent increases and I think that the  
4 calculations by LaMarchand which is from the paper that  
5 Mr. Furr referred to, they talked about a possible  
6 confounding effect or potential confounding effect of up to  
7 20 percent. The studies to which I referred, the actual  
8 lung cancer studies to which I referred, actually found no  
9 confounding effect. Even Brownson who found an effect from  
10 diet, I think it was high fat, that the highest exposure  
11 group, didn't find any interaction effect between  
12 environmental tobacco smoke and diet.

13 DR. DAVIS: And in fact further on this page with  
14 the paragraph beginning "It was found," the report states,  
15 "It was found that nine of the studies have data on diet  
16 although only five of them use a form of analysis that  
17 assesses the impact of diet on ETS association. None of  
18 those five studies," and then it lists them, "found that  
19 diet made a significant difference." That's the point that  
20 you were making a moment ago, is that right?

21 DR. BAYARD: That's correct.

22 DR. DAVIS: Dr. Bayard, you were asked about dose  
23 response earlier today and is it correct to say that dose  
24 response is only one part of the criteria that we  
25 traditionally use to define causality?

1 DR. BAYARD: Yes.

2 DR. DAVIS: And the criteria that are  
3 traditionally used to define associations as being causal  
4 were referred to in the report, if I understand correctly,  
5 on page 5-66 and 5-67.

6 DR. BAYARD: That's correct. It was also in  
7 chapter one.

8 DR. DAVIS: And the evidence does show, in your  
9 opinion, a dose response relationship between ETS exposure  
10 and lung cancer risk, is that correct?

11 DR. BAYARD: That's correct.

12 DR. DAVIS: So that one criterion you believe is  
13 met, dose response relationship is met for the spousal  
14 studies.

15 DR. BAYARD: That's correct. As a whole.

16 DR. DAVIS: As a whole. That's right. And it's  
17 statistically significant by some of your tests for  
18 statistical significance, if I understand correctly.

19 DR. BAYARD: Yes. I think even more to the point  
20 with respect to dose response relationship and OSHA's need  
21 for study I think would be the Fontham '94 study.

22 DR. DAVIS: And the criterion temporal  
23 relationship on page 5-66 explains that the disease occurs  
24 within a biologically reasonable timeframe after the initial  
25 exposure to account for a specific health effect. Do you

1 believe that that criterion is met by ETS for lung cancer?

2 DR. BAYARD: Yes.

3 DR. DAVIS: And the next one is consistency. Now,  
4 that would refer to similar results in different countries  
5 by different investigators using different methodologies,  
6 studying different populations. Is that what that refers  
7 to?

8 DR. BAYARD: Tiers 1, 2 and 3. Yes.

9 DR. DAVIS: And generally you do have a consistent  
10 association between ETS exposure and lung cancer when you  
11 look at those different populations performed by different  
12 investigators using different methodologies, is that  
13 correct?

14 DR. BAYARD: Yes.

15 DR. DAVIS: And the conclusion of the EPA report  
16 is that most of these other criteria are met well enough to  
17 support the conclusion that this is a causal association  
18 we're talking about, is that correct?

19 DR. BAYARD: That's correct but I want to point  
20 out that you're only dealing with the ETS epidemiology  
21 studies in this section. ETS does not come out of thin air,  
22 as I've said many times, it comes from the burning of  
23 tobacco smoke. And when one assesses causality and  
24 sufficiency, one certainly has to include the epidemiology  
25 studies on active smoking and lung cancer.

1 DR. DAVIS: Dr. Bayard, the executive summary of  
2 the risk assessment provides some of the summary relative  
3 risks that you calculated? For example, I don't have a page  
4 number at the moment. Actually, it's page 1-10.

5 DR. BAYARD: Yes.

6 DR. DAVIS: The first bullet, the first complete  
7 bullet there. The fourth line down, it says for the United  
8 States, the summary estimate of relative risk for nine case  
9 controlled plus two cohort studies is 1.19 and then the  
10 confidence interval is shown. And the report points out  
11 that this summary relative risk and I believe the ones that  
12 follow are after adjustment for smoker misclassification.  
13 Is that correct?

14 DR. BAYARD: That's correct. But before  
15 adjustment for background exposure. In fact, I don't think  
16 we did adjustment for background exposure for the other  
17 countries, just for the U.S.

18 DR. DAVIS: Okay. That's the point I wanted to  
19 make, that most of these or all of these summary relative  
20 risks on these pages are actually quite conservative  
21 estimates because they don't account for background  
22 exposure.

23 DR. BAYARD: They refer to the effect of spousal  
24 smoking, they refer to the effect of spousal smoking plus  
25 background versus background. So if you can get a clean

1 control population, your risks should increase, risk  
2 estimates should increase.

3 DR. DAVIS: Right. Have you heard any substantive  
4 criticisms of the risk assessment after it was published in  
5 final form that you hadn't already heard before it was  
6 released when you went through that lengthy process of  
7 putting out the earlier draft for public comment and so on?

8 DR. BAYARD: I'm trying to think.

9 DR. DAVIS: Let me rephrase the question, if I  
10 can. You received a fair amount of public comment, did you  
11 not, with that first draft that was released for public  
12 comment?

13 DR. BAYARD: Yes. We modified the risk assessment  
14 extensively based on both the public comment and the SAB  
15 comments.

16 DR. DAVIS: Thank you. How would you compare the  
17 burden of ETS on public health versus that of other indoor  
18 air pollutants? Do you have any sense of that?

19 DR. BAYARD: Well, from a population perspective,  
20 it tends to be quite large. I've read some other figures  
21 but --

22 DR. DAVIS: Your estimate on lung cancer was 3000  
23 deaths?

24 DR. BAYARD: Yes. That's the lung cancer but  
25 there are some very sensitive populations that are affected

1 by ETS and not from lung cancer. I'm not talking about  
2 heart disease because EPA hasn't done an analysis on it but  
3 we've certainly done analysis on the non-cancer respiratory  
4 effects.

5 DR. DAVIS: Most of the other Group A carcinogens,  
6 for example, are they responsible for deaths that are in the  
7 thousands such as ETS? Benzene or asbestos or some of the  
8 others?

9 DR. BAYARD: No.

10 DR. DAVIS: So the burden from ETS is much higher  
11 than from most of the other Group A carcinogens, as far as  
12 you understand.

13 DR. BAYARD: To the general public, yes.

14 DR. DAVIS: I want to ask you a question or two  
15 about your analysis by tiers. Can you give us a sense of  
16 what factors you used to decide whether to put a study in  
17 tier 1 or tier 2 and so on?

18 DR. BAYARD: I actually had a slide on that. It's  
19 extensive. I'd have to look it up and if we could -- unless  
20 I presented the slide, the answer will be extensive.

21 DR. DAVIS: Okay. Well, let me pose this question  
22 to you. Let me rephrase it so that you may not have to look  
23 it up. Would the tier 1 studies typically be better, would  
24 they typically be more likely to attempt to control for  
25 possible confounders?

1 DR. BAYARD: Both by design and by analysis.

2 That's correct. Those are two of the criteria.

3 DR. DAVIS: Okay. And let me turn your attention  
4 to the table on page 5-17.

5 (Pause)

6 DR. DAVIS: Actually, what I want is not page 5-17  
7 but table 5-17.

8 JUDGE VITTON: How much longer are you going to  
9 be, Dr. Davis?

10 DR. DAVIS: Probably about ten minutes.

11 Now, this table shows the relative risk estimates  
12 by country and by tier, is that correct?

13 DR. BAYARD: Yes.

14 DR. DAVIS: And if some of these potential  
15 confounders were actually doing a lot of confounding of the  
16 association, as you attempt to control for those  
17 confounders, should the relative risk change?

18 DR. BAYARD: Yes. If I understand the question  
19 correctly.

20 DR. DAVIS: Well, let me put it to you this way.  
21 If you have a relative risk, say, 1.4 that is unadjusted and  
22 then you attempt to adjust for confounders --

23 DR. BAYARD: Potential confounders.

24 DR. DAVIS: Potential confounders. And if that  
25 relative risk comes down significantly, does that mean that

1 there was confounding?

2 DR. BAYARD: No.

3 DR. DAVIS: If you adjust for confounding and the  
4 relative risk changes?

5 DR. BAYARD: No. From a statistical point of  
6 view, the way you adjust is you put in an interaction term  
7 and if your interaction term is significant, you can't  
8 adjust for the confounders.

9 DR. DAVIS: All right. Let me ask the question  
10 this way. You said that the tier 1 studies were better able  
11 to control for confounding than tier 2, tier 3, tier 4  
12 studies. Is that correct?

13 DR. BAYARD: Mmm-hmm.

14 DR. DAVIS: So if you still see an increased risk  
15 in the tier 1 studies, that gives you more reason to believe  
16 that confounding was not a major problem. Do you agree with  
17 that?

18 DR. BAYARD: Yes.

19 DR. DAVIS: The best designed studies, the ones  
20 that do control as best they can for confounding --

21 DR. BAYARD: Potential confounding.

22 DR. DAVIS: -- for potential confounding, if they  
23 still have an increased risk of lung cancer, then that gives  
24 you confidence that that's a real elevated risk.

25 DR. BAYARD: That's correct.

1 DR. DAVIS: Now, if you look at some of the  
2 relative risks here, for most countries, it seems that the  
3 relative risk where you include tier 1 in most cases is just  
4 as high or higher than the relative risk where you do not  
5 include tier 1. Let me just -- let's look at the United  
6 States for example. You have the 1.28 relative risk.

7 DR. BAYARD: That's correct.

8 DR. DAVIS: And is that now for the studies that  
9 are listed under both tier 1 and tier 2?

10 DR. BAYARD: The 1.28 is the Fontham study  
11 adjusted for smoker misclassification and other potential  
12 confounders. Other potential biases and confounders.

13 DR. DAVIS: All right. There's a footnote 2 at  
14 the heading for the column on the far left. It says "Each  
15 line contains the studies from the previous tiers plus those  
16 added."

17 DR. BAYARD: That's right.

18 DR. DAVIS: So explain that to me for these U.S.  
19 studies.

20 DR. BAYARD: Well, the 1.28 refers to the only  
21 study, the only U.S. study in tier 1, which was the Fontham  
22 '91 study. The 1.22 is when you pool Fontham, Butler,  
23 Corea, Garfinkel, Humble, Janerich, Gabert and Wu. So that  
24 no instead of just the Fontham study which provides the  
25 1.28, you now have eight studies and the total pooled

1 estimate from eight studies is 1.22, all adjusted for smoker  
2 misclassification.

3 DR. DAVIS: Let me move on to one other thing and,  
4 Judge, I'm just about done.

5 In the document that you released, EPA released,  
6 "Setting the Record Straight" --

7 DR. BAYARD: Yes.

8 DR. DAVIS: -- there's a figure in there that is  
9 entitled "30 Epidemiology Studies of ETS and Lung Cancer  
10 Risk." And at the bottom, there are some probability  
11 estimates.

12 MR. SHEEHAN: I'm sorry, Dr. Davis, what page are  
13 you on?

14 DR. DAVIS: Well, I don't have the original in  
15 front of me. It's the figure that says "30 Epidemiology  
16 Studies of ETS and Lung Cancer."

17 MR. SHEEHAN: He's got it now.

18 DR. DAVIS: Okay. At the bottom, on the far left,  
19 for example, it points out that nine of the 30 studies were  
20 statistically significant in terms of the overall risk with  
21 lung cancer.

22 DR. BAYARD: At the 5 percent level, one tailed.

23 DR. DAVIS: Right. And there was a 1 in 10,000  
24 chance that that would have occurred by chance alone.

25 DR. BAYARD: Yes.

1 DR. DAVIS: Okay.

2 DR. BAYARD: If there were no effect. That's  
3 right.

4 DR. DAVIS: If there were no effect. And  
5 similarly that nine -- it says 17 of 17 studies which  
6 characterized by exposure level showed an increased risk at  
7 the highest exposure level, nine of those were statistically  
8 significant. There's a 1 in 10 million probability that  
9 that would have occurred by chance.

10 DR. BAYARD: That's correct.

11 DR. DAVIS: And then so on, moving from left to  
12 right, incredibly low probability that these findings would  
13 have occurred by chance, 1 in a billion, 1 in 10,000.

14 DR. BAYARD: That's correct. But those aren't  
15 independent. You don't multiply 1 in 10,000 by 1 in 10  
16 million. These are just --

17 DR. DAVIS: No, I'm not attempting to do that.  
18 I'm just making the point, for example, that when one would  
19 hear a comment in isolation that most of these studies were  
20 not statistically significant one needs to keep in mind  
21 these sorts of numbers. Would you agree with that?

22 DR. BAYARD: Yes. One needs to also keep in mind  
23 that these studies had very low power, most of them, and  
24 that was what I tried to bring out in my direct testimony,  
25 so you would expect very few of these studies to be

1 statistically significant, just by power considerations  
2 alone.

3 DR. DAVIS: Now, those probabilities, the 1 in  
4 10,000, the 1 in 10 million, those probability estimates  
5 were derived by various statistical tests, is that correct?

6 DR. BAYARD: It was a simple binomial, that if  
7 there's no effect, what's the probability of getting these  
8 statistically significant results. The probability of  
9 getting one statistically significant result out of one test  
10 is .05. The probability of getting one statistically  
11 significant results of out two tests would then be .05 times  
12 .95 times two. And it builds up as a binomial probability.  
13 It's a simple counting rule, it's not a particularly  
14 sophisticated test at all.

15 DR. DAVIS: These numbers were published in the  
16 EPA risk assessment, these probability estimates?

17 DR. BAYARD: That's correct.

18 DR. DAVIS: I think a comment was made during  
19 these proceedings a couple of months ago that these numbers  
20 were not in the EPA report but they might have been put in  
21 there in a different form such as P less than 10 to the  
22 negative seven.

23 DR. BAYARD: They are in the EPA report.

24 DR. DAVIS: Right. For example, on the top of  
25 page 1-8, there is a figure there, P less than 10 to the

1 negative seven and that would be equivalent to the 1 in  
2 10 million figure, is that correct?

3 DR. BAYARD: That's correct.

4 JUDGE VITTON: Can you wrap it up, please,  
5 Doctor?

6 DR. BAYARD: Excuse me?

7 JUDGE VITTON: I'm talking to Dr. Davis.

8 DR. DAVIS: Your Honor, that's all I have. Thank  
9 you very much.

10 JUDGE VITTON: Five minutes.

11 (Whereupon, a brief recess was taken.)

12 JUDGE VITTON: We resume our questions of  
13 Dr. Bayard. I have called Mr. Furr back to the podium for  
14 further questioning. Mr. Eli and Mr. Andrade have conceded  
15 their time to Mr. Furr and I will allocate one more hour of  
16 time for Mr. Furr.

17 MR. FURR: Thank you, Your Honor.

18 Dr. Bayard, earlier today, didn't you testify that  
19 EPA had addressed many of the criticisms that its Scientific  
20 Advisory Board had made of the quantitative risk estimates  
21 contained in the report?

22 DR. BAYARD: Yes.

23 MR. FURR: Dr. Bayard, is that your colleague,  
24 Dr. Jinot, behind you?

25 DR. BAYARD: Yes.

1 MR. FURR: Didn't Dr. Jinot attend the 1993 Tox  
2 Forum on July 12th through 16th in Aspen, Colorado?

3 DR. BAYARD: Yes, I think so.

4 MR. FURR: And that meeting took place about six  
5 months after release of the final risk assessment, didn't  
6 it?

7 DR. BAYARD: Yes.

8 MR. FURR: And one of the topics at that meeting  
9 was the Environmental Protection Agency's report on ETS,  
10 wasn't it?

11 JUDGE VITTON: Well, wait a minute. If you're  
12 going to ask him questions --

13 Were you there?

14 DR. BAYARD: I was not there.

15 MR. FURR: He's been with EPA, Your Honor, an EPA  
16 attendee was at the meeting.

17 MS. SHERMAN: He doesn't know everything that  
18 everybody did.

19 MR. FURR: I hope he knows what his co-author of  
20 the report has done with respect to the risk assessment.

21 JUDGE VITTON: But if you're going to ask him  
22 questions about what occurred at that conference -- if you  
23 know but if you don't let's just cut it short and just say  
24 you don't, okay?

25 DR. BAYARD: Yes.

1 MR. FURR: Yes, you know what occurred? You know  
2 that Dr. Jan Stolwijk from Yale University made a  
3 presentation on the SAB's role in the production of the ETS  
4 risk assessment, don't you?

5 DR. BAYARD: I believe he did but I don't know  
6 that for sure.

7 MR. FURR: Dr. Stolwijk was vice chairman of the  
8 SAB subcommittee that reviewed the risk assessment, wasn't  
9 he?

10 DR. BAYARD: That's correct.

11 MR. FURR: I want to hand you a copy of the  
12 transcript of that meeting. It's titled "The Tox Forum 1993  
13 Annual Summer Meeting."

14 MR. WEINBERG: Your Honor, time is short. Some of  
15 us might want to ask some questions. If he wants to have  
16 this in the transcript and he wants to put it in the record,  
17 fine, let him put it in the record but this witness wasn't  
18 there.

19 JUDGE VITTONI: Okay. Let me find out what the  
20 question is first, though, all right?

21 What's the question with respect to this document?

22 MR. FURR: Dr. Bayard, isn't it true that six  
23 months after release of the final EPA risk assessment that  
24 the vice chairman of the SAB committee responsible for  
25 reviewing the risk assessment was still criticizing the

1 EPA's quantitative risk estimates?

2 DR. BAYARD: I'm going to respond to that by  
3 saying I don't know. I'm also going to respond to that by  
4 saying that we have the SAB report. The SAB report which  
5 was submitted to the administrator on November 20, 1992 and  
6 submitted to the record represents the SAB point of view. I  
7 am perfectly willing to go with that and if you want to  
8 make -- picking on any one individual member, well at least  
9 discuss what the consensus of the whole committee was.

10 JUDGE VITTON: Mr. Furr, because of the limited  
11 time, we will enter that -- if you want to submit that for  
12 the record, if there is some criticism there or whatever  
13 purpose you want to use it for, I'll take it into the  
14 record.

15 MR. FURR: Your Honor, the point I wanted to  
16 illustrate is that in fact the SAB did not unanimously  
17 endorse the quantitative risk assessment contained in the  
18 EPA risk assessment.

19 MR. SHEEHAN: Your Honor, I think Mr. Furr is  
20 testifying now. The report is in and it speaks for itself.

21 JUDGE VITTON: Yes. We'll put it in for the  
22 record and you can make whatever argument you want based on  
23 what it says, okay?

24 MR. FURR: Dr. Bayard, I want to ask you some  
25 questions now about statistical significance and the role of

1 chance. That's a very -- an assessment of statistical  
2 significance or the role of chance, the likelihood of chance  
3 producing an observed statistical association is a very  
4 fundamental statistical analysis that is always conducted,  
5 isn't it?

6 DR. BAYARD: Yes.

7 MR. FURR: In fact, one assesses the likelihood  
8 that chance is producing an observed association by  
9 assessing statistical significance, correct?

10 DR. BAYARD: Yes.

11 MR. FURR: I want to ask you a series of questions  
12 about EPA's assessment of statistical significance. These  
13 questions are all going to have to do with EPA's assessment  
14 of whether the ever versus never exposed risk estimates were  
15 statistically significant. You understand what I'm talking  
16 about, don't you?

17 DR. BAYARD: Yes. For the ETS lung cancer  
18 studies, the 30 studies.

19 MR. FURR: Right.

20 Dr. Bayard, we discussed earlier, you testified  
21 before Congressman Rose's subcommittee on July 21, 1993,  
22 didn't you?

23 DR. BAYARD: I think it was July 23rd.

24 MR. FURR: But you testified before the  
25 subcommittee.

1 DR. BAYARD: Yes.

2 MR. FURR: Do you still have the transcript up  
3 there?

4 DR. BAYARD: Yes.

5 MR. FURR: During your testimony, Dr. Bayard,  
6 didn't you testify that for the ever versus never exposed  
7 groups that either one or two of the U.S. spousal smoking  
8 studies were statistically significantly at the 95 percent  
9 level?

10 DR. BAYARD: Could you please direct me.

11 MR. FURR: Didn't you testify before the Rose  
12 subcommittee that either one or two of the U.S. spousal  
13 smoking studies were statistically significant at the 95  
14 percent level.

15 MR. SHEEHAN: Could you refer us to a page, if you  
16 have a page?

17 MR. FURR: Let's turn to page 18 of the  
18 transcript.

19 Let me rephrase the question.

20 I'm looking at your last response on the page.

21 Didn't you testify that for the ever versus never  
22 exposed that the Fontham and Correa studies were  
23 statistically significant at the 95 percent level?

24 DR. BAYARD: I didn't talk about the 95 percent  
25 level. I talked about the five percent significance level.

1 MR. FURR: By that you meant 95 percent confidence  
2 intervals, didn't you?

3 DR. BAYARD: No. You don't make statistical  
4 decisions on confidence intervals. You make them on  
5 statistical significance tests.

6 MR. FURR: Dr. Bayard, none of the U.S. spousal  
7 smoking studies are statistically significant at the 95  
8 percent confidence level, are they?

9 DR. BAYARD: You don't test at confidence levels.  
10 You test at significance levels.

11 MR. FURR: What significance level corresponds to  
12 the use of 95 percent confidence intervals?

13 DR. BAYARD: Usually a  $P=.05$  level.

14 MR. FURR: At the  $P=.05$  level, none of the U.S.  
15 spousal smoking studies are statistically significant for  
16 the ever versus never exposed group, are they?

17 DR. BAYARD: Adjusted or unadjusted for smoker  
18 status misclassification?

19 MR. FURR: Unadjusted.

20 (Pause)

21 DR. BAYARD: May I look? I'll be right there.

22 MR. FURR: Sure.

23 (Pause)

24 DR. BAYARD: At the five percent statistical  
25 significance level one-tail, I see only the Fontham study,

1 1991, out of the 11 U.S. studies statistically significant  
2 at the five percent level. At the two-tail test, which I  
3 think is what you're asking.

4 MR. FURR: Yes.

5 DR. BAYARD: The Fontham study would just not be  
6 quite statistically significant. I think the 95 percent  
7 confidence limit, lower 95 percent confidence limit is .99.

8 MR. FURR: And none of the other studies were  
9 statistically significant for the two-tailed  $P=.05$  test was  
10 it?

11 DR. BAYARD: No, but I would like to just explain,  
12 Your Honor.

13 MR. FURR: Either they were or they weren't.

14 DR. BAYARD: My explanation is that these figures  
15 are provided on Table 59, page 528 and 529, and one has to  
16 look at the very low power of these tests to detect  
17 statistical significance.

18 MR. FURR: You've made that point many times  
19 today.

20 DR. BAYARD: Let me just finish. The statistical  
21 significance is a function of sample size as well as  
22 strength of association.

23 Considering that of the 11 studies I think seven  
24 of them had under 45 cases, one would not expect the studies  
25 to have very much power. So when that happens, one looks

1 for other techniques for which to analyze the data. That's  
2 one of the reasons where went to a technique where we could  
3 combine data of similar design types.

4 MR. FURR: Dr. Bayard, in the 1990 review draft of  
5 the ETS risk assessment, didn't EPA predominantly use 95  
6 percent confidence intervals?

7 DR. BAYARD: For lung cancer.

8 MR. FURR: Why did you indicate...

9 DR. BAYARD: I'm sorry, I was just trying to...

10 MR. FURR: ...lung cancer.

11 DR. BAYARD: Yes, in the 1990 draft we used 95  
12 percent confidence intervals. However, we also used one-  
13 tail tests. We used one-tail tests in the 1990 draft. we  
14 used one-tail tests in the 1992 draft. We used one-tail  
15 tests in the final...

16 MR. FURR: But my question is about the confidence  
17 interval.

18 DR. BAYARD: The confidence interval does not  
19 distinguish statistical significance.

20 MR. FURR: But you did use predominantly 95  
21 percent confidence intervals in the 1990 draft.

22 DR. BAYARD: Yes.

23 MR. FURR: Why did you indicate to Congressman  
24 Rose's subcommittee that the EPA determined to use 90  
25 percent confidence intervals before EPA ever looked at the

1 data?

2 DR. BAYARD: Are you referring to page 19?

3 MR. FURR: Yes.

4 DR. BAYARD: May I read it for just a minute?

5 MR. FURR: Sure.

6 (Pause)

7 DR. BAYARD: Are you referring to the last  
8 statement on the page?

9 MR. FURR: It is at the bottom of the page, yes.

10 (Pause)

11 DR. BAYARD: In the interest of time, let me try  
12 to phrase the question and see if you agree with the  
13 question.

14 MR. FURR: Okay.

15 DR. BAYARD: I think your question is why did we  
16 decide to do a one-tail test.

17 MR. FURR: No, that's not the question. The  
18 question is why did you testify to Congress that you  
19 determined to use 90 percent confidence intervals before you  
20 had ever looked at the data?

21 DR. BAYARD: I think what I testified was that we  
22 determined to use a one-tail test. If you have strong  
23 enough belief that any effect you have is going to be  
24 adverse, you use a one-tail test, and that is exactly what  
25 we did. My belief is that any effect of environmental

1 tobacco smoke would be an adverse one for lung cancer. If  
2 there is an effect for lung cancer, it is not liable to help  
3 you. In order to... That statement was supported by the  
4 plethora of data we have on active smoking and lung cancer.

5 MR. FURR: Dr. Bayard, when EPA first began  
6 examining the ETS epidemiologic studies it utilized a two-  
7 sided 95 percent confidence interval to assess the role of  
8 chance, didn't it?

9 DR. BAYARD: No, it used a two-sided confidence  
10 interval to express the uncertainty around the estimate. It  
11 used a one-sided tail significance test to express the role  
12 of chance, and it used it in its first draft, it used it in  
13 its second draft, and it used it in its final report.

14 MR. FURR: What did EPA use in the 1988 draft by  
15 the Office of Air and Radiation?

16 DR. BAYARD: I do not know. I did not do that.

17 MR. FURR: But that draft was one of the reasons  
18 that the risk assessment that you were involved in was  
19 begun, wasn't it?

20 DR. BAYARD: Yes, and I'd like to explain that. I  
21 was asked to review the draft. I thought the draft was not  
22 adequate for EPA's needs, and I suggested that a more  
23 rigorous approach was needed.

24 MR. FURR: You reviewed the draft.

25 DR. BAYARD: I reviewed it cursorily. I didn't

1 review it page by page. I did notice it bore a very strong  
2 resemblance to the 1986 Surgeon General's and NRC report.  
3 As such, I thought that EPA should evaluate the data on its  
4 own.

5 MR. FURR: If you reviewed that draft, you know  
6 that two-sided test was used then, don't you?

7 DR. BAYARD: I didn't look to see what it was for.  
8 If I did know at the time, I don't know it now.

9 MR. FURR: You also know, don't you, that a meta-  
10 analysis was performed and presented in that 1988 draft  
11 which was not statistically significant using a two-sided  
12 test, don't you?

13 DR. BAYARD: I'm sorry. If I did know it then, I  
14 don't know it now. I don't remember.

15 MR. FURR: Did you know it when you appeared  
16 before Congressman Rose's subcommittee?

17 DR. BAYARD: The answer is no, but I'm trying to  
18 remember what I ever knew about that draft other than it was  
19 very similar to the 1986 Surgeon General's and NRC report.

20 MR. FURR: You simply don't remember what you  
21 knew.

22 DR. BAYARD: I think I told you just about most of  
23 what I remember about that.

24 MR. FURR: Did you send that draft to Ken Brown?

25 DR. BAYARD: I will say I think so. The reason I

1 think so is because I would try to give Ken Brown everything  
2 I had on this, but I don't remember specifically doing that.

3 MR. FURR: Let me hand you a copy of the draft and  
4 ask you to look at the cover page.

5 (Document handed to Dr. Bayard)

6 DR. BAYARD: Thank you.

7 (Pause)

8 MR. FURR: Highlighted on that cover page is a  
9 note that says, "Send to Ken Brown," is that correct?

10 DR. BAYARD: That's correct.

11 MR. FURR: Is that your handwriting?

12 DR. BAYARD: I know Bayard on the upper right is  
13 my handwriting. This one looks like my handwriting also.

14 MR. FURR: So you sent that draft to Ken Brown  
15 then. And Dr. Brown was the contractor responsible for  
16 chapters 5 and 6 of the EPA risk assessment.

17 MR. SHEEHAN: Your Honor, I'm going to object at  
18 this point. I think we're starting to get into the  
19 deliberative area again.

20 MS. NEUWIRTH: In addition, this report was  
21 prepared by the Office of Air and Radiation. Dr. Bayard is  
22 from the Office of Research and Development.

23 MR. FURR: Your Honor, he has claimed that EPA did  
24 not rely on this draft, and I'm attempting to impeach him on  
25 that issue.

1 JUDGE VITTON: Dr. Brown is an author or part of  
2 the authors as I recall?

3 DR. BAYARD: Dr. Brown is an author of this  
4 report, yes. A co-author of the EPA report. He is not an  
5 author of this draft.

6 MR. FURR: I am not inquiring into the  
7 deliberations of this draft. Simply the awareness of the  
8 existence of this draft and its findings by Dr. Bayard and  
9 Dr. Brown.

10 JUDGE VITTON: Okay, go ahead.

11 MR. FURR: We know then that Dr. Brown also knew  
12 that a two-sided statistical significance test of the meta-  
13 analysis was statistically insignificant.

14 DR. BAYARD: Yeah, we...

15 MR. MYERS: Your Honor, all we know is that this  
16 has some handwriting on it.

17 MR. SHEEHAN: We don't know what Dr. Brown knew.  
18 He can't testify to that.

19 MR. FURR: Did you ever have any discussions...

20 JUDGE VITTON: Let's find out if he does know  
21 anything from Dr. Brown. That's all.

22 MR. MYERS: That's the deliberative process.

23 MR. FURR: I'm not going to inquire into those  
24 discussions. I'm going to ask if they occurred to establish  
25 his awareness.

1                   You sent this to Dr. Brown, Dr. Bayard. Did you  
2 ever inquire of Dr. Brown whether he had received this  
3 draft?

4                   MS. NEUWIRTH: Isn't that inquiring about  
5 discussions?

6                   JUDGE VITTON: Can you answer the question?  
7 Just a second.

8                   Can you answer the question Dr. Bayard?

9                   DR. BAYARD: I'll try. What's the question?

10                  MR. FURR: Did you ever inquire of Dr. Brown  
11 whether he received this draft?

12                  DR. BAYARD: I don't remember. If I sent this to  
13 Ken, and from reading the instruction that I put on this I  
14 would imagine that I did send it to Dr. Brown, it would have  
15 been somewhere in '88 or '89, I believe, in that era. I  
16 don't even think he came on board until late '88 or '89. So  
17 it wouldn't have been... It would have been at least, I  
18 would imagine, five or six years ago.

19                  MR. FURR: And it would have been before EPA  
20 determined to use a one-sided statistical significance test,  
21 wouldn't it?

22                  DR. BAYARD: Oh, I think so.

23                  MR. FURR: Thank you.

24                  DR. BAYARD: I think we were still in the data  
25 gathering... We hadn't even got into the data gathering at

1 that time.

2 MR. FURR: In the EPA's review of the  
3 epidemiologic studies of disease in children, EPA utilized a  
4 two-sided test for statistical significance, didn't it?

5 DR. BAYARD: That's correct.

6 MR. FURR: Did EPA expect that environmental  
7 tobacco smoke exposure to children might have beneficial  
8 health effects?

9 DR. BAYARD: No, but there wasn't the plethora of  
10 evidence on lung cancer in adults that there had been from  
11 smoking. Children don't smoke, for example.

12 MR. FURR: Dr. Bayard, we know more about the  
13 mechanisms of the diseases for childhood respiratory  
14 illnesses than we do for lung cancer, don't we?

15 DR. BAYARD: With respect to tobacco smoke related  
16 cancers, I think that's correct.

17 MR. FURR: With respect to any cancers, I assume.

18 DR. BAYARD: I think some cancers are fairly well  
19 known.

20 MR. FURR: The mechanism of the cancer?

21 DR. BAYARD: Yeah, there's a mouse cancer, for  
22 instance, that is known to occur from a point mutation in  
23 one gene. I think there are several other cancers that have  
24 been well established. I think the eye, for example, no  
25 it's not that. There's a cancer of the eye that's known to

1 occur from two specific mutations, from genes. But I think  
2 with respect to lung cancer, I think there are multiple  
3 causes. It's not from one specific gene.

4 MR. FURR: Was the determination to use 90 percent  
5 confidence intervals made by EPA or was it recommended to  
6 them by outside consultants?

7 DR. BAYARD: For the lung cancer?

8 MR. FURR: For the lung cancer?

9 DR. BAYARD: As I remember it, if you'll allow me,  
10 I think it was Dr. Brown that first came in with that but I  
11 can't say for sure.

12 MR. FURR: Let me hand you a copy of an article  
13 that appeared in Investors Business Daily on January 28,  
14 1993, and ask you to turn to page four.

15 (Document handed to Dr. Bayard)

16 DR. BAYARD: Thank you.

17 (Pause)

18 MR. FURR: Do you see the highlighted material  
19 there?

20 DR. BAYARD: "Reilly said simply.."

21 MR. FURR: The former EPA Commissioner was William  
22 Reilly. Excuse me, Administrator was William Reilly,  
23 correct?

24 DR. BAYARD: Correct.

25 MR. FURR: Isn't he quoted there as saying, "With

1 respect to the confidence interval, we have here a 90  
2 percent confidence interval, and that was in fact what was  
3 recommended to us by the scientific community as being  
4 appropriate for this data."

5 (Pause)

6 MR. FURR: Is that correct?

7 DR. BAYARD: Yes. That's what Mr. Reilly said.

8 MR. FURR: Was he accurate in what he said?

9 DR. BAYARD: Mr. Reilly didn't clear that  
10 statement with me, but...

11 (Laughter)

12 MR. FURR: I doubt he cleared many statements...

13 (Laughter)

14 MR. FURR: If it isn't accurate, just tell us.

15 DR. BAYARD: I tried to tell you as best...

16 (Pause)

17 DR. BAYARD: I don't know the basis for that  
18 statement, but I think I answered that the 90 percent...  
19 I'm sorry.

20 The one-tailed five percent level was, the one  
21 tailed five percent significance level was first used by Ken  
22 as his idea. That's what I think...

23 MR. FURR: By Ken, do you mean Dr. Brown that you  
24 sent the 1988 draft risk assessment to?

25 DR. BAYARD: That same Dr. Brown.

1 MR. FURR: Isn't it true, Dr. Bayard, that Dr.  
2 Kabat of the Scientific Advisory Board admonished EPA that  
3 the use of a 90 percent confidence interval instead of the  
4 95 percent confidence intervals should be discouraged?

5 DR. BAYARD: The answer is I don't know. The  
6 answer I would like to explain. That is that we're getting  
7 into statements of individual members as opposed to the  
8 total report of the committee. What goes on in a meeting is  
9 what I consider deliberative process. What comes out of the  
10 committee and the report to the EPA was a result of that.  
11 The committee approved our methodology.

12 MR. FURR: Dr. Bayard, I asked you earlier, you're  
13 not contending that the SAB subcommittee that reviewed the  
14 risk assessment, approved and placed its stamp of approval  
15 on every single analysis or statement contained in the risk  
16 assessment, are you?

17 DR. BAYARD: No, I am not. However, this  
18 methodology, I think, was raised to the Science Advisory  
19 Board. I think I spoke with one of the members about it  
20 myself and raised the point. Our purpose in going before  
21 the Science Advisory Board was to apprise them of what we  
22 did and ask for their advice and concurrence of our  
23 suggestions for revisions.

24 MR. FURR: Can you point to anywhere in the SAB  
25 reports that the Science Advisory Board approves the use of

1 90 percent confidence intervals?

2 DR. BAYARD: On the specific reports, report one,  
3 and report two?

4 MR. FURR: And any others that I may not know  
5 about.

6 DR. BAYARD: I will go over the reports, the  
7 reports have already been submitted to OSHA. I know we  
8 asked about the methodology. But I will go through the  
9 reports.

10 MR. FURR: Dr. Bayard, you know that the SAB never  
11 specifically approved the use of 90 percent confidence  
12 levels, don't you?

13 MR. SHEEHAN: Your Honor, he just said he'd go  
14 through the reports.

15 JUDGE VITTON: I think we're beating this  
16 horse...

17 MR. FURR: I know he said he'd go through the  
18 reports, but he never answered my question.

19 MR. SHEEHAN: He'd like to see the reports before  
20 he answers your question is what he's saying.

21 DR. BAYARD: Mr. Furr, I haven't gone through the  
22 transcript of the record, and I don't want to take your word  
23 for it that we haven't discussed this with the SAB.

24 I will go through the reports, I will go through  
25 the transcripts, and if appropriate, I will submit the

1 material to OSHA for the record.

2 JUDGE VITTON: Aren't those already in the  
3 record, though? Are they going to be part of the record or  
4 what?

5 Dr. Bayard?

6 DR. BAYARD: I have not submitted the SAB  
7 transcripts for the record.

8 MR. FURR: Your Honor, this illustrates the  
9 problem again, I'm afraid. Dr. Bayard is justifying the use  
10 of the 90 percent confidence intervals based on what he  
11 calls deliberative process, off the record conversations  
12 with SAB members. He's using that to support his testimony,  
13 and I'm not being permitted to inquire into these  
14 deliberative...

15 MR. MYERS: Your Honor...

16 MR. FURR: May I finish, please?

17 MR. MYERS: ...based on a scientific reasons.  
18 He's explained those scientific reasons.

19 MR. FURR: Thank you, Mr. Myers. And Your Honor,  
20 I'm not being permitted to probe those conversations. So  
21 really, we're only getting one side of the picture here.

22 MR. SHEEHAN: Your Honor, he's trying to reopen it  
23 again.

24 JUDGE VITTON: Mr. Furr, I think we're getting  
25 into the process that I wanted you to stay away from.

1       Whatever the EPA report is, the EPA report is. You can  
2       explore what that is and challenge him. I've given you the  
3       opportunity, if you wanted to try to impeach his statements  
4       on a couple of occasions. That's fine. But I don't think  
5       we're getting anyplace here.

6               MR. FURR: I agree that's the issue we're getting  
7       into. I just want to make the point that the reason we're  
8       getting into it is he continues to rely upon those types of  
9       internal reviews as support for the positions that he's  
10      taken.

11             JUDGE VITTON: I don't really see it that way  
12      based on the answers that I've heard so far.

13             Let's move on.

14             MR. FURR: Dr. Bayard, of the 20 epidemiologic  
15      studies of spousal smoking and lung cancer that EPA  
16      reviewed, isn't it true that 27 of them reported their  
17      results with a standard two-sided 95 .05 level of  
18      significance?

19             DR. BAYARD: I think that is not true. The way I  
20      remember it when I did my count, four of them used 90  
21      percent confidence intervals; 18 of them used 95 percent  
22      confidence intervals; and eight of them used no confidence  
23      intervals at all.

24             MR. FURR: For the 18 studies that you recall  
25      using 95 percent confidence intervals, do you believe that

1 the investigators erred in using 95 percent confidence  
2 intervals?

3 DR. BAYARD: No. I believe that the investigators  
4 choose to use those confidence intervals to express their  
5 uncertainty about their estimate. When you combine data,  
6 though, you ought to use a meta-analysis to pool your data,  
7 you have to choose one level. You can choose a one-tail  
8 test, you can do a two-tailed test. You can report your  
9 data as 95 percent levels, you can report it as two, but it  
10 helps to try to be consistent.

11 MR. FURR: You would agree, wouldn't you, that one  
12 should not choose to report the data in one way or another  
13 based on an outcome determinative considerations. In other  
14 words, one should not choose to use a one-sided statistical  
15 significance test in order to achieve statistical  
16 significance that they know could not be achieved with a  
17 two-sided test.

18 DR. BAYARD: I firmly agree with that. And I  
19 would say that we did not do that.

20 MR. FURR: I didn't ask you that.

21 DR. BAYARD: No, but I just want to complete my  
22 answer, thank you.

23 We chose a one-tail test based on the plethora of  
24 evidence indicating that if there was to be an effect on  
25 lung cancer of environmental tobacco smoke, it would be an

1 adverse one and not a helpful one.

2 Now if we were testing something like beta  
3 carotene we might choose to use a one-tail test in the other  
4 direction. We might choose to do a two-tail test. It  
5 depends on the amount of evidence we would have on beta  
6 carotene and lung cancer in nonsmokers or in smokers.

7 MR. FURR: Earlier, Dr. Davis, I think it was,  
8 asked you about a number of analyses that EPA performed that  
9 I'll call count-based analyses. By that I mean the analyses  
10 where EPA assessed the likelihood of a certain proportion of  
11 studies or comparisons being statistically significant or  
12 positive.

13 DR. BAYARD: That's correct.

14 MR. FURR: Do you understand what I'm talking  
15 about?

16 DR. BAYARD: Yes.

17 MR. FURR: I think you said this was simply an  
18 exercise in binomial distribution?

19 DR. BAYARD: Pretty much.

20 MR. FURR: That's somewhat like flipping a coin,  
21 isn't it?

22 DR. BAYARD: Pretty much.

23 MR. FURR: It presumes that you're flipping a fair  
24 coin, doesn't it?

25 DR. BAYARD: That's correct.

1 MR. FURR: In other words, it presumes that the  
2 studies themselves are not, that the risk estimates being  
3 produced are not a product of bias or confounding.

4 DR. BAYARD: That's correct. Or at least that you  
5 try to account for all the upward bias.

6 MR. FURR: And if in fact one has not been able to  
7 account for the sources of bias and confounding, then these  
8 coin flipping analyses really aren't very informative, are  
9 they?

10 DR. BAYARD: That's correct.

11 MR. FURR: I want to talk to you about another  
12 epidemiologic principle, and that's the concept of  
13 evaluating the strength of an association. By that I mean  
14 simply how large the reported association is. You  
15 understand what I'm talking about...

16 DR. BAYARD: Yes, sir.

17 MR. FURR: It's EPA's position that the strength  
18 of the association is an important factor in interpreting  
19 whether an association has causal significance, isn't that  
20 correct?

21 DR. BAYARD: Are you referring to the guidelines?

22 MR. FURR: Among other references.

23 DR. BAYARD: Would you show me the reference?

24 MR. FURR: You have a copy of the guidelines up  
25 here don't you?

1 DR. BAYARD: Yes.

2 (Pause)

3 MR. FURR: You agree with that proposition, don't  
4 you? We don't have to document it in the guidelines if you  
5 agree with it.

6 DR. BAYARD: Just repeat it so I can get it right.

7 MR. FURR: That the strength of an association is  
8 an important factor to consider in determining whether or  
9 not a causal inference should be drawn from that  
10 association.

11 DR. BAYARD: It is a factor to consider, and it's  
12 one of the factors we considered, and it's one of the issues  
13 we just went over not too long ago where we talked about the  
14 strength of association being related to the amount of  
15 exposure.

16 MR. FURR: It's an important factor, isn't it?

17 DR. BAYARD: Strength of an association is a  
18 factor. It can be an important factor in some areas, but  
19 there are other areas where there are more important factors.  
20 I can explain but I don't think you want me to.

21 MR. FURR: Let's talk about why the strength of an  
22 association is an important factor.

23 DR. BAYARD: Oh, you do. Okay.

24 (Laughter)

25 MR. FURR: Dr. Bayard, I want to show you a paper

1 by Dr. Ernst Wynder. You're familiar with Dr. Wynder aren't  
2 you?

3 DR. BAYARD: I've never met him, but I've seen his  
4 work.

5 MR. FURR: You know him by reputation?

6 DR. BAYARD: He has several reputations.

7 (Laughter)

8 DR. BAYARD: No, no one doesn't have a total  
9 reputation in one, which is always one-sided.

10 MR. FURR: I'm only talking about his professional  
11 reputation right now.

12 (Laughter)

13 DR. BAYARD: I've heard many things about Dr.  
14 Wynder.

15 MR. FURR: You consider him to be a renowned  
16 epidemiologist, don't you?

17 DR. BAYARD: Dr. Wynder, I believe, was one of the  
18 first people to make the finding that smoking causes lung  
19 cancer, I believe, and that was way back in the early '50s,  
20 is that right?

21 MR. FURR: The point there is that he's certainly  
22 no ally of the tobacco industry, is he?

23 DR. BAYARD: You tell me.

24 (Laughter)

25 MR. FURR: I want to show you a paper published by

1 Dr. Wynder, in Volume 16 of Preventive Medicine in 1987  
2 titled, "Workshop on Guidelines to the Epidemiology of Weak  
3 Associations." I'd ask you to take a look at it.

4 (Document handed to Dr. Bayard)

5 DR. BAYARD: Thank you.

6 (Pause)

7 MR. FURR: Have you ever seen that paper before?

8 DR. BAYARD: I don't recall ever seeing it before.

9 MR. FURR: I'd ask you to take a look at the  
10 highlighted material at the bottom of page 139. I want to  
11 read part of that and ask you a few questions about it, if  
12 you'd read along with me.

13 "When risks are small, and especially when effects  
14 occur many years after their causes, detecting them,  
15 estimating their magnitude, and assessing their importance  
16 for the community in light of other relative factors, pose  
17 problems of study design, data collection, analysis and  
18 interpretation which can be exceedingly difficult."

19 Do you agree with that statement, Dr. Bayard?

20 DR. BAYARD: One minute.

21 (Pause)

22 DR. BAYARD: If you take out the adverb  
23 "exceedingly" I think... I think I would agree with it in  
24 general.

25 MR. FURR: So you would agree if it just read

1 "which can be difficult"?

2 DR. BAYARD: I'm sorry?

3 MR. FURR: You're saying that you agree, but you  
4 would just change the last sentence to read, "Which can be  
5 difficult."

6 DR. BAYARD: It can be.

7 MR. FURR: I want to hand you another paper, Dr.  
8 Bayard. This is an editorial by Marcia Angell that appeared  
9 in the September 20, 1990 New England Journal of Medicine.

10 (Document handed to Dr. Bayard.)

11 MR. FURR: Are you familiar with Dr. Angell by  
12 reputation?

13 DR. BAYARD: No.

14 MR. FURR: You're not aware that she's one of the  
15 assistant editors of the New England Journal of Medicine?

16 DR. BAYARD: No.

17 MR. FURR: Let me ask you if you've ever seen this  
18 paper before?

19 (Pause)

20 MR. FURR: Do you read the New England Journal of  
21 Medicine, Dr. Bayard?

22 DR. BAYARD: Occasionally. I'm not a subscriber  
23 to it, but we do have it in our library and many articles of  
24 interest have appeared there.

25 MR. FURR: Did you ever read that paper?

1 (Pause)

2 MR. FURR: If you're having difficult with that  
3 one, I'll withdraw it and ask you another question.

4 Would you take a look at the...

5 JUDGE VITTON: Is it familiar at all?

6 DR. BAYARD: I may have. It all depends on which  
7 subject I'm on at the time. It could be radon, it could be  
8 dioxin, it could be environmental tobacco smoke. A lot of  
9 editorials relate to articles in the Journal at that month  
10 in the New England Journal. So I don't know whether I read  
11 that. That's why I was trying to determine which study in  
12 the Journal they were referring to.

13 That's why I was looking and trying to see which  
14 study they were specifically thinking about. Can you tell  
15 me that?

16 MR. FURR: I'm not testifying, Dr. Bayard, but my  
17 understanding of this article is it is not in reference to  
18 any particular study, but it's just an editorial on, as it's  
19 titled, "The Interpretation of Epidemiologic Studies."

20 JUDGE VITTON: What's your question with respect  
21 to it? Maybe that...

22 MR. FURR: Can you take a look at page 824, the  
23 bottom left hand column. Is there some language highlighted  
24 there, Dr. Bayard? Second page.

25 DR. BAYARD: Yes. Second page, "An important

1 reason for being concerned about the size of the effect is  
2 that unknown or inadequately accounted for confounding  
3 variables can easily produce artificial small..."

4 MR. FURR: That's what I was thinking of. Do you  
5 agree with that statement?

6 DR. BAYARD: I agree with it if we're dealing with  
7 one study. Maybe if we're dealing with two or three or four  
8 studies. But when we have a plethora of evidence from  
9 multiple studies, consistency becomes more important than  
10 size.

11 For example, if we had small ones and large ones,  
12 I'd worry more about the large ones than the small ones, if  
13 we had just a few large ones or one large one and many small  
14 ones. I'd say that large one is way out of base.

15 So the statement of worrying about the size of an  
16 association is important, I think, when you're dealing with  
17 one study. That's why we tried to deal with all the  
18 studies.

19 MR. FURR: In fact, Dr. Bayard, aren't the summary  
20 risk estimates from the meta-analysis performed on the  
21 country groups? Aren't those summary risk estimates  
22 statistically inconsistent?

23 DR. BAYARD: The pooled risk? Yes, if I  
24 understand your question correctly. What I think you're  
25 asking is, are the pooled estimates heterogeneous between

1 countries. Different between countries.

2 MR. FURR: Right.

3 DR. BAYARD: The answer is yes, and I think it's  
4 reasonable to assume, as Dr. Wells testified when you  
5 questioned him, that the reason for some of these  
6 differences would be because in Japan, for example, where  
7 the risks are high, it's because the women are typically  
8 nonsmokers and they historically, they got most of their  
9 exposure from their husbands. So the spousal risks would be  
10 higher. That similarly would be the case in Greece. Dr.  
11 Wells provided a reference which I believe was Garfinkel  
12 1981.

13 So the answer is yes. The risks between countries  
14 for the pooled ever versus never differ, and that doesn't,  
15 in my mind, detract from the findings. In fact I think it  
16 would add to them.

17 MR. FURR: In the EPA risk assessment, EPA offers  
18 a slightly different explanation, don't they?

19 DR. BAYARD: Please?

20 MR. FURR: Doesn't EPA state that the  
21 heterogeneity of the observed risk estimates among countries  
22 may reflect related lifestyle characteristics in different  
23 countries, that is confounding?

24 DR. BAYARD: Please, I'm sorry.

25 MR. FURR: Even before we find the page, don't you

1 agree that that's another explanation?

2 DR. BAYARD: I'd like to carry on this  
3 discussion...

4 MR. FURR: Okay, take a look at page 18 of the EPA  
5 risk assessment.

6 DR. BAYARD: No, I mean I'm happy, I was happy  
7 enough not to look at the pages if you wanted to. But if  
8 you want...

9 MR. FURR: Your lawyer didn't seem to be.

10 (Pause)

11 DR. BAYARD: Excuse me? Where on the page is  
12 this?

13 MR. FURR: 1-8.

14 DR. BAYARD: Yes. And are you referring to the  
15 middle paragraph on the page?

16 MR. FURR: Yes.

17 DR. BAYARD: Yes. And, in fact, one of the  
18 reasons I presented to you was the middle -- the third line  
19 of that paragraph starting with the sentence, "For example,  
20 the observed differences may reflect true differences in  
21 lung cancer rates for never smokers in ETS exposure levels  
22 from non-spousal sources." Well, that's the same as saying  
23 ETS exposure levels from spousal sources. It's just the  
24 complement of it. So if a woman in Greece gets most of her  
25 exposure from her husband and she doesn't socialize -- and

1 she socializes with other women who don't smoke, she's not  
2 going to get much exposure from these other women because in  
3 Greece in that time period women didn't smoke. So when  
4 these women socialized with them, they weren't exposed to  
5 ETS so they had a relatively clean background and that was  
6 the whole argument of what I was presenting this morning,  
7 that when you have a clean background you probably will show  
8 higher risks.

9 MR. FURR: Dr. Bayard, that's just -- that  
10 explanation that you just gave us is not supported by data,  
11 is it? That's really just speculation on your part, isn't  
12 it?

13 DR. BAYARD: The argument of clean background I  
14 think is supported but the idea of different lifestyles?

15 MR. FURR: That's correct.

16 DR. BAYARD: I would refer you to the article by  
17 Dr. Garfinkel.

18 MR. FURR: Let's move on. I want to ask you about  
19 what it means to be a weak versus a strong association.  
20 When Dr. Ford testified, he took the position that  
21 epidemiologic associations under 2.0 are generally  
22 considered to be weak. Do you agree?

23 DR. BAYARD: For any one association, yes.

24 MR. FURR: Why is it not weak when there's more  
25 than one association that you're looking at?

1 DR. BAYARD: Because it has the strength of  
2 studies. You've got many studies done different ways and  
3 with not the same potential confounders. You've got  
4 repeatability and you've got studies from different  
5 countries. So it seems to me, and I've made the argument  
6 throughout the report, that there is strength in numbers  
7 here, that when you get increases in different countries  
8 with different potential confounders, with different  
9 independent researchers, that consistency of increased risks  
10 are more important than whether or not they're 2 or 1.5.  
11 It's the consistency, that they are consistently increased  
12 in different countries.

13 MR. FURR: Well, earlier you stated that  
14 LaMarchand estimated that the confounding from beta carotene  
15 alone could produce a 20 percent elevation in increased  
16 risk, isn't that correct?

17 DR. BAYARD: I think that was a hypothetical  
18 calculation and I think we referred to that in chapter five,  
19 page 66, that that didn't make it true, it means that  
20 potentially -- it's a potential confounder and under some  
21 hypothetical calculations that he made, he said, well, it  
22 could account for about a 20 percent increase in risk.  
23 However, the reason I made the argument of -- I'm sorry, I  
24 want to take that part of the sentence back. The increased  
25 risks in the highest exposure group would dismiss the

1 argument of a confounder of, say, something like diet  
2 because the increased risks were in the 80 to 100 percent  
3 range on average in the highest exposure group, so it's hard  
4 to imagine how diet could account for those increased risks  
5 in the highest exposure group.

6 MR. FURR: Dr. Bayard, did the EPA obtain any of  
7 the raw data for any of the epidemiologic studies?

8 DR. BAYARD: Yes.

9 MR. FURR: What studies?

10 DR. BAYARD: I don't know for sure because I did  
11 not attempt to obtain the raw data myself. That was done by  
12 Dr. Brown and Dr. Wells.

13 MR. FURR: Could you provide -- earlier you said  
14 that EPA's policy is to share data that it obtains and uses  
15 in its analyses, so could you provide as a post-hearing  
16 comment the data from the raw studies the EPA obtained?

17 DR. BAYARD: I will discuss that with my lawyers  
18 and if we used those data in the assessment, I would like to  
19 share those data. The decision is not mine because I don't  
20 have the data.

21 MR. FURR: But as a matter of scientific  
22 principle, you'd like to share the data.

23 DR. BAYARD: If -- I mean, as a matter of  
24 scientific principle, I assume you want the data for the  
25 ultimate purpose of deciding whether or not there's a health

1 effect and if one wants to decide there's a health effect,  
2 they want to decide it probably because they want to protect  
3 the health of the public and that seems to make sense. And  
4 if one wants to protect the health of the public, then one  
5 wants to share all the data on health effects. So I expect  
6 that everyone would want to share data on health effects,  
7 including the tobacco companies when they do their  
8 scientific --

9 MR. FURR: Your Honor --

10 JUDGE VITTON: Okay.

11 MR. FURR: May I inquire of EPA's counsel whether  
12 they have any objections to putting that data in the record?

13 MR. SHEEHAN: Your Honor, we would take it under  
14 advisement and get back to you on that.

15 JUDGE VITTON: All right.

16 MR. FURR: Dr. Bayard, for the data sets that EPA  
17 obtained, do you know whether they did any analyses of the  
18 high exposure groups?

19 DR. BAYARD: For every -- I'm trying to answer  
20 that question. We tried to do highest exposure group  
21 analysis whenever we could. I think we tried to get if not  
22 the raw data certainly the summary data for the highest  
23 exposure groups, the mid level exposure and all the exposure  
24 groups which would have helped us do an unadjusted analysis  
25 with the exception of smoker status misclassification, in

1 which case all the summary data are included in the report.  
2 I don't know whether or not we have additional raw data that  
3 is not in the publications.

4 MR. FURR: Do you know whether analyses were  
5 performed on the high exposure groups of that raw data,  
6 other than what's reported in the report?

7 DR. BAYARD: Any analyses that were performed on  
8 the highest exposure data other than what was -- no.

9 MR. FURR: No, you don't know?

10 DR. BAYARD: That's the answer. I don't know.  
11 You said do I know and I said, no, I don't know. I assume,  
12 knowing Dr. Brown, that if any analyses were done on the  
13 highest exposure data they would be included in the report.

14 MR. FURR: One moment, Your Honor.

15 (Pause)

16 MR. FURR: Dr. Bayard, isn't it EPA's position  
17 that associations under 2.0 for multi-factorial diseases are  
18 untrustworthy as far as inferring causation?

19 DR. BAYARD: It's certainly not our position.

20 MR. FURR: It's not EPA's position?

21 DR. BAYARD: It's certainly not the position with  
22 respect to the environmental tobacco smoke report.

23 MR. FURR: Depends on what you're looking at?

24 DR. BAYARD: That's a good question. Thank you.  
25 It depends on what -- it is one of the factors that does

1 into determining causality. It is not the sole factor in  
2 determining causality and we've been through that before.

3 MR. FURR: But in general, associations of under  
4 2.0 are less trustworthy for drawing causal inferences.

5 DR. BAYARD: On one study. That's why we don't  
6 look at one study. We keep -- I want to keep bringing you  
7 back, I want to bring you back to the exposure response  
8 information, I want to bring you back to the highest  
9 exposure group. I want to bring you back to the multiple  
10 analyses we did to look at potential confounders and biases.  
11 So I agree with you, if you're looking at one study you  
12 can't make one conclusion. Furthermore, you also have to  
13 look at your supporting data, whether or not there's any  
14 biological plausibility, for example. That's it.

15 MR. FURR: Dr. Bayard, are you familiar with an  
16 EPA health assessment document for 2,3,7,8-  
17 tetracholorodibenzoperodioxin?

18 DR. BAYARD: Yes.

19 MR. FURR: You are familiar with that document.

20 DR. BAYARD: I am familiar with several  
21 health assessment documents on 2,3,7,8-  
22 tetracholoroperodibenzodioxin. I've written parts of them  
23 myself.

24 MR. FURR: Are there more than one epidemiologic  
25 studies of dioxin?

1 DR. BAYARD: Yes. There are many, many  
2 epidemiologic studies of dioxin.

3 MR. FURR: I want to ask you to take a look at ta  
4 page from the EPA document whose title I just read.

5 DR. BAYARD: We can call it dioxin. That's  
6 usually what it's called.

7 (Pause)

8 MR. FURR: You can look at the language on the  
9 bottom of page 7-241, if you would. Were you involved with  
10 this document, by the way?

11 DR. BAYARD: Yes.

12 MR. FURR: And this document evaluated more than  
13 one epidemiologic study, didn't it?

14 DR. BAYARD: Yes.

15 MR. FURR: Let's take a look at the highlighted  
16 language on the bottom of 7-241. Tell me if I read this  
17 correctly, please. "In terms of the magnitude or strength  
18 of the association, this criterion refers to the degree to  
19 which the measure of association, e.g., the odds ratio or  
20 relative risks, exceed the null value of one. The stronger  
21 the association between exposure and effect the more  
22 convincing is the argument for causation. There is no  
23 definite cut point to numerically define a meaningful  
24 measure of association. Other factors such as the  
25 prevalence of the exposure in the population affect the

1 significance of the measure. Because so many adverse health  
2 effects are multi-factorial in etiology, a general rule of  
3 thumb is a relative risk less than two renders a  
4 cause-effect relationship less likely." Did I read that  
5 correctly?

6 DR. BAYARD: I think so.

7 MR. FURR: And that was a statement in an EPA  
8 document reviewing more than on epidemiologic study, wasn't  
9 it?

10 DR. BAYARD: The answer is yes. I also want to  
11 add, however, that I don't know whether this refers to the  
12 cumulative review of all the epidemiologic studies. I see  
13 it as a statement exclusive of the rest of the reviews and  
14 so not only is it a statement which is exclusive of the  
15 reviews but you haven't quoted from the summary or the  
16 conclusion so it's much harder to put this statement into  
17 perspective.

18 MR. FURR: I'm sure it is. Dr. Bayard, no  
19 other --

20 MR. SHEEHAN: Your Honor, for the record, I would  
21 like to point out that this is a review draft, not a final  
22 document.

23 JUDGE VITTON: It will be put in the record.

24 MR. FURR: No other substance classified as a  
25 Group A carcinogen by EPA has been done so based on a

1 relative risk of less than 2.0, has it?

2 DR. BAYARD: I think the answer is yes and I think  
3 when we look at direct blue, direct black and direct brown,  
4 those are substances that are benzidine dye based. But I  
5 don't think there are any epidemiology studies at all on  
6 direct black, direct blue and direct brown.

7 MR. FURR: So no other substance has been  
8 classified as a Group A carcinogen based on epidemiologic  
9 studies of under 2.0? Is that correct?

10 DR. BAYARD: Let me look at that. I'm just going  
11 to refresh my memory on that.

12 (Pause)

13 MR. SHEEHAN: Your Honor, while he's refreshing  
14 his memory, I do want the record to reflect that this  
15 document says on it that this is a preliminary draft that  
16 has not been formally released by the EPA.

17 MR. FURR: That point has already been made.

18 MR. SHEEHAN: And should not at this stage be  
19 construed to represent agency policy. It has been  
20 circulated for comment on its technical accuracy and policy  
21 implications.

22 JUDGE VITTON: Okay. But the entire document  
23 with that statement will be made part of the record, so it  
24 will be in there.

25 Mr. Furr, I think you've got about three minutes.

1 MR. FURR: Three minutes?

2 JUDGE VITTON: Yes.

3 Dr. Bayard?

4 DR. BAYARD: I'm sorry. You're probably right,  
5 although I would have to go look at all the studies.

6 However, I want to point --

7 MR. FURR: I'll withdraw that question.

8 I only have three minutes? Is that what I really  
9 have?

10 DR. BAYARD: Your Honor --

11 JUDGE VITTON: I'm sorry, I just got lost here.

12 DR. BAYARD: Can I just finish my answer?

13 JUDGE VITTON: No, wait a minute. I just got  
14 lost here a second.

15 DR. BAYARD: But I was interrupted.

16 JUDGE VITTON: And now you're interrupting me.

17 Mr. Furr, you have three minutes. We started 57  
18 minutes ago.

19 MR. FURR: Dr. Bayard, isn't it true that you have  
20 on more than one occasion made overtures to employees of  
21 tobacco companies with respect to the possibility of  
22 obtaining employment in either the tobacco industry or a  
23 related industry?

24 MR. SHEEHAN: Objection, Your Honor. This is way  
25 beyond the scope of this hearing.

1 JUDGE VITTON: Repeat that question again?

2 MR. FURR: Sure. It's very interesting. Isn't it  
3 true that on more than one occasion, Dr. Bayard, you have  
4 made overtures to employees of tobacco companies with  
5 respect to the possibility of obtaining employment in either  
6 the tobacco industry or a related industry?

7 MR. SHEEHAN: Your Honor --

8 DR. BAYARD: Absolutely not. I love my job.

9 JUDGE VITTON: All right. Mr. Furr --

10 MR. FURR: Your Honor, I think the decision makers  
11 might be interested in hearing just how firm Dr. Bayard's  
12 alliances are.

13 DR. BAYARD: For me --

14 JUDGE VITTON: I'm going to leave this up to  
15 Dr. Bayard.

16 Dr. Bayard --

17 DR. BAYARD: If I may --

18 JUDGE VITTON: Dr. Bayard, just --

19 DR. BAYARD: I would like to state for the record,  
20 because I don't think that's a very nice question, not that  
21 I'm disparaging working for the tobacco companies, I don't  
22 mean to do that --

23 JUDGE VITTON: Look. Why don't you consult with  
24 your lawyer there a second?

25 I'll give you another minute.

1 (Pause)

2 MR. FURR: Isn't it true, Dr. Bayard, that you  
3 have suggested to tobacco company employees that the most  
4 obvious solution to the problems created by the EPA's risk  
5 assessment would be to hire you away from the Environmental  
6 Protection Agency?

7 MR. SHEEHAN: He's not going to answer that  
8 question, Your Honor.

9 DR. BAYARD: It's character assassination. I'd  
10 really like to answer that question.

11 MR. SHEEHAN: No.

12 JUDGE VITTON: Okay. Let's move on, Mr. Furr.

13 MR. FURR: Dr. Bayard, haven't you in fact  
14 suggested that perhaps the most discrete approach would be  
15 for the tobacco industry to arrange to have you hired by a  
16 supplier for the industry?

17 MR. SHEEHAN: Looks like our time's up, Your  
18 Honor.

19 JUDGE VITTON: Mr. Furr, do you want to ask him a  
20 substantive question before we finish up here?

21 MR. FURR: How much time do I have?

22 JUDGE VITTON: You have one minute.

23 MR. FURR: Do you have slide 45? Dr. Bayard,  
24 could you take a look at slide 45 in your evidence today?  
25 Do you have that?

1 DR. BAYARD: Yes.

2 MR. FURR: Doesn't that slide show -- let me back  
3 up. This is a slide depicting data collected by Riboli et  
4 al. in 13 cities, did you say?

5 DR. BAYARD: Yes.

6 MR. FURR: A very large study?

7 DR. BAYARD: Thirteen hundred and sixty-nine  
8 women.

9 MR. FURR: Excuse me?

10 DR. BAYARD: Thirteen hundred and sixty-nine  
11 women.

12 MR. FURR: It's been a very expensive study,  
13 hasn't it?

14 DR. BAYARD: I have no idea.

15 MR. FURR: Don't these bar graphs show that the  
16 cotinine levels generated by home exposure are approximately  
17 twice those generated by workplace exposure?

18 (Pause)

19 DR. BAYARD: I think that's right. It's a little  
20 late and so I'm having a little trouble but I think it's  
21 right and I think -- excuse me --

22 MR. FURR: These go to --

23 DR. BAYARD: I'm sorry, just let me finish. I  
24 think that's consistent with my argument before that the  
25 home exposure levels and workplace exposure levels tend to

1 be about the same but since one spends about twice as much  
2 time at home as they do at work, you would expect that the  
3 cotinine levels from home are probably going to be about  
4 twice as much as the cotinine levels from work. I think  
5 it's fairly consistent. I would point out, though, that we  
6 tried to get more data than just these summary data from  
7 Dr. Riboli. He just didn't have the means of getting this  
8 to us. This is a breakdown -- this is a summary of 13  
9 separate cities and it doesn't reflect the -- it may or may  
10 not reflect the U.S. so the answer is that, yes.

11 MR. FURR: You're pointing out that these cotinine  
12 levels that were measured in the subjects really reflect the  
13 combination of not only the concentrations of ETS that they  
14 were exposed to but also it's a measure of the time activity  
15 patterns that they spent in those environments.

16 DR. BAYARD: That's why biomarkers, I think, would  
17 be a good measure.

18 MR. FURR: So based on --

19 JUDGE VITTON: That's it, Mr. Furr. We have to  
20 cut you off.

21 MR. FURR: May I ask one more?

22 JUDGE VITTON: All right. Your last question.  
23 Let's keep the answer short.

24 MR. FURR: Based on a linear dose response model,  
25 then --

1 JUDGE VITTON: Finish up.

2 MR. FURR: -- it would be your opinion that the  
3 home exposure is likely to pose twice the risk as the  
4 workplace exposure.

5 DR. BAYARD: If this is a representative sample.

6 MR. FURR: Thank you.

7 JUDGE VITTON: Thank you very much. Thank you,  
8 Mr. Furr.

9 Let's see. Mr. Meyers, do you have any questions  
10 at all?

11 MR. MEYERS: I would be willing to defer and let  
12 OSHA go first if they would wish.

13 JUDGE VITTON: Ms. Sherman, do you have any  
14 questions?

15 MS. SHERMAN: Yes.

16 Good afternoon.

17 DR. BAYARD: Good afternoon, Ms. Sherman.

18 MS. SHERMAN: I believe that much earlier today,  
19 much, much earlier today, you and Mr. Furr discussed a work  
20 called "Choices in Risk Assessment"?

21 DR. BAYARD: Yes.

22 MS. SHERMAN: That's true?

23 DR. BAYARD: Yes.

24 MS. SHERMAN: Was this report written by a  
25 subcontractor to Sandia Laboratories?

1 DR. BAYARD: That's my understanding.

2 MS. SHERMAN: Who was that? Do you know?

3 DR. BAYARD: I know the project officer was Steve  
4 Malloy but the authors are in the book but I don't know the  
5 authors. Go ahead.

6 MS. SHERMAN: You say the project officer.

7 DR. BAYARD: Yes.

8 MS. SHERMAN: The person in charge of writing the  
9 study, if you will?

10 DR. BAYARD: Of getting it written. I don't know  
11 if he's in charge of writing it.

12 MS. SHERMAN: Have you ever spoken with Mr. Malloy  
13 concerning environmental tobacco smoke risk assessment  
14 issues?

15 DR. BAYARD: Yes, I have.

16 MS. SHERMAN: What was the content of your  
17 conversation with Mr. Malloy?

18 DR. BAYARD: Well, I think first I'd like to say  
19 that I'm on record as having spoken to Mr. Malloy at a  
20 speech he gave, it must have been about a month ago, stating  
21 that he lobbied me for two years on behalf of the tobacco  
22 companies.

23 MS. SHERMAN: And that was your only -- so that  
24 was not your only conversation with Mr. Malloy?

25 DR. BAYARD: No. No. We used to have

1 extensive -- not extensive but a lot of discussions on risk  
2 assessment issues while we were writing the report,  
3 somewhere in the --

4 MS. SHERMAN: While you were writing the EPA  
5 report.

6 DR. BAYARD: Yes. Somewhere in the timeframe  
7 of -- I'd say 1989 to 1992, 1990 to '92.

8 MS. SHERMAN: And who did Mr. Malloy represent at  
9 the time that you were having these discussions with him  
10 about the EPA report?

11 DR. BAYARD: Mr. Malloy worked for Mr. Tosey, who  
12 has a lot of companies under the name Multi National  
13 Business Corporation or several other companies.

14 MS. SHERMAN: Have you reviewed the report called  
15 "Choices in Risk Assessment" yourself?

16 DR. BAYARD: I have certainly reviewed the  
17 environmental tobacco smoke portion of it.

18 MS. SHERMAN: And are you familiar with chapter  
19 ten? Is that the environmental tobacco smoke section?

20 DR. BAYARD: I think so but I don't have -- I have  
21 it here somewhere.

22 MS. SHERMAN: Do you believe it is an objective  
23 review of the risk assessment in OSHA's proposal?

24 DR. BAYARD: I believe it is not an objective  
25 review. I've stated so. And the reasons I believe it's not

1 an objective review, I stated this to Mr. Malloy at the  
2 seminar that he gave to the Environmental Protection Agency,  
3 the reasons I believe that it is not an objective review is  
4 because nowhere in his review does he mention either the EPA  
5 environmental tobacco smoke report, the National Research  
6 Council Report, the Surgeon General's report or the Science  
7 Advisory Board review of the EPA report and this wouldn't be  
8 disturbing except in every other chapter he mentions the EPA  
9 and the SAB many times but this is notably absent from his  
10 analysis of the OSHA report. Of the OSHA risk assessment.

11 MS. SHERMAN: Based on your experience with risk  
12 assessment, how much weight do you give the human data in  
13 conducting hazard identification on a substance?

14 DR. BAYARD: We prefer human data whenever we can.  
15 Human data are always, we feel, better than -- not always  
16 but we feel they're usually better than animal data because  
17 we don't have to have the uncertainty of extrapolating from  
18 the animals to humans. However, there are times when human  
19 data just aren't adequate for an analysis.

20 MS. SHERMAN: You don't have to worry about  
21 scaling factors or anything else when you're dealing with  
22 most human data, is that it?

23 DR. BAYARD: There's no animal to human scaling  
24 factors but there certainly can be extrapolation factors  
25 from high to low doses.

1 MS. SHERMAN: In the case of environmental tobacco  
2 smoke, is there evidence that you believe is better than the  
3 human data in the record?

4 DR. BAYARD: I feel today that I've presented the  
5 best data to you that I know. Especially in my Direct  
6 testimony, I feel that the EPA report contained the best  
7 information that we had at the time. I feel the Fontham '94  
8 study is the best study. So I think OSHA's on the right  
9 track by using the Fontham data.

10 MS. SHERMAN: So as to lung cancer at the very  
11 least, the human data represented by the Fontham data is the  
12 best data in your opinion?

13 DR. BAYARD: I thought they were the best for  
14 spousal, and I think they're the best for workplace  
15 exposures, too.

16 MS. SHERMAN: Is there any reason to expect that  
17 the effect of a carcinogen would be any different in  
18 different micro environments? That is in the home, in a  
19 method of transportation in the workplace?

20 DR. BAYARD: I think I've talked about that, but  
21 Mr. Furr did bring up a point that said it's not only  
22 concentration levels, but concentration levels times time.  
23 I don't expect that for... I'm sorry, I'm getting far  
24 afield.

25 In general, I think if I'm interpreting your

1 question correctly, my response is that if we're seeing it  
2 in the home there's no reason to expect that we wouldn't be  
3 seeing it in other places where there are comparable  
4 exposure levels.

5 MS. SHERMAN: OSHA did not adjust for smoker  
6 misclassification in its lung cancer risk assessment. Do  
7 you think that OSHA should have adjusted for smoker  
8 misclassification?

9 DR. BAYARD: With respect to the Fontham study,  
10 no. Because you're looking at the... I'm sorry, I want to  
11 think about that for a minute.

12 You're talking about smoker misclassification  
13 status.

14 MS. SHERMAN: Yes.

15 DR. BAYARD: I think smoker misclassification  
16 status is not a factor when you're looking at workplace  
17 exposure studies. The answer would be no.

18 MS. SHERMAN: I believe that OSHA took the  
19 position that Fontham adjusted for smoker misclassification  
20 to a certain extent in the study design. Would you agree?

21 DR. BAYARD: It wasn't an adjustment, it was a  
22 design. So what they did was eliminate the cases who had,  
23 they had any inkling would have former or current smokers,  
24 and they did that by means of a multiple series of  
25 questions, either going through the doctor's report or the

1 medical record first, and then going through I think two  
2 series of interviews, and finally on an analysis of their  
3 urinary cotinine to dismiss any potential smokers.

4 MS. SHERMAN: Have you read the Oak Ridge study?

5 DR. BAYARD: No. I've seen some of the tables of  
6 it, and I've read some of Dr. Jenkins' testimony. I've  
7 heard Dr. Jenkins give a presentation. I think I heard him  
8 give two presentations, but I haven't read the study.

9 MS. SHERMAN: Getting over for a minute to the  
10 Riboli study, isn't it true that the Riboli study could be  
11 criticized as containing non-representative samples?

12 DR. BAYARD: Non-representative of what?

13 MS. SHERMAN: Of the general population.

14 DR. BAYARD: Well, I think most studies could. I  
15 think that Dr. Riboli also got his sample from volunteers as  
16 did, for example, Dr. Guerin and Dr. Jenkins.

17 I don't know for sure, though. I would have to  
18 re-read the study.

19 Now having said that, I can say that we also used  
20 portions of the Riboli study in our own report. We used the  
21 data from Los Angeles and New Orleans, I think.

22 MS. SHERMAN: Thank you very much for your time.  
23 I don't have any further questions.

24 DR. BAYARD: Thank you.

25 JUDGE VITTON: Mr. Myers?

1 MR. MYERS: I have just one or two, Your Honor.

2 JUDGE VITTON: Okay.

3 (Pause)

4 MR. MYERS: Dr. Bayard, my name is Matthew Myers.  
5 You may have heard, I'm here representing a number of  
6 health-related organizations and victims of environmental  
7 tobacco smoke.

8 I'm a little confused about one thing that you  
9 said in the very, very last set of questions Mr. Furr asked  
10 you. I just want to make sure that I understand. It was  
11 questions about relative exposure and workplace versus home.

12 When I finished listening to the answer I became  
13 unclear as to whether or not based upon your review of all  
14 of the evidence it was your conclusion that given your  
15 analysis of those relative exposures whether there was  
16 adequate scientific data to include that exposure to  
17 environmental tobacco smoke in the workplace increased the  
18 risk of lung cancer.

19 DR. BAYARD: Of course.

20 It's not that this is an industrial chemical.  
21 Where there's exposure, there should be risk. It's as  
22 simple as that.

23 MR. MYERS: You commented that if you spend twice  
24 the time in the home you'd expect to see higher cotinine  
25 levels in the home than in the workplace.

1 DR. BAYARD: Huh uh.

2 MR. MYERS: How does that affect your assessment  
3 of the relative risk in the workplace and our ability to use  
4 the home data for making certain judgments?

5 DR. BAYARD: I have to make a small correction  
6 here. We're not talking about levels in the home,  
7 concentration levels in the home being twice as high. We're  
8 talking about cotinine levels in people exposed to spousal  
9 smoking being twice as high as people who are exposed to  
10 similar concentrations in the workplace. Probably because  
11 they're at home twice as long as they are in the workplace.

12 To extrapolate from one to another, a factor of  
13 two to a risk assessor is peanuts. So it's no problem for a  
14 risk assessor to extrapolate from the home to the workplace.

15 MR. MEYERS: So you weren't saying that the home  
16 exposure is twice as dangerous or increased the risks twice  
17 as much. I'm trying to understand. Can we still use that  
18 home data, in your professional opinion, in reaching  
19 conclusions about the impact of exposure in the workplace?

20 DR. BAYARD: The quantitative impact.

21 MR. MEYERS: That's right. Well, the risk,  
22 whether or not there in fact is an increased risk and  
23 whether it tells us something meaningful about that risk.

24 DR. BAYARD: Yes. I thought I explained that.  
25 And both qualitatively and quantitatively. Certainly

1 qualitatively there should be no question that if  
2 environmental tobacco smoke increases the risk from spousal  
3 exposure, it should at similar levels anywhere, whether  
4 you're in a train or whether you're just standing next to  
5 someone and he's blowing smoke on you, it's going to  
6 increase your risk. Now, quantitatively, I have maintained  
7 that you can make an extrapolation from the home to the  
8 workplace based on time/activity patterns and established  
9 risks from home. However, they require certain adjustments.  
10 I don't think it's as simple as just saying I'll take the  
11 home exposure levels and I'll go to the workplace and I'll  
12 take the workplace exposure levels and if the home exposure  
13 levels are the same as the workplace it's just a simple  
14 factor of two because you're home twice as long.

15           What we've got is a measure of spousal exposure  
16 and spousal exposure means you have to consider not only the  
17 home where the spouse is, you have to consider riding in the  
18 car, you have to consider when you go out to dinner with  
19 your spouse and to derive a whole time/activity pattern to  
20 establish levels that one would derive from a spouse versus  
21 levels that one would derive from passive smoking in the  
22 workplace.

23           On the other hand, I also discussed that OSHA  
24 seemed to be certainly well within its bounds by considering  
25 some workplace studies and specifically if you could find

1 other studies of workplace exposure as good as the Fontham  
2 study, then look at that. It may mean contacting Fontham  
3 and getting the proper control group, which I also  
4 discussed, but I see no reason why either approach shouldn't  
5 be valid.

6 MR. MEYERS: So is it fair to conclude, and this  
7 is really my last question, then, that your conclusion about  
8 what we're seeing in relative cotinine levels versus home  
9 exposure versus workplace exposure in your professional  
10 opinion has not undermined OSHA's conclusion that workplace  
11 exposure does result in a meaningful increase in lung cancer  
12 risk?

13 DR. BAYARD: I think I've answered the question  
14 and that is where there is exposure there is going to be  
15 risk. Some of the problems that I see with the cotinine  
16 levels now, they're just not derived from representative  
17 samples, samples which are representative of true workplace  
18 exposures. And since workplace exposures vary so much  
19 anyway, you may even have to be site specific or type of  
20 site specific. You may have to say, well, I'll go examine  
21 my restaurants and see what the risks are in restaurants  
22 versus offices. And you may find that the risks are low  
23 enough in offices that you don't have to worry about it.  
24 And you may find that the risks in restaurants are so high  
25 that you would feel compelled to put out some sort of --

1 well, not you but OSHA would feel compelled --

2 MR. MEYERS: The bottom line is the data from the  
3 Riboli study in terms of exposure, duration of exposures, in  
4 your professional opinion, doesn't weaken OSHA's conclusion  
5 about the relative risk of the ETS in the workplace.

6 DR. BAYARD: No.

7 JUDGE VITTON: Thank you, Mr. Meyers.

8 MR. WEINBERG: Your Honor, one burning question.  
9 Might I ask it?

10 JUDGE VITTON: I was going to ask anyway,  
11 Mr. Weinberg, if you had that one burning question. Go  
12 ahead. Come on.

13 MR. WEINBERG: I probably could be heard from  
14 here --

15 JUDGE VITTON: Why don't you come on down here  
16 just to be sure, okay? I think I owe you. I've cut you off  
17 a number of times during this proceeding.

18 MR. WEINBERG: Dr. Bayard, my name is Myron  
19 Weinberg. I represent docket number 103.

20 This past few minutes you've been discussion  
21 extrapolation from one site to another, the ability.

22 DR. BAYARD: Yes.

23 MR. WEINBERG: Doesn't that total extrapolation  
24 depend on the fact that the material to which you are  
25 exposed in one place is the same as the material to which

1 you're exposed in another place?

2 For example, if environmental tobacco smoke is  
3 aged differently in the workplace under ultraviolet light  
4 than it would be aged in the home under incandescent light,  
5 wouldn't you change your mind as to whether or not you could  
6 simply extrapolate from the home to the workplace?

7 The basic question, doesn't simple extrapolation  
8 require that you're talking about the same substance in all  
9 sites to which you are extrapolating?

10 DR. BAYARD: I think in a way that it's a  
11 hypothetical question and I think we would have to kind of  
12 see some data before we could make that decision.

13 Also, I need to bring up an interesting point,  
14 though. For example, if you have environmental tobacco  
15 smoke in the workplace and it adds to the effect of other  
16 chemicals, then one might be a little bit more concerned  
17 about it in the workplace. One would have to model it, of  
18 course.

19 MR. WEINBERG: And then you'd have to model  
20 whether it detracts from the effects of the chemicals in the  
21 workplace as well.

22 DR. BAYARD: Well, that's what modeling is. But  
23 one would also have to have some data to indicate that it  
24 would detract from those chemicals.

25 MR. WEINBERG: An important thing, you do have to

1 look at the substance to which the exposure is in order to  
2 translate from one location to another.

3 DR. BAYARD: You have to but you have to have some  
4 evidence for a reason to look for it. If one could  
5 establish a database that aging, for example, got rid of all  
6 the formaldehyde or that certain irritants and other  
7 carcinogens were changed from one location to another, I  
8 mean, it sure seems a reasonable approach. However, it sure  
9 seems to me difficult to establish that database.

10 MR. WEINBERG: On the other hand, some people  
11 translate from mainstream to environmental tobacco smoke and  
12 it's clear that vinyl chloride which is a known carcinogen  
13 has never been demonstrated in environmental tobacco smoke.  
14 It's not there. There are changes which occur under various  
15 aging. I'm just making a point that when you do  
16 translations, you have to look at the substances to which  
17 people are exposed as you translate from one site to  
18 another. I just wanted to understand how you thought about  
19 that.

20 DR. BAYARD: Thank you.

21 JUDGE VITTON: Thank you, Mr. Weinberg.

22 MR. WEINBERG: Thank you.

23 JUDGE VITTON: That completes our examination and  
24 our testimony for today.

25 Thank you, Dr. Bayard. We appreciate your time

1 today.

2 DR. BAYARD: Thank you, Judge.

3 (Whereupon, the witness was excused.)

4 JUDGE VITTON: We will resume Monday morning at  
5 9:30.

6 MR. FURR: Your Honor --

7 JUDGE VITTON: Yes, Mr. Furr?

8 MR. FURR: Before we go off the record, we need  
9 the exhibits that were identified.

10 JUDGE VITTON: Let's go off the record a second.

11 (Whereupon, a brief recess was taken.)

12 JUDGE VITTON: Nine-thirty Monday morning.

13 (Whereupon, at 6:10 p.m., the hearing was  
14 adjourned, to be reconvened Monday, March 13, 1995 at  
15 9:30 a.m.)

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TITLE: OSHA  
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LOCATION: Washington, D.C.

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