IN THE CIRCUIT COURT OF HARRISON COUNTY
WEST VIRGINIA

LENORA PERRINE, CAROLYN HOLBERT, WAUNONA MESSINGER, REBECCAH MORLOCK, ANTHONY BEEZEL, MARY ELLEN MONTGOMERY, MARY LUZADER, TRUMAN R. DESIST, LARRY BEEZEL, and JOSEPH BRADSHAW, individuals residing in West Virginia, on behalf of themselves and all others similarly situated,

Plaintiffs,

vs. CIVIL ACTION
NO. 04-C-296-2

E.I. DU PONT DE NEMOURS AND COMPANY, a Delaware corporation doing business in West Virginia, etal,

Defendants.

VOLUME XVII

OCTOBER 3, 2007

HARRISON COUNTY COURTHOUSE

Proceedings had in the above-entitled action before the Honorable Thomas S. Bedell, and a jury, in the Harrison County Courthouse, Clarksburg, West Virginia, on the 3rd day of October, 2007.

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I N D E X

P R O C E E D I N G S

THE COURT: Be seated, please. Let's go on the record. Comes on for proceedings at the present time a matter encaptioned Lenora Perrine, et al versus E.I. DuPont DeNemours & Company, et al, Defendants. The matter bears Case No. 04-C-296-2.

The Court would note that today is Wednesday, the 3rd day of October, 2007. These matters come on by way of further proceedings of the jury proceedings which recessed at the close of the day on the 2nd day of October, 2007.

Will counsel be so kind as to note their appearance for the record and further note the presence or the absence of their respective clients or others whose attendance they care to note.

Mr. Taylor?

MR. TAYLOR: Thank you, your Honor. With me today is Joseph Lane, Gary Rich, Angela Mason, our client, Lenora Perrine, and Brian Barr.

THE COURT: Okay. Mr. Hall?

MR. HALL: Good morning, your Honor, Jeff Hall for DuPont. With me is Brian Prestes, also

Shawn Gallagher, who will be handling the examination of our first witness, David Thomas and Mr. Yalvigi.

THE COURT: Counsel, let me note that appropriate, as far as the timing goes, that the Defendant DuPont moved for judgment as a matter of law at the close of the plaintiffs' case yesterday on the medical monitoring phase and further has submitted their written motion for judgment as a matter of law on medical monitoring or, in the alternative, to decertify the class.

Let me ask counsel who will again address that to approach the bench and we'll argue it at side bar.

(Counsel approached the bench and the following proceedings were had out of the hearing of the audience:)

MR. PRESTES: Good morning, Judge, Brian Prestes on behalf of DuPont. DuPont moves for judgment as a matter of law, or in the alternative, to decertify the class.

There is no sufficient evidentiary basis for the jury to find for the plaintiffs on a number of the critical elements of Bower. I'd

like to focus on three points, and with respect to the others, rest on the paper that we have filed this morning.

Under the first element of Bower, the plaintiff must show significant exposure. Bower makes clear that a significant exposure is what is required, not mere exposure. Doctor Werntz yesterday testified that he merely assumed exposure. He did no tests.

In fact, he testified he had never met a single member of the class and didn't even know how many class representatives there were. Doctor Werntz testified that he relied on Doctor Brown, who is a soil scientist, not a medical doctor.

Doctor Brown is not an expert in cancer causation, and Doctor Brown has no basis for opining on the levels of exposure that are sufficient to justify or necessitate medical monitoring.

With respect to Doctor Brown's testimony and the insufficiency of that testimony, we'd rely and incorporate our Phase 1 judgment as a matter of law briefing.

But just to provide two specific

examples, Doctor Brown conducted no risk assessment on lead, and his contour maps on lead show extremely low levels of lead, below the levels that Doctor Brown himself identified as clean.

Moreover, the only tests of presence of lead in the bodies of anyone then introduced in this case -- the ATSDR tests show no significant exposure to lead. So on that point, we believe that DuPont is entitled to judgment as a matter of law.

Two other points quickly, Judge. The first is that we believe the plaintiffs have failed to show an increased risk of disease. That's Bower Element 4. The plaintiffs must show a significantly increased risk of disease caused by the exposure they've alleged.

Again on this point, Doctor Werntz, the plaintiffs' only Phase 2 witness, provided only nonspecific testimony, not testimony relating to anything having to do with this class.

Again he relied on Brown, and I'd incorporate the argument I just made about the insufficiency of Doctor Brown's testimony to

support the opinion that the plaintiffs are at an increased risk of disease, especially as it relates to lead for which Doctor Brown conducted no risk assessment whatsoever.

My final point relates to the fifth element of Bower, which is that the plaintiffs must show medical testing is reasonably necessary. And the way Bower has defined that is reasonably necessary testing is testing that a qualified physician would prescribe.

First off, many of the diseases that Doctor Werntz described in his testimony yesterday are diseases for which Doctor Werntz himself concedes medical monitoring is not appropriate.

Doctor Werntz went through a laundry list of perhaps 20 diseases, but in the end, with the assistance of one of plaintiffs' demonstratives, testified there were only five cancers and three noncancer diseases, and so DuPont submits that to the extent any of those other diseases are in play, they cannot form the basis of a medical monitoring claim.

They may go to show - if plaintiffs' evidence is taken as correct - that lead or

arsenic or cadmium have the potential to be harmful, but they certainly cannot be the basis of a medical monitoring program.

Two other quick examples. First, Doctor Werntz testified -- we think Doctor Werntz's testimony shows that CT scans are not reasonably necessary. He testified that those scans are not recommended by the United States Preventive Services Task Force, which Doctor Werntz conceded himself is the consensus.

Although Doctor Werntz himself does recommend those CT scans, the bare unsupported opinion of Doctor Werntz, in the face of his own testimony that the consensus of qualified physicians does not recommend those tests, we believe entitles DuPont to judgment as a matter of law, at least as it relates to the CT scan element of plaintiffs' proposed medical monitoring plan.

Indeed -- now, Doctor Werntz relies on only a single article for his CT scan recommendation. It is an article from the Journal of the American Medical Association, which itself concluded - as I believe was shown on cross examination - that these CT scans are an

experimental procedure based on a, quote, "uncorroborated premise."

And finally, one other specific example, which is kidney and stomach cancer screening. Again, Doctor Werntz conceded that, quote, "No organization recommends screening for kidney cancer and stomach cancer." And to the extent that Doctor Werntz's opinion itself is not supported by any facts and to the extent that it's contradicted by the vast weight of science -- of the medical consensus on Doctor Werntz's own view, we believe that DuPont is entitled to a judgment as a matter of law on those points.

With that, your Honor, and with respect to the other points in our paper, DuPont is content to rest on its papers. Thank you.

THE COURT: Ms. Mason?

MS. MASON: The plaintiffs have established every one of the elements that is required in Bower through Doctor Werntz's testimony. The defendant's motion is based primarily on trying to relitigate Doctor Brown's conclusion and the jury's acceptance of those conclusions concerning his risk assessment.

It is true that Doctor Brown did not perform a risk assessment on lead itself; however, he did measure lead levels and -- not in humans, and Doctor Werntz testified that lead has additive effect, and because of that additive effect, it is reasonably necessary to consider that a significant risk, and increased -- and increasing the risk of contracting a serious latent disease.

Doctor Werntz, in addition to relying on Doctor Brown, performed his own qualitative analysis that he testified to yesterday. Doctor Werntz established the significant risk of 1 in 100,000, and confirmed that Doctor Brown's risk assessment is three times that, I believe, in the furthest-away zone, Zone 3.

So it exceeds Doctor Werntz's determinations -- actually exceed the levels that even Doctor Brown found in his risk assessment. In other words, Doctor Werntz reviewed it himself and made his own conclusions about what constitutes significant risk.

It would be in -- we -- Doctor Werntz presented testimony on various diseases that can be caused by all three of the elements at issue.

Those three diseases -- goes to show that they are hazardous, that he restricted his medical monitoring to what he would consider to be, as a practicing physician in West Virginia, reasonably necessary to screen for and practical as well.

Are there any other issues that you'd want us to specifically address?

THE COURT: Counsel, anything in conclusion?

MR. PRESTES: Yes, your Honor, just three quick points. First of all, Ms. Mason has suggested that we are trying to relitigate the jury's acceptance of Doctor Brown's conclusions.

We don't think that's accurate. Though it is true that in the Phase 1 verdict, the jury did conclude that there had been exposure, as I previously suggested, the relevant question here is whether there has been significant exposure.

In addition, the Phase 1 verdict found exposure to persons or property, and thus there is no Phase 1 verdict making -- accepting the conclusion that there has been significant exposure to persons.

Moreover, to the extent there is any such

conclusion, we believe that there is no sufficient evidence to support that conclusion.

Second, I'm somewhat confused by counsel's reference to Doctor Brown's risk assessment and how they provide a basis for Doctor Werntz's opinion, because as is clear from the record, Doctor Brown conducted no risk assessment on lead, and hence, there is no basis in Doctor Brown's testimony or in the evidence that has come in to this case so far for Doctor Werntz to testify that any class members have been exposed to a risk relating to the exposure to lead that would justify medical monitoring.

Finally, it sounds like we are in agreement on the issue relating to the laundry list of diseases that Doctor Werntz testified to and the fact that although he testified to, say, 20 or more diseases; only a small number of those five cancers and three noncancers would even conceivably form the basis of the medical monitoring program that the plaintiffs urge.

THE COURT: Counsel, let me indicate although counsel for DuPont makes a compelling argument and if -- if his client was smart,

instead of Doctor Hall, that counsel ought to be making the closing summation.

Most, if not everything, counsel's introduced really goes to the weight of the evidence, which is really an issue for the trier of fact.

The Court cannot say as a matter of law that the plaintiffs failed in any one of the Bowers factors or in any of the subarguments that have been made. Seems to me that those are -- they may very well be compelling arguments, but one that must be offered to the triers of fact, and the Court - even accepting those in a light most favorable - cannot say as a matter of law that the plaintiffs failed to meet its burden at this point.

So for those reasons and all other reasons appearing of record, the motion for judgment as a matter of law - or in the alternative, to decertify class - will be denied and the defendant's exceptions preserved.

MS. MASON: Thank you, your Honor.

MR. PRESTES: Thank you.

(Counsel returned to counsel table and

the proceedings continued in the presence and hearing of the audience as follows:)

THE COURT: We have about ten minutes, counsel. They're deliberating lunch.

(A recess was taken after which the proceedings continued as follows:)

THE COURT: Be seated, please. Mr. Bailiff, you may return the jury to its box, sir, please.

(The jury returned to the courtroom.)

THE COURT: You may be seated. Good morning, ladies and gentlemen of the jury. Let me note the appearance of all 11 of our jurors who are again present before the Court and the parties and their respective counsel.

I believe the plaintiff now having rested, it's the Defendant DuPont's presentation of evidence. Mr. Hall, DuPont may call its first witness, sir.

MR. HALL: Yes, your Honor, DuPont calls David Garabrant. Mr. Gallagher will handle the examination, your Honor.

THE COURT: If you'll come forward, sir, and be sworn, please.

(The witness was sworn.)
THE COURT: You may have a seat up here in the witness stand, please.
D A V I D G A R A B R A N T
was called as a witness by the Defendant, and having been first duly sworn, testified as follows:
DIRECT EXAMINATION
BY MR. GALLAGHER:
Q. Good morning. Would you tell us your name?
A. Yes, David Hay Garabrant.
Q. Are you a medical doctor?
A. Yes.
Q. What is your specialty?
A. Occupational and environmental medicine and also internal medicine.
Q. Do you study the human health effects of exposure to chemicals in the environment?
A. That's what I've spent my career doing.
Q. Have you been asked to provide expert opinions in this case concerning the medical monitoring program suggested by the plaintiffs' expert, Doctor Werntz?

A. Yes, I have.
Q. And in particular, have you been asked to consider whether Doctor Werntz's conclusions about the risk of cancer and other diseases are warranted in light of the evidence?
A. Yes.
Q. Before we get to your opinions on that, let's talk about your background. Could you describe for the jury your education?
A. Sure. I got my undergraduate degree, my college degree, in chemical engineering. I went to Tufts University outside of Boston, graduated in 1972. Then I went directly to medical school, also in Boston, at Tufts University School of Medicine, received my M.D. degree in 1976.
I interned at Georgetown University Hospital in Washington, D.C. in internal medicine in 1976/'77, then I did a fellowship in medical ambulatory care also at Georgetown, 1977 to 1978.
Then from 1978 to 1980, I did a residency in occupational medicine at the Harvard School of Public Health in Boston. At that same time, I also received two graduate degrees, one in public health and a master of science in physiology from

Harvard.
In 1980 to 1981, I completed my senior residency in internal medicine at Boston University medical center in Boston. And so by -- in 1981, I finished my training.
Q. Are you currently licensed to practice medicine?
A. I am currently licensed to practice medicine in Michigan, I have an active license. I'm also licensed to practice in Massachusetts, Washington, D.C., Maryland and California. Those licenses are inactive because I don't reside in those states.
Q. Are you board certified to practice in any specialties in medicine, special areas?
A. Yes. I'm board certified in preventive medicine, and within preventive medicine, the field of occupational medicine. I'm also board certified in internal medicine.
Q. How long have you been practicing occupational and internal medicine?
A. Well, since I finished my residency in 1981, so that's 26 years.
Q. Are you still practicing medicine today?

A. Yes.
Q. As -- over the course of your career since 1981, has the nature of your practice changed?
A. Yes. I'm on the faculty at the University of Michigan, and been there for -- it will be 19 years this fall. And over that 19 years, my time has shifted much more towards doing research and much less in the clinic caring for patients.
Q. Do you still see patients, though?
A. I do.
Q. At the University of Michigan, do you teach classes to students in the medical school and graduate schools?
A. I teach in the School of Public Health principally, and also in the School of Medicine.
Q. What subjects do you teach?
A. I teach risk assessment, I teach occupational environmental diseases. I teach research methods in occupational and environmental epidemiology, and I run a number of different seminar courses. They vary from year to year.
Q. You mentioned that at the University, you

do research. Is there a particular area that you focus on in your research?

A. Yes. I've spent my whole career doing epidemiology, studies of people who handle chemicals and who are exposed to chemicals. So I've spent my whole career out in work places, in factories, in the auto industry, in mines, various industrial facilities, chemical plants, looking at cancer risks among people who are exposed to chemicals, sometimes looking at risks of lung disease, sometimes looking at risks of nervous system or neurologic disease.

That's what I've spent my career doing. And also looking at environmental exposures and disease risks related to those exposures.

Q. Now, does that research that you do, does that involve preparing studies that summarize your findings?

A. Well, it certainly involves writing the findings up and writing them for publication in the peer-reviewed literature. I do that routinely.

Q. Are studies that are performed in your area typically published in a scholarly journal of

some sort?

A. Certainly try to get them published in scholarly journals, yeah.

Q. Can you describe that process for us? What do you have to do to get your research published in a journal?

A. Yeah. So you have to do the research. You have to have written up what the results are and what it means, and you typically put that in a manuscript form. Every journal has its own format. You have to look at the journal format. And then you mail it to the journal, or now you submit them electronically.

The journal receives your manuscript, they send you now an e-mail saying, "Yeah, we received it," and then the editorial board of the journal sends it out to other scientists who are qualified in the area of your research to read your manuscript and review it and write a review, and they write comments and criticisms about the manuscript.

They send those back to the editorial board in the journal, and the journal looks at them, and they say, "Okay, well, we're either

going to accept this manuscript," or "We're not going to accept it." And typically when they don't accept it, they write you back and say, "We're sorry, your manuscript is not accepted, comments from the reviewers are attached. If you are willing to respond to those comments, we would be happy to reconsider your manuscript."

And so you read the comments and you say, "Yeah, you know, I can do those things," and good reviews often suggest things that make your manuscript better. So they say, "You know, you really should have presented this data," or "We'd like to see this table" or whatever, and so then you revise your manuscript and you resubmit it, and hopefully it gets accepted.

Sometimes it doesn't. Sometimes you submit it to a different journal; sometimes you start over again.

And that process -- so then if it gets accepted, then it gets published in that peer-reviewed journal, such as the Journal of Occupational Environmental Medicine or Environmental Health Perspectives or one of the professional journals that publishes original

research.

Q. Is that process called peer review?

A. That's called peer review, yes.

Q. And is it called peer review because you have other scientists that work in the field, people who know the area, look at your research and pass judgment on whether it meets the criteria for your science?

A. Yeah, the idea of having peers is that people that are qualified in the area that you are working in are reading and critiquing your work, and so that's peer review.

Q. How many of your studies have gone through this peer review process and been published in scholarly journals?

A. Oh, I don't know the exact count. It's over 100 at the moment.

Q. Do you participate in the peer review process as one of the people who reviews the studies when they come in to the journals?

A. Oh, sure, yeah.

Q. What journals do you perform peer review for?

A. I review for the Journal of Occupational

Environmental Medicine, Environmental Health Perspectives, American Journal of Industrial Medicine, Cancer Epidemiology, Biomarkers and Prevention, Occupational and Environmental Medicine. There are more. I'm trying to think -- ones I haven't done recently --

Q. It's a bunch of journals?

A. Yeah, there are probably six or seven journals that send me manuscripts and ask me to review them.

Q. Does that take time out of your workday to do that?

A. It sure does.

Q. Do you get paid to do that?

A. No. No, everybody does that because it's part of being a scientist. It's part of participating as a scientist. You know, the reward is that you get to see what's being done, you get to see what people are working on, you get to see what they're thinking about, and it's very engaging work.

Q. You mentioned earlier -- you used the term "epidemiology" to explain research that you do. Can you give us a very short -- we'll get

into it more later. But can you give us a very short explanation of what that is? What is it, epidemiology?

A. Epidemiology, the word comes from the same word "epidemic," and epidemic is an unusual occurrence of disease.

So there's some background rate of typhoid or cholera or tuberculosis and then all of the sudden, there's a big surge. That's an epidemic, okay. Epidemiology is the study of patterns of diseases in the human population, and it has its routes in studying infectious diseases like tuberculosis, cholera, typhoid, those types of diseases, and now it includes studying patterns of cancer risk, patterns of lung disease, patterns of cardiovascular disease and trying to uncover why people get these diseases and why people are at increased risk.

Q. Is epidemiology one of the ways that scientists study the causes of cancer?

A. Oh, absolutely. In the human population, it's probably the principle area of study to try and uncover what puts people at increased risk of cancer.

Q. In your research as an occupational -- in occupational medicine since 1980, have you conducted research into the causes of cancer?

A. Oh, yeah, I've -- a considerable part of my career has been spent doing cancer epidemiology.

Q. Without going over the entire body of what you've done, are there any studies or projects that you've worked on that you are particularly proud of, what you think is your most significant contribution to the body of scientific knowledge on the causes of cancer?

A. I think the one that I like the most is a paper that my colleagues and I published in 1984 that showed that people who have physically active occupations are at lower risk of colon cancer than people who have sedentary occupations.

And we published that in the American Journal of Epidemiology in 1984. It's been 23 years. And that -- that study has been confirmed now over 50 times, and now that finding is in all the textbooks, that physical activity reduces your risk of colon cancer.

And I see it -- you know, I see it in the

news, I see it in, you know, Living Well and Good Housekeeping and all the things you buy in the grocery store. It's basically accepted now as an established fact, that physical activity reduces your risk of colon cancer.

And we were the first people to report that. So I'm -- I'm proud of that one, because I think it had some impact, and maybe it's prevented some cancer.

Q. Have you done any research into the incidence of cancer in the workplace?

A. Oh, sure, yeah. We've -- actually another one I'm very proud of, we did a study for a chemical company, the Rohm and Haas Corporation in Philadelphia. Rohm and Haas had been doing -- had been following their cohort of workers that worked for us in one of their plants in Philadelphia for years looking at cancer risk, and they received an excess of pancreas cancer in their work force, so they came to me, because I had some interest in pancreas cancer, and they said, "What should we do? We think we've an excess?"

And I said, "Well, we really ought to do

a case control study to try to see whether the people with pancreas cancer have been handling chemicals differently or different chemicals than the workers who don't have pancreas cancer." That approach would be a case control study.

And because I was a chemical engineer in college, I actually had some familiarity with industrial processes and industrial chemistry, so I actually did the study. I worked on-site in the plant interviewing workers for months, and we found that people who had handled DDT and two pesticides derived from DDT that were made in that plant were at very high risk of pancreas cancer, and we published that study in the journal of the National Cancer Institute, the JNCI, and it was actually a lead article.

It was a very important article. And it said, "You know, we've got a seven-fold risk among pancreas cancer among people who handle DDT and two very similar chemicals made from DDT."

Q. You talk about some of your research. In your practice of medicine, have you been involved in medical monitoring programs?

A. Yes.

Q. Can you describe your experience with medical monitoring?

A. Yeah. We've got to back up a step. After I finished my residency in 1981, I was asked to join the faculty of the School of Medicine at the University of Southern California. USC had - and still has - a very fine department of preventive medicine that does Cancer Epidemiology studies, so it's a department of 23 or 24 physicians and statisticians who do Cancer Epidemiology, so I joined the faculty at USC, and I was at USC for seven years.

While I was there, I did medical monitoring for a number of different populations of people. I did medical monitoring for two brass foundries. When you make brass, one of the ingredients is lead, and because lead boils off at a lower temperature than the other metals in brass, pouring molten brass generates lead fume, so you have to monitor brass workers typically for lead exposure.

So I sat up and ran medical monitoring for lead exposure on these brass foundry workers.

I also, with two colleagues, ran the

medical surveillance programs for the City of Pasadena, California. Pasadena had a large number of people who required medical surveillance, had police officers, firefighters, emergency responders. Pasadena has its own electrical power plant, so they have power plant workers who are potentially exposed to a number of hazardous materials, and they also have their own parks department that handles pesticides, they have water and sewer workers, so there are confined space entry issues.

So we did all the medical monitoring for all of the City of Pasadena employees.

Q. Through your work in research and medical monitoring, have you become familiar with the human health effects and the exposure to substances like arsenic, cadmium and lead?

A. Yes, I have.

MR. GALLAGHER: Your Honor, we tender Doctor Garabrant as an expert in occupational and environmental health, in epidemiology and in particular, the study of the human health effects from exposure to substances in the environment.

THE COURT: Any objection or voir dire as

to qualifications, Mr. Barr?

MR. BARR: Your Honor, we'll do our questions on cross.

THE COURT: Let me so qualify the witness, and you may proceed.

MR. GALLAGHER: Your Honor, is it okay if I go over there and put a board up on the easel?

THE COURT: Yes, sir.

Q. Doctor Garabrant, did you review the expert report that Doctor Werntz prepared to describe his proposed medical monitoring program?

A. Yes, I did.

Q. And did you -- do you understand that Doctor Werntz has testified that there -- in the class area, there is a significant exposure that leads to a significantly increased risk of certain types of cancer and certain noncancer ailments? You understand that from reading his report?

A. Yes, I do.

Q. Specifically with respect to that question, whether there is a significant exposure leading to a significantly increased risk of cancer or noncancer risks in the class area, do you have an opinion as to whether Doctor Werntz's

conclusions are well-founded?
A. Yeah, I do.
Q. What is your opinion on that?
A. Well, I don't think they're well-founded. I don't think he's gone through the process of putting together the scientific evidence that would adequately provide a foundation for his opinions. And I don't think that scientific evidence actually exists that would provide that foundation.
Q. I'd like to talk about the cancer risks and the noncancer risks separately and start with the cancer risks. With respect to the risk of cancer, how do scientists make a decision about whether an exposure to something like arsenic, cadmium or lead is likely or is known to cause cancer?
A. Let me just go through the steps. First, there has to be research that looks at populations of people who are exposed to that chemical, and that research has to be published, and it has to show that people who are exposed are at increased risk of that chemical.
And that evidence has to include

estimates of the exposure, and you have to know about the exposure pathway, in other words, whether it's a setting where people are handling the chemical because there's dust or fume or vapor in the air, or whether they're getting that chemical through drinking water, so ingestion. So the exposure pathway matters, and the exposure level matters.
And is there an association of increased cancer risk with that exposure? When a study like that is done, we often -- in fact, we almost always expect to see it replicated. In other words, we expect to see that other people have studied the same issue and have found similar or identical results.
You know, and results do vary, you know, from chance a little bit. But we expect to see replication. And so a scientist who says that a chemical causes a disease like cancer has to go back and find that scientific evidence and has to assemble it and has to try and figure out, "Okay, there's 10 or 15 or 20 studies that have looked at that issue. What does that body of literature actually say when you try to put it together into

one coherent picture?"
That's what we try to do.
Q. And you prepared a series of slides to help us understand what epidemiological studies look like, the studies you're talking about that you had collected and look at.
A. Yeah, sure.
Q. Is this the presentation you prepared?
A. Yes.
Q. So tell us what -- if you could introduce to us your presentation on studies. What are we going to talk about here?
A. Before I can talk about what the epidemiology says about cancer, I've got to talk a little bit about what epidemiology does and how we do it.
Okay. So as I've already said, epidemiology is that field of science that studies the distribution of diseases and their causes or the exposures in human populations. Epidemiology is not about laboratory animals; it's about people who live in society and do what people do and who go to work and have exposures at work and come home and eat foods and smoke cigarettes and do

what people normally do.
And the purpose is to understand the causes of disease in human -- in the human population.
Q. Are there different types of epidemiological studies that have been performed to get at that question?
A. There are two principal types of studies, cohort studies and case control studies. Okay --
Q. Can you tell us what a cohort study is?
A. A cohort study is, I think, the one that's intuitively clearest. To do a cohort study, you find a group of people who are exposed to the chemical. Okay? And let's use an example like coffee drinking.
So you would identify a population of people who drink coffee, and then you'd want to compare them to people who don't drink coffee, right? Because you're interested in, "Well, does coffee cause -- we'll use the example of pancreas cancer, because it's a famous one.
So you want to know, does coffee drinking cause pancreas cancer? You say, "Let's get together a cohort of coffee drinkers, let's get

together a cohort of people who don't drink coffee." And so you follow both groups for years - because cancer is a very slow disease - you follow both of these groups for 10, 20, 30, 40 years, and as time passes, you identify who got pancreas cancer in both groups.

Okay, now we can go to the next slide.

Q. Just so it's clear, when you're doing this cohort study and comparing the groups, you have the exposed group that you've illustrated here, and then you have the nonexposed group, right?

A. Exactly.

Q. The exposed group is the group that has been exposed to the thing you're studying, right?

A. Right.

Q. And the nonexposed group is the group that's never been exposed to what you're looking for.

A. Right. And we could use arsenic as an identical example, in other words, a group of people exposed to arsenic and a group of people not exposed to arsenic.

Q. Okay. So what do you do next?

A. You follow them, as I said, in cohort studies for cancer, typically for decades, okay? And you see who got the disease under study. And I've marked here with purple boxes that in both groups, you get cancer. Because there is a background of cancer in every population.

And so the issue now is whether the rate of cancer is higher in the exposed group than it is in the nonexposed group. All right? And so you have to compare exposed to nonexposed, and you want to see what the rates are different, okay?

Q. So how do you do that?

A. Well, essentially you count them up, all right? And so in the top diagram, I've got the two groups have equal rates of disease because in both groups, you've got two out of twelve with the disease.

In the middle, I've got the exposed group has a higher rate of disease, you've got four out of twelve compared to two out of twelve, so that's a doubling of the risk.

And in the bottom diagram, the exposed group has a lower rate of disease, okay? So two out of twelve on the left in the exposed group,

four out of twelve on the right in the nonexposed group, so they have a halving of the risk, in other words, one half. Which is equally as big as two-fold. Like one half and two-fold are equally large ratios.

Q. Okay. So just so it's clear, the three basic possibilities are the two groups will be roughly the same or that one or the other will show a greater incidence of the disease.

A. That's correct.

Q. All right. So then what do you do with the results to provide a conclusion about this?

A. Okay, we calculate what's called a risk ratio or a rate ratio, which is very simply taking what happens among the exposed group and dividing it by what happens among the nonexposed group.

So in this example, in the exposed, two out of twelve got the disease. We put that in the numerator, and then two out of twelve in the nonexposed group got the disease, we put that in the denominator, and the ratio is what matters.

Okay? If the ratio is 1, it says the two groups are equal. If the ratio is above 1, it says the exposed group is at increased risk. So

the risk ratio is above 1, there's increased risk in the exposed, and if the risk ratio is below 1, it says the exposed group has less disease.

Sometimes -- you'll say, "Well, how could you have less disease?" It goes back to my physical activity study. We found that the people who exercised had lower risks than the people who didn't, so the exposure prevented cancer, so it can go either way. Or you can have no association at all. Okay, so --

Q. What --

A. We covered this. The positive association, so here would be a risk ratio of four-fold, and the next one, here would be a risk ratio of one quarter or .25. Okay, so you -- you know, that's how we calculate these relative risks or risk ratios, and we call this a measure of association.

Is the exposure associated with disease risk? Okay.

Q. So in this case, you've got a negative association.

A. You can have a negative association.

Q. And does take mean -- if you see a

negative association, that's less than that 1 value that was the value of them being the same.

A. Right, if it's below 1, it's a negative association.

Q. And if you have a negative association, does that mean that people who were not exposed to the chemical actually got more cancer than the people who were?

A. That's correct, the exposed group has lessor the nonexposed group has more.

Q. So that's a cohort study. Can you explain what a case control study is, the second type?

A. Yeah. I mentioned that pancreas cancer study I did for the chemical company. We did a case control study. Case control study is just a little different design. What you do is, you go out and find people who've got the disease you're interested in.

So here we were interested in pancreas cancer. So you've got to go out and find them, typically by working with the hospitals, working with the tumor registries in the area, working with the state registrar of death information to

get death certificates, so you assemble 50 or 100 or 300 people who've got pancreas cancer, and then you want to compare them to people who do not have pancreas cancer.

And the people who don't have pancreas cancer, we call controls. So the cases are people with the disease; the controls are people who don't have it. And the controls are supposed to come from -- to do the study right, they are supposed to be drawn from the population, the same population from which the pancreas cancer cases came.

So the issue is, okay, we've got people who got cancer, people who don't. What's different about them? All right? What's different about them? If we did that study for lung cancer and we looked at smoking habits, we would realize very quickly that the people with lung cancer had smoked much more than the people who don't have lung cancer, and of course, we have to match them on age and sex and --

Okay. And so what we do then - if I can have the next slide - is that we typically interview them or interview their next of kin or

assemble their work records and find out what their past exposures were, and we do that for both groups, right? And so here I've -- I've highlighted now in dark green among the cases, four of the twelve had exposure, or the way we actually calculate it is, among the cases, four had exposure, eight did not, and we calculate it as odds.

Anybody who's ever bet on horses or a dog race knows what odds are. It's the number of times something will occur if divided by the times it doesn't occur. So we get the exposure odds among the cases. We get the exposure odds among the controls, and then we just compare them, put the odds of the cases, the odds over the cases over the odds on the controls, and we call that an odds ratio, and it tells us the same thing as that cohort study tells us.

It tells us whether exposure is associated with disease. All right? If the cases have had more exposure than the controls, the odds ratio is above 1. If the cases and controls have had the same amount of exposure, the odds ratio equals 1.

All right? So it's the same idea that you're looking to see whether exposure is more common among people with disease, and we calculate a relative risk. In this instance, it's called an odds ratio, but it's the same idea as we saw before. And if it's greater than 1, it's a positive association.

Okay.

Q. So can you explain -- summarizing that, what do the studies - cohort studies and the case control studies - at the end of the day, what do you get?

A. Okay. In both types of studies, we look for this measure of relative risk, and that is called an association between exposure and disease. If it's 1.0, it means there's no association at all, the two groups are the same. If it's greater than 1, it's a positive association; if it's less than 1, it's a negative association.

So it doesn't matter what the study design is, we measure it the same way.

Q. Now, if you have a relative risk greater than 1, does that mean, as a matter of science,

that the thing you are studying causes cancer?
A. Sometimes, but sometimes not.
Q. Can you explain why it can sometimes not mean cancer?
THE WITNESS: Next slide, if you would.
A. Okay. So associations occur for a number of reasons. The first is that it represents a causal -- a causal relationship, the exposure causes the disease. But sometimes it's due to a badly-designed study, that there's some bias in the study, so you have to look carefully.
Sometimes it's due to what we call "confounding," which means there's some other factor that you didn't include in the study that is causing an apparent association. For example, if I study lung cancer and coffee drinking, I would find that they're associated. But they're associated because people who drink a lot of coffee tend to smoke more.
And so that's confounding due to smoking. The true cause is something that I didn't include in my study - it's the smoking - and it produces this apparent association between coffee drinking and lung cancer, and so you have to deal with

that, or when you -- when you read a study, you have to think, "You know, is there some confounding factor that they didn't deal with?"
Okay, because that can -- that can give you a -- an erroneous conclusion. Okay, and then I think the most important one, at the bottom, is chance, okay? Associations occur by chance, and we have to evaluate the role of chance, and scientists always do that. They always calculate "P" values or confidence intervals to evaluate, "Well, what's the role of chance in these findings?"
Q. The confidence interval, can you explain what that means?
A. Sure. We calculate this measure of association called relative risk, and then we calculate the confidence interval around it, and conventionally, it's a 95 percent confidence interval, and that confidence interval is the range within which the relative risk would fall 95 percent of the time if we did the study over and over and over again.
And you know, we see these in the newspaper all the time, when you read that they've

taken a poll of how many people approve of the president's performance and, you know, let's say they find 32 percent plus or minus 4 percent, that plus or minus 4 percent is the confidence interval.
That is, what they're saying is, for a sample as big as we took - and these surveys often include 800 or 900 people - we are 95 percent confident that the true answer falls between 32 plus or minus 4, so between 28 and 36. That's a confidence interval.
And so you see it all the time, and you always see it in scientific studies, because it gives you a sense for, "Okay, is it possible that chance explains this?" Now, the important point --
THE WITNESS: If I can have the next slide.
A. So -- often we see graphs like this where the relative risk is presented as a little box or a little circle or something in the middle of a range, and then a bar on either side represents the confidence interval.
If that confidence interval includes 1.0,

what it means is, "Well, okay, these results are reasonably compatible with the idea that there's really no association." In other words, we saw a relative risk of 1.38, but it's reasonably likely that the truth could be 1.
"We saw 1.38, but it could be 1." On the other hand, if that confidence interval -- if the relative risk is up there way high and the confidence interval doesn't include 1, then you say, "You know, this is unlikely to be a chance finding," so chance is effectively not the explanation for it.
So we always evaluate the role of chance in assessing these relative risks.
Q. In any one study that you've looked at, any one epidemiological study about cancer, would you see a relative risk reported with a confidence interval?
A. You typically do. Older studies didn't used to do it, but for the past 20 years, it's been typical to do it that way.
Q. Now, as a scientist, would you draw a conclusion about causation based on one study?
A. I -- I would be very reluctant to do

that, and I would not do that if there were other studies that were properly conducted. I would look at all of the evidence. I wouldn't rely on a single study. I'd look at all the evidence.

Q. When it comes to studying the causes of cancer, what substances cause cancer and under what circumstances, are there bodies -- are there groups of scientists who compile all the evidence and put it all in one place for you to look at, summarize it for you?

A. Yes.

Q. Is the International Agency for Research on Cancer one of them?

A. Sure, yeah. The International Agency for Research on Cancer is actually part of the United Nations. Within the UN, there's the World Health Organization. We all remember UNICEF when we were kids, you know, collecting money in grammar school for UNICEF, vaccinating kids abroad. That's part of the World Health Organization.

World Health Organization also includes an agency called IARC, or the International Agency for Research on Cancer, and its mission is to do cancer research and particularly to evaluate which

chemicals and industries and occupations and foods and pharmaceuticals cause cancer in humans. It's a very important function.

And so IARC does that, and they use a -- a grouping system, Groups 1, 2A, 2B, 3 and 4.

Agents that are put into Group 1 are ones where the scientists who are convened feel that the evidence is adequate to say "this chemical causes cancer in humans." Okay?

2A means the animal evidence is sufficient to say it's an animal carcinogen, but the human evidence isn't good enough. There's some human evidence, but it's just not good enough.

And the -- the conclusion is, "Well, that chemical is probably carcinogenic to humans," but the evidence isn't good enough to say that we know it causes cancer in humans.

And then for agents where there's little human evidence or sometimes no human evidence, but the animal evidence is still good, those are typically put in 2B where it's "possibly carcinogenic to humans." In other words, the human evidence isn't there, but we know it's an

animal carcinogen, it might be carcinogenic to humans.

Group 3 is chemicals for which there is just not enough evidence to make any decision, we can't classify it. And then Group 4 - and you can see that there's only one chemical that's ever been put in Group 4 - the evidence is strong and adequate to say, "This thing probably doesn't cause cancer."

So -- so the -- the plan is to figure out which chemicals cause cancer and to assess the strength of the evidence.

Q. Are there other agencies like IARC that perform a similar function of classifying substances by what the evidence shows about their cancer risk?

A. Yes. And I put together a little slide. Across the top row, you can see the U.S. Environmental Protection Agency does this, IARC does this, the American Conference of Governmental Industrial Hygienists, the ACGIH, does it, and the National Toxicology Program, or the NTP, does it. And you can see that they have different letters and numbers, which makes it very confusing.

But the categorization system is very similar across agencies. So a Group 1 or a Group A or a Group A-1 means "This agent causes cancer in humans, we have enough evidence to say that," and then the ones below that mean, "Well, there's limited evidence in humans, but some animal evidence" or "No evidence in humans but some evidence in animals," and it goes on from there.

Q. Have you looked at the classifications that the organizations provide for arsenic, cadmium and lead?

A. Yes, I have.

Q. Is there any difference in their -- in the treatment that the EPA provides in terms of where it classifies it versus IARC and the other agencies?

A. Well, we've got to go through the agents. Arsenic is a human carcinogen by everybody's classification. There's no argument about that. Cadmium is a human carcinogen according to IARC, but is in the next category down according to two of the classifications, and I don't have my own list in front of me.

I think it's EPA and ACGIH. So there's

some minor disagreement about the strength of the evidence for cadmium.

And then for lead, none of these agencies classifies lead as a human carcinogen.

Q. You mentioned that these agencies classify arsenic as a human carcinogen. Why isn't -- the question here is, is arsenic in the environment? Why isn't that classification the end of the story? I mean, why not just stop right there and say, "The EPA says this is known to cause cancer?" Why don't we just stop with that?

A. Well, there's much more to the story than that.

You know, if I could just get one of the IARC books, these are -- these are complicated decisions. The assessment of arsenic -- this is the volume on arsenic in drinking water put out by IARC, Volume 84, published in 2004.

This is the volume that includes cadmium, Volume 58, published in 1993. This is the volume on lead, inorganic and organic lead compounds. It's published in 2006, just came out earlier this year. I mean, it's a whole book on what the evidence says, and a full and fair assessment of

what that evidence means.

And when IARC does that, they consider exposure levels, they consider whether the exposure occurs by inhalation or whether it occurs in drinking water or whether it occurs by skin contact. They consider the dose. They consider all of the human epidemiology.

They consider all of the animal experiments in laboratories and rats and mice and hamsters and other species, so it requires a lot of work to consider the entire body of evidence, and it's critically important to think about the exposure pathway and the levels of exposure that put people at risk as well as what types of cancer occur from a certain exposure pathway.

Q. That's --

A. Things you inhale may cause cancer in the lungs but nowhere else, whereas things in drinking water may cause cancer in different organs, because the exposure pathway matters.

Q. That's a point I want you to elaborate on. You said it's important to consider what type of cancer. Are all cancers the same?

A. No, no. I think most people have had

personal experience with cancer, either through a parent or a sibling or an aunt or uncle or grandparent or children. And cancers are very different.

So for example, nonmelanoma skin cancer is almost always curable; lung cancer is rarely curable. Pancreas cancer is one of the worst. I mean, the five-year survival for pancreas cancer is horrible.

So cancers have different presentations, they have different treatment, and they have different survival. They are different diseases.

Q. Do they also have different causes?

A. Yes, they have different causes as well.

Q. So just because something causes lung cancer in certain circumstances through certain exposure routes, that doesn't mean that it's going to cause skin cancer through another route.

A. That's correct. I mean, the cancer risks are specific to the chemical and the pathway of exposure. And it differs, pathway by pathway.

Q. Now, does the designation that's given to a -- to an element like arsenic by EPA or the IARC, does that designation tell you what kind of

cancer it's known to cause, through what -- by what route of exposure or at what dose?

A. No. No, this is a -- a summary sentence that says, "The evidence is adequate to say, this is a carcinogen to humans" or "The evidence is not adequate to say this is a carcinogen to humans, but it's adequate to say it's probably or possibly carcinogen to humans."

Q. Yesterday, Doctor Werntz suggested that living in the area around a smelter necessarily increases your risk of cancer. Do you agree with that conclusion?

A. No, I do not.

Q. Why not?

A. Let's look at the evidence.

Q. What evidence do you need to look at?

A. Well, the epidemiologic evidence, that studies that have looked at populations that live around smelters. And these studies have been done in many, many locations throughout the world.

Q. And do you need to look at that evidence separately for arsenic and separately for cadmium and separately for lead?

A. If you can, yes.

Q. Why --
A. If the evidence exists, that's what you should do.
Q. Why do you want to look at the evidence separately for arsenic and separately for cadmium and separately for lead?
A. Well, first off, our assessment of whether these chemicals cause cancer in humans is different for each of those. The types of cancer they cause are different, and they differ in terms of which pathways of exposure lead to cancer and which cancers.

So you really have to be specific about the agent and the route of exposure and the chemicals that it's referring to.
Q. In your work, did you look separately at arsenic, cadmium and lead?
A. Yes.
Q. So let's start with arsenic. Doctor Werntz includes monitoring in his medical monitoring program for, I think, four different types of cancer that he says are -- are at issue here. He talks about lung cancer, he talks about bladder and kidney and skin cancer.

Have you considered whether there is a significant increased risk of those four cancers as a result of the exposure that we have in this class area to arsenic?
A. I have. And we should look at the evidence.
Q. What does the scientific literature say, in general, about whether arsenic causes cancer or under what circumstances it causes cancer?
A. Well, it's very clear that arsenic causes lung cancer when you inhale it, and that evidence comes from people who are in industrial operations where there's arsenic exposure. So there's no argument about that.

If you worked in arsenic smelting, arsenic refining and there's high level of exposure to arsenic by inhalation, it's associated with an increased risk of lung cancer, and in fact, that is much of the evidence upon which our assessment that arsenic is a carcinogen is based.

It comes from the occupational studies. Okay. So inhalation of arsenic, particularly in occupational settings where the exposures are high, is linked to increased risk of lung cancer.

Q. Can you explain what this chart is you prepared?
A. Yeah. This is just a schematic to try and make it clear that the risks of cancer are different for inhalation than they are for drinking water. Okay? So for inhalation, particularly in occupational settings, lung cancer is a risk.
Q. Is there any indication in the scientific literature that inhalation of arsenic at any level - whether it's in the environment or occupational exposures - that that pathway, that route, exposure, causes bladder, skin or kidney cancer?
A. I don't believe so. I don't believe so. It's lung cancer in the inhalation literature.
Q. And then you have a separate route down here for drinking water where you list some additional cancers. Tell us why you provided that.
A. There are a number of areas around the world that have very high arsenic levels in their drinking water. Taiwan, particularly the southwest coast of Taiwan, Chile, and Bangladesh, and these -- they have large populations who have

drunk highly contaminated water for decades.

There have been an extensive number of studies of cancer risks in Taiwan where people have drunk arsenic-contaminated water from wells. And those studies show very clearly that people are at increased risk of lung cancer, bladder and kidney cancer, and skin cancer.

And the evidence from Chile also shows the same patterns. So I think there's no argument that drinking water contaminated with arsenic at high levels is associated with increased risk of those four types of cancer: Lung, bladder, kidney and skin.
Q. Now, in your chart, you indicate that lung cancer is associated with exposure to arsenic when you have inhalation and occupational exposures. What does that mean?
A. It means that the -- the evidence that inhalation of arsenic causes lung cancer really comes from occupational studies. It doesn't come from environmental studies. In fact, there have been a lot of environmental studies that have looked at that, and they have not given any clear indication of increased risk.

And I'd like to review those. You know, some are positive, and some are negative as well.
Q. And one last question on this side, and then we'll go to those studies. When you indicate that drinking water is a -- arsenic is associated with these cancers when there's exposure in drinking water, as the high levels that you indicate there, has there been any indication in this case that there are levels or exposures of that type?
A. No. The studies in Taiwan involve populations where the drinking water levels are -- the majority of the wells are over 300 micrograms per liter, and the levels are 300, 600, 800 and above.
From the evidence I've seen in this case, the highest value found in any reading in any well here was 80. And it's my understanding that the population's been on municipal water since 1964, so even though there was a single well reading at 80, that the class area hasn't drunk that type of water at all - or rarely - and even that is well below the levels in Taiwan that are known to put people at risk of these cancers.

Q. Have you read Doctor Brown's analysis?
A. I have.
Q. Have you read Doctor Werntz's analysis?
A. I have.
Q. Did either of those experts identify drinking water like the drinking water in Taiwan or Bangladesh or these other areas as a source of exposure to people in the class area?
A. No.
Q. So you said you wanted to talk about the studies. Have you considered the studies that exist on -- on whether living near a smelter or an industrial site like that, whether that can increase your risk of lung cancer?
A. I have.
Q. And does this slide include the studies that you looked at?
A. It includes some of them. There are more that I have reviewed in addition. There would be a few more slides of those. But I think these give the picture. And this slide includes the one that Doctor Werntz considered, and others that he didn't consider.
Q. So in your summary slide you've provided,

did you include the studies that Doctor Werntz relied on?
A. Yes.
Q. And did you include some additional studies that you found through your own research?
A. Yes.
Q. Let's walk through this. And tell us what the studies show about whether exposure to arsenic in the environment like you would get living around a smelter, whether that puts you at an increased risk of lung cancer, if you could walk us through.
A. Sure, yeah. So I've listed across the top the name of the study. I should probably get them out. Hold on a second.
Okay. So there's a lot of literature. Okay. So I've listed the author, so the first one is by Brown published in 1984, and then a brief description of the population that was studied, and so you can see in the Brown study, this is a study of men whose usual residence was near a zinc smelter, and then I've got the relative risk, and in parentheses, the confidence interval.
And you can see in the Brown study, the

relative risk was 1.6, so it was -- it was above 1, slightly, and you can see that the confidence interval included 1, went from .8 to 3.4. So that's not a statistically significant finding.
I want to point out that the authors of the Brown study calculated a 90 percent confidence interval, which is a little narrower than the one we usually calculate, which is 95. So I recalculated the confidence interval, and it's a little bit wider. Doesn't change the answer.
So Brown found the positive association. But it was reasonably compatible with there being no real association, that is, 1.0, and that the results may be due to chance. Okay.
Q. Let me just ask you, based upon the Brown study, could you conclude that people who lived in the area of a zinc smelter and had exposures like the exposures we're talking about here in this case, could you conclude that they had -- were at a significantly increased risk of lung cancer?
A. No, you would not conclude that. They do not have significantly increased risk. They have nonsignificantly increased risk. In other words, it's possible that this is just chance.

Q. What's the next study you looked at that you summarized in your table?
A. The next one is Pershagen, 1985, and that study was updated recently and published again in 2003.
Q. What does it mean to update a study?
A. Well, they had followed that population for more years, all right? So 18 years later, we've got an update where they followed the population. Remember I said in cohort studies, it takes decades. So you've got a lot more evidence - 18 more years - and much more data to work with to try and measure the cancer rates.
Okay. So in the original Pershagen study -- these are two parishes near a copper, lead and zinc smelter in Sweden, and the original report said it was a two-fold relative risk, and it was statistically significant in men; the confidence interval goes from 1.2 to 3.4.
But the update that came out just four years ago gives the results now for men and for women, and they've come down. So in men, it's 1.38 and it's no longer significant, confidence interval goes from .89 to 2.14, and in women,

there's a negative association. It's .88. It's not significantly negative.
So the point here is that it's a little higher in men, a little lower in women, but on balance, it's pretty close to 1.0. In other words, it doesn't appear in the Pershagen study, as updated recently, that there's evidence of increased risk of lung cancer among people who lived near a smelter.
Q. What is the next study you looked at?
A. The Bartlesville, Oklahoma study. This was one done by ATSDR. It's part of the Centers for Disease Control. This is residents near a zinc smelter. They didn't give the relative risk, at least the numbers aren't in the report.
But they did say the occurrence of lung cancer was as expected from the state rates. So what they're doing is, they're making a comparison of the rates in the Bartlesville population to the rates in the rest of the state. They said, "It's the same; it's as expected."
Q. What does that tell you about whether living near a zinc smelter puts you at a significantly increased risk of lung cancer?

A. Well, that's a clear statement it does not. It's -- you've got the rate that you would expect among the rest of the state that's not exposed.
Q. Now, the next study you have listed here is the Tokudome study. Is that one of the studies that Doctor Werntz relied upon in support of his conclusion that there was a risk here?
A. Yes.
Q. Tell us about the Tokudome study.
A. Well, the Tokudome study was a study of workers in a copper smelter. This is not a study of people who lived near a smelter; it's a study of the workers.
Q. Why is that an important distinction?
A. This is really important because these are highly-exposed people who worked in and around it.
Q. Not the people who lived around it.
A. This was not a study of people who lived in the vicinity of a smelter, no.
Q. Was there anything about the Tokudome study that you could look at and draw at least some conclusions about whether people living

around a smelter are at increased risk of lung cancer?
A. Well, I want to be cautious about making leaps of faith. Let's talk exactly what the Tokudome study says. In the workers in the smelter, the refinery workers, there was an 11.89 or almost 12-fold risk. They didn't give the confidence interval.
This is an old study. It goes back 30 years, 1976. But they did calculate a "P" value, and they said this is statistically significant with a "P" value of less than .01. Okay, so 12-fold risk in the workers.
They also included clerical workers who worked in the smelter -- not out in the smelting operation, but in offices on the site. Those people were at no significantly increased risk, so the relative risk was 1.75 and it was not statistically significant.
Q. So can you extrapolate out to the community around the smelter?
A. No, you cannot.
But what it does say is that even among the workers on-site who were in office settings,

there was no significantly increased risk, and it suggests that the risk comes from being out in the plant under heavy exposures.

Q. Doctor Werntz relied upon that study to support his conclusion that there was a risk of lung cancer here. Do you have an opinion whether you can draw that conclusion from the Tokudome study?

A. I don't think that's fair. This is not a study of people who live near a smelter. It's not fair to draw that conclusion. It's not what the study says.

Q. What's the next study you looked at?

A. Okay. Frost looked at females who lived within three miles of a copper smelter and arsenic refinery, found a relative risk of.82, so lightly less than 1. Confidence interval included 1, so not significantly different than 1. That study basically says there's no association.

Q. And what about the Hinwood study you reviewed?

A. Okay, Hinwood is a study from Australia where they looked at areas that had high surface soil or drinking water arsenic concentrations.

Some of those came from mine tailings from mining operations; some were naturally occurring, and the Hinwood study found a relative risk of 1.0.

Q. So what does --

A. Exactly on the "no association" mark.

Q. What does that tell you about our situation here?

A. To the extent that it's applicable, it talks about contaminated water, contaminated soil not conferring any risk of lung cancer to people who lived in those contaminated areas.

Q. What's the last study included on your slide?

A. The Murray -- the Murray, Utah study, also done by ATSDR. This was a study of residents near a lead smelter. They didn't give the relative risk, but they did conclude that they considered the Murray smelter site to be no apparent public health hazard. They did not feel there was any reason to consider this a hazard.

Q. Now, on the question of whether the exposure to arsenic in the class area puts people at a significantly increased risk of lung cancer, did you review just these studies, or do you look

at more?

A. I looked at quite a few more in addition.

Q. Did you look at all the studies that Doctor Werntz relied upon?

A. I certainly did.

Q. And based on your review of the studies that Doctor Werntz relied upon and the wider body of scientific knowledge, do you have an opinion as to whether the people in the class area are at a significantly increased risk of lung cancer as a result of arsenic exposure?

A. Well, they're just not. The scientific evidence would not support that conclusion.

Q. Now, in your slide you showed us before, you talked about the pathways for arsenic, and bladder cancer, skin cancer and kidney cancer. Are those three cancers that Doctor Werntz mentions in his report and about which he concludes there's a significant risk here?

A. Yes.

Q. Did you look at the studies on those?

A. I did.

Q. And is this the slide you prepared to summarize what the studies say about whether

people who live near a smelter are at a significantly increased risk of kidney cancer, bladder cancer and skin cancer?

A. Yes.

Q. Can you walk us through your analysis of that question?

A. Yeah. The -- these studies, which are a subset of the ones on the previous slide, were studies in which the authors gathered information on other cancers. In most cases, they didn't report the results.

Q. What does that mean -- first let me just ask: As we did with lung cancer, are you talking about studies that are relevant to the question of whether people living near a smelter are at risk of these cancers?

A. Yeah. That -- yes. This isn't -- this is an assembly of the evidence that tries to answer that question.

Q. And when those studies say that the cancers are not reported, what does that tell you as an epidemiologist?

A. Okay. Well, let's -- let's just go back one step. When you do a cohort study, you have a

population of people and you follow them over time and you get their medical records together or their death certificates together, you get all of their records on every cause of death, and so it's routine that you get lung cancer, kidney, bladder, skin, pancreas, prostate, brain, you know, every type of cancer, diabetes and lung disease and heart disease, you name it. That's how you do a cohort study.

Okay. So some of these were cohort studies. They had the data for particularly kidney cancer and bladder cancer. Now, they typically don't have skin cancer, okay, because skin cancer is rarely fatal and it doesn't come up in tumor registries or death certificates.

But for bladder and kidney, they had the data. Okay, for Bartlesville, the first one, they didn't report the results, but they did say the occurrence of kidney cancer was as expected from the state rates. So there was no excess of kidney cancer.

They did not report -- they didn't say anything about bladder cancer, but they clearly had the data. Okay.

Tokudome had the data, didn't report the results for any other cancer sites.

Hinwood, the Australian study, did report the risks for bladder cancer and kidney cancer and you can see that there's a slight negative association for bladder and a slight positive association for kidney. Neither of them is significant. Confidence interval would include 1.

The Murray, Utah study did not report any other cancers, but they concluded that the site was not an apparent public health hazard. If there had been an excess of cancer, I doubt that they would have concluded there's no public health hazard.

And then the last one is Wong. That's a study that I haven't presented before. That's actually a study of skin cancer, and that's among residents near the Anaconda copper smelter and Anaconda mine where Doctor Wong did not give the odds ratios; he simply presented the skin cancer incidence rates and showed that they were significantly lower around the copper smelter than in nonexposed counties in the same state.

So again, that's not evidence of an

excess; in fact, it's evidence of a deficit, a negative association for skin cancer.

Q. So have you considered studies that Doctor Werntz relied upon in reaching his conclusion that there was a risk of bladder, kidney and skin cancer here?

A. I certainly have. Many of these are the studies that Doctor Werntz used.

Q. And have you considered additional studies you found through your own research?

A. I have indeed, yes.

Q. And what is your conclusion as to whether the people living in the area of the smelter are at a significantly increased risk of bladder cancer, kidney cancer or skin cancer?

A. Well, there's not scientific evidence that would support that conclusion. It's that simple.

Q. So the next element that Doctor Werntz looked at - we've talked about arsenic - he looked at cadmium. What does the scientific evidence say about whether cadmium causes cancer and under what circumstances?

MR. GALLAGHER: Your Honor, if -- I'm

starting a new topic, so if you want to take a mid-morning break, now would work, or we can --

THE COURT: Why don't you do that. Do you want the witness to answer your question, and then we'll take our break? If he recalls the question.

MR. GALLAGHER: I'll ask it again and so --

THE COURT: Okay.

Q. What does the scientific evidence -- what does the literature say about cadmium and whether it causes cancer and under what circumstances?

A. Well, IARC has classified cadmium as a human carcinogen, and it is associated with lung cancer under circumstances of occupational inhalation exposure. So if you inhale cadmium dust or fume in sufficient quantities, there is an increased risk of lung cancer.

Now, some of the agencies disagree. They put IARC -- or they put cadmium in the next category down, "probably carcinogenic," by IARC says it's a human carcinogen, and I would agree with that. The evidence is sufficient --

Q. Is --

A. -- lung cancer by inhalation.
Q. Is cadmium in the literature, including the IARC, is cadmium associated with any other cancers through any other route of exposure?
A. The simple answer is no. Cadmium is a metal that's been investigated extensively since I was in Public Health School -- before I was in Public Health School in the late '70s. There was an initial report that it was associated with prostate cancer, and a lot of people tried to investigate that. It didn't bear out.
I know Doctor Werntz claims it was associated with kidney cancer. That doesn't bear out. The only one that bears out is the lung cancer association.
Q. Why don't we come back to that topic and the evidence about the risk around this smelter after our break.
A. Okay.
THE COURT: Ladies and gentlemen of the jury, why don't you go with your bailiff about ten or fifteen minutes, then we'll call for the continuation of the testimony.
(The jury exited the courtroom.)

THE COURT: Counsel, why don't we take about ten minutes, please.
(A recess was taken after which the proceedings continued as follows:)
MR. BARR: Your Honor, may we approach real quick?
THE COURT: Yes, please.
(Counsel approached the bench and the following proceedings were had out of the hearing of the audience:)
MR. GALLAGHER: There's one issue I wanted to raise that may come up in the further examination of Doctor Garabrant. I understand there's been a ruling on Lenora Perrine's blood lead level.
One of the topics we're addressing is medical monitoring and whether there's sufficient evidence of significant exposure, and one of the things that he's going to talk about is that you would see that in blood lead, you would see it if it were there, and I'm prepared to stop right there --
THE COURT: Okay.
MR. GALLAGHER: -- but I think that --

and he can discuss the ATSDR study which has been talked about a lot. I think that Doctor Werntz yesterday opened the door to further discussion beyond the ATSDR study when he testified in response to questioning that -- the question was, "If you took blood lead tests of people who lived in the class area in light of ongoing exposure," so talking about today, "would their blood lead test be elevated," and he answered "Yes," which is the question, and he volunteered, "Yes, that was what was found," the suggestion being --
MR. BARR: Could you keep that open?
THE COURT: Didn't he also -- and then he went on to talk about the ASTR -- whatever the acronym is
MR. BARR: ATSDR.
THE COURT: Thank you.
-- and that, so --
MR. GALLAGHER: He did, but that was with respect to 1996. And the question where he volunteered, "That was what was found" was the question that was about "ongoing today."
So the suggestion I think he left with the jury was that there are tests out there that

they haven't heard about.
THE COURT: Now, it's -- I mean, he was talking about the ATSDR study, so let me -- and I appreciate you raising it in this manner, so --
MR. GALLAGHER: Okay.
THE COURT: -- that's the exact way to do it to keep from getting crabbed at by the judge. I remember those days. So let me ask -- and if you need to woodshed the witness --
MR. GALLAGHER: He knows that there's a ruling, your Honor, and he knows that he's not to talk about it, and what I have prepared is a question of, "Is there any evidence?"
And his answer to that is, "No," he's not going to talk -- he's going to say, "I have not seen any evidence of elevated blood levels," and we can leave it at that, that and the ATSDR --
THE COURT: Okay, thank you.
(Counsel returned to counsel table and the proceedings continued in the presence and hearing of the audience as follows:)
THE COURT: Mr. Bailiff, you may return the jury to its box, please.
(The jury returned to the courtroom.)

THE COURT: All 11 of our jurors again being present before the Court and the parties and the respective counsel, you may continue your examination, sir.

BY MR. GALLAGHER:

Q. Before we took our break, Doctor, you were telling us now what the scientific literature says about the risks of cancer and exposure to cadmium. Have you prepared a slide that -- like the one you did for arsenic that summarizes that body of knowledge?

A. Yes.

Q. And is this the slide you prepared?

A. Yes.

Q. If you could, tell us what you're illustrating here.

A. Okay. What the slide is showing is that cadmium is associated with lung cancer in settings where there is inhalation in occupational settings. Okay? So the evidence that cadmium is a carcinogen really comes from studies of workers who have been heavily exposed in industry and they are at increased risk of lung cancer.

I'm not aware of any evidence that links

cadmium to cancer via drinking water. I don't believe that is an exposure pathway that carries any risk of cancer.

You know, it should be mentioned that cadmium is in the food supply, small amounts of it occur in fruits and vegetables and grains, and we ingest cadmium in small amounts every day, and there's no evidence that that's a risk factor for cancer.

Q. Doctor Werntz offered the opinion that we should have medical monitoring for lung cancer and a variety of other cancers, I think kidney cancer was one of them based on the exposure to cadmium in the class area.

Do you have an opinion as to whether there is scientific evidence to support a finding that people who live in the class area are at significantly increased risk of any cancer due to exposure to cadmium?

A. Well, I do have an opinion, and I think that the materials I've just shown strongly support an opinion that there's no reason to think there's increased risk of cancer from cadmium exposure resulting from living near the smelter.

Q. Now, with -- with arsenic, we went through a list of studies, and you told us what the studies showed about whether living near a smelter puts you at an increased risk. Did you do a similar analysis for cadmium and the risk of lung cancer?

A. I did.

Q. And is this the slide you prepared to summarize your review of the studies?

A. Yes.

Q. First let me ask you: What studies did you include in your review on cadmium?

A. Okay. These are the same studies that I had on the slide of arsenic in lung cancer because the smelter studies all included arsenic and cadmium exposure. So the fact of living near a smelter relates to the issue of whether cadmium carries risk of lung cancer as well as it did for the issue of whether arsenic causes -- puts you at increased risk of lung cancer, so it's the same set of studies, and it's the same set of results that we have already discussed.

And it's the same conclusion that the evidence in populations who have lived near

smelters does not support a conclusion that they are at increased risk of lung cancer as a result of cadmium exposure.

Q. Same -- same studies, same conclusion as you had with arsenic?

A. That's correct.

Q. Now, Doctor Werntz also talks about kidney cancer and the potential for -- the risk of kidney cancer here. Have you analyzed that question by looking at the studies and the scientific literature?

A. I have.

Q. Let's first talk about the studies you looked at in particular. What did you look at with respect to studies about people living near or exposed near smelters and the risk of kidney cancer?

A. There are really only the three, and we discussed them all previously: The Bartlesville, Oklahoma study of residents near a zinc smelter where they said the occurrence of kidney cancer was as expected from the State rates, which is a round-about way of saying "No increased risk."

The Tokudome study which didn't report

the results, and my comment earlier is worth saying again, the fact that they didn't report it, it's reasonable -- it's reasonable to conclude that there wasn't any significant excess.

And then the Murray, Utah study by ATSDR, residents near a lead smelter, ATSDR considered that smelter site to be no apparent public health hazard. If there had been a significant excess of kidney cancer, I think they would have said, and they didn't. They said, "There's no apparent public health hazard."

Q. Now, beyond the smelter studies, is there a body of scientific knowledge, studies that have been done, to test the question of whether cadmium causes kidney cancer through an inhalation pathway? Whether you -- whether it's near a smelter or in some other scenario, is there literature on that?

A. Oh, yeah. Yeah, there is literature on cadmium and kidney cancer that has looked carefully at that, and it is not supportive of the conclusion that cadmium causes kidney cancer.

Q. Did you prepare a slide to summarize your review of that -- another body of literature

beyond the smelter studies and what it has to say about whether cadmium causes kidney cancer?

A. Yes.

Q. And this is your slide?

A. Yes, this is my slide.

Q. Tell us what you found when you looked at the body of literature on this question of whether cadmium causes kidney cancer.

A. Yeah. The agencies that have reviewed the evidence of carcinogenicity of cadmium have not concluded that cadmium is associated with kidney cancer in humans. They haven't made that conclusion.

And there have been a good number of studies of cadmium exposure in industrial settings, typically workers, battery workers exposed to cadmium, you know, nickel cadmium, NiCad batteries, lab workers exposed to cadmium, cadmium alloy workers in the metal industry, cadmium pigment workers.

Cadmium pigments have been used extensively in paints. They produce beautiful oranges and yellows and reds. And other cadmium-exposed workers in a variety of industries, and

the hypothesis that cadmium is associated with kidney cancer doesn't bare out. It's not.

Q. Has this question of whether cadmium causes kidney cancer, has it been extensively studied by scientists looking for the causes of cancer?

A. I believe that would be fair to say.

Q. And in that entire body of scientific knowledge -- including the IARC report and everything else -- is there a scientific basis for concluding that people in the class area are at a significantly increased risk of kidney cancer as a result of exposure to cadmium?

A. No, there's not. There is not a scientific basis for that conclusion, and I don't think that's a valid conclusion.

Q. Now, earlier you showed us the summary of the IARC monograph and the different classifications for substances and whether they're carcinogens. You recall that?

A. Yes.

Q. And you showed us some slides that show that arsenic and cadmium can cause cancer in certain circumstances, right?

A. Yes.

Q. Are you saying that these substances -- that arsenic and cadmium -- that they don't cause cancer?

A. No. No.

Q. To sum it up for us, what is your opinion with respect to arsenic and cadmium, and specifically the question of whether there is a significantly increased risk of cancer as a result of exposure to arsenic or cadmium that you had in this class area?

A. Okay. Arsenic causes lung cancer by inhalation in industrial settings. There is not evidence that arsenic puts people at increased risk of lung cancer from living near smelters.

Arsenic causes lung, kidney, bladder and skin cancer when it's in your drinking water. I'm not aware that drinking water is an issue in this case. It wasn't in Doctor Brown's report; it wasn't in Doctor Werntz's report; and I haven't seen any evidence that there was drinking water contamination sufficient to have put anybody in increased risk.

So I think the drinking water exposures

are not -- there's no basis to say that that's a risk factor.

Cadmium causes lung cancer by inhalation in industrial settings. I think that IARC has that right, even though other agencies don't agree, I think IARC has that right.

There is not evidence that people who live downwind of smelter sites are at increased risk of lung cancer if they inhaled or -- I should say if there is cadmium exposure from those sites. There's just not evidence that they're at an increased risk.

So -- and I guess lastly, cadmium is not known to cause kidney cancer, as Doctor Werntz believes. The science doesn't support that conclusion.

So when you add it all up, I don't think that there is a scientific foundation that in any way supports a conclusion that there's increased risk of those cancers as a result of living near this site.

There's simply no basis for it.

Q. The last thing that Doctor Werntz said on the subject of cancer was that exposure to lead

puts you at an increased risk of cancer. What does the scientific literature say about lead and cancer?

A. We've already discussed that none of the agencies rank lead as a human carcinogen. Most of them rank it as a probable or possible carcinogen based on animal evidence, recognizing that the human evidence is not adequate to say that this is a human carcinogen.

If you look a little more deeply into the animal evidence, the evidence that lead compounds cause cancer in animals -- and we're talking about rodents, rats, mice -- is evidence derived from lead compounds like lead acetate, lead subacetate, lead phosphate.

It's not -- there is not evidence that compounds like lead oxide, lead sulfide, lead sulphate cause cancer even in animals. And those compounds, lead acetate, lead subacetate, lead phosphate, I don't even think are in issue in this case, I don't think that they would be present in a smelter.

So lead may cause cancer in animals, but it involves lead compounds that really aren't

present here. The compounds that may be present here, I don't think there's even evidence in animals to support it.

Q. Yesterday Doctor Werntz, in his testimony, talked about the IARC monograph on lead in Volume 23, and they put up on the screen this quote from that monograph, and this quote says, "There is sufficient evidence that lead subacetate is carcinogenic to mice and rats and that lead acetate and lead phosphate are carcinogenic to rats."

Just so it's clear, do you believe that this is sufficient evidence from which to conclude that people in the class area are at a significantly increased risk of any cancer as a result of exposure to lead?

A. No.

Q. And can you explain for us, using the monograph, this quote that Doctor Werntz pointed to, can you explain to us why not?

A. Well, you know, the first is, you know, lead acetate -- okay, vinegar is acidic acid, okay, and so lead acetate is the lead salt of vinegar. Now, I don't think that lead acetate is

present in the smelting environment. I can't imagine what it has to do with smelting. I was a chemical engineer in college, it doesn't make sense to me.

And the same thing with lead subacetate, it's another salt -- it's another lead salt of vinegar. It's not here. So the evidence that Doctor Werntz is relying on is about lead compounds that are not at issue here.

So that's -- so that's Issue No. 1.

Issue No. 2, I don't dispute that those compounds are carcinogenic to rats or to mice, but it's still a leap of faith to say that they're carcinogenic to humans, and as you can see in this quote, IARC does not make that leap of faith.

It says, "In the absence of adequate human data, it's reasonable for practical purposes to regard these compounds as if they presented a carcinogenic risk to humans."

What that means is, "We don't have evidence, but in order to be protective of humans, we'll assume that the evidence -- that these could be humans carcinogens." That's a reasonable decision to make, but it's not evidence that they

cause cancer.
I think the most important point here is that these are not -- these are not compounds that would be present in a smelting environment. This is not what's going on.
Q. So the first point we made, the materials that are being talked about here are forms of lead that are not at issue in this case?
A. As far as I understand.
Q. And the second point would be that what this monograph shows is that those materials that aren't at issue in this case cause cancer to mice and rats, right?
A. Right.
Q. And is there any basis for extrapolating from that to a conclusion that people who live in this class area near the smelter are at a substantially or significantly increased risk of any kind of cancer?
A. No. That's not a reasonable conclusion from this -- from this scientific evidence.
Q. Now, have people studied -- not rats and mice, but have people looked at whether there is human evidence that exposure to lead causes

cancer?
A. Oh, yeah, there's a big body of literature on that.
Q. And has -- has anybody made an effort to go through that body of literature on the human evidence and summarize it and say, "Is this something that causes cancer" or "Is it not?"
A. Well, yes. First off, IARC has done that. There's a whole book. That's what this book is about that just came out earlier this year. It's exactly aimed at that, and IARC's conclusion was "Evidence is not adequate in humans, we're not going to put it in Category 1, the evidence is adequate in animals, and it's based on exactly what's on the slide here, and we're going to put it in Category 2A."
So the evidence in humans is not adequate.
Q. Have you looked at the literature, including a study by -- report by a gentleman named Kyle Steenland and Paolo Boffetta called "Lead and Cancer in Humans, Where Are We Now?"
A. Yes.
Q. Is this one of the things you read in

evaluating Doctor Werntz's opinion that lead causes cancer in humans?
A. Yes.
Q. And what is this report?
A. Okay. Kyle Steenland, I know Doctor Steenland, he's on the faculty at Emory now. He used to be at NIOSH. He's a statistician, and Paolo Boffetta is a physician. He's in charge of the environmental epidemiology unit at IARC in France, and a very talented scientist, they both are.
They tried to summarize -- or did summarize -- the epidemiologic evidence regarding lead and cancer in humans, and this was published in the American Journal of Industrial Medicine in 2000.
So it's seven years old. The IARC publication is newer and seeks to do roughly the same thing. But yeah, this is a well-written summary of the evidence, in my opinion.
Q. And what was their conclusion about whether there is a risk of cancer in humans from exposure to lead?
A. Well, I don't know if you can blow it up,

but their conclusion is right on the screen.
THE WITNESS: Actually, stay on the first page, if you would.
A. Can you make that larger?
Q. Sure. Where do you want --
A. Where it says "Conclusion." Right down -- right there. Yeah, that's it. Okay. So their conclusion is: "Overall, there is only weak evidence associating lead with cancer; the most likely candidates are lung cancer, stomach cancer and gliomas." Gliomas are brain tumors.
Okay, so it's not that there's no evidence, but it's weak evidence.
Okay. And then if we could go to their conclusion at the end of the paper, right there, maybe you could blow that up.
Okay. And so here -- here's how they conclude their paper: "Despite the fact that lead is one of the earliest recognized occupational toxins," and you know, lead's been around for thousands of years, abdomen and we know a tremendous amount about its toxicity.
"There exists relative few studies of cancer among lead-exposed workers with well-

documented high exposures." And of course, these are the most informative studies for identifying lead causes cancer.

They identify eight principal studies with well-documented high exposures. "The evidence is suggestive of an association with lung cancer and stomach cancer but remains limited." One of the problems is "a possible confounding by arsenic is a concern."

In other words -- we talked about confounding -- it's possible that the associations that are observed between lead and cancer are really due to arsenic that hasn't been properly controlled for in the analysis.

So they're saying possible confounding by arsenic is a concern, in the study that the highest lung cancer, relative risk. There's weaker evidence of an association of kidney cancer and the gliomas, the brain tumors. Most of the studies did not have data on dose-response that would provide a better basis for inference than comparisons of exposed to non exposed people.

One of things that I haven't discussed is when you have a carcinogen, you expect to see that

as exposure increases, the risk of cancer increases. That's called dose response, all right? So people with low exposure, low risk; people with more exposure, higher risk; people with very high exposure, higher risks even.

We look for those responses in the studies, it's not there for lead.

So -- not to read the whole thing, the bottom line is, when you come down, their final conclusion is, there's only weak evidence and it is not clear that lead is a human carcinogen.

That, I think, was a fair assessment in 19 -- in 2000, and I think that's a fair assessment today.

Q. And did you -- did you review the studies about exposure in the area of smelters, the ones we talked about earlier, for arsenic and cadmium, and did you see in those any evidence that people who live near a smelter are at risk of cancer or -- any kind of cancer because of lead?

A. Well, it's -- it's the same set of studies we've already discussed, and we've looked at the cancer risks, and the direct answer to your question is, no, there's nothing in them that

says, "Oh, lead would lead to an increased risk of cancer."

It's the studies that we've discussed. It's not clear that there's increased risk to cancer no matter what the exposures are, whether it's arsenic or lead or cadmium or combinations of the three.

And some of those studies were lead smelter operations.

Q. So we talked about cancer risks, and now I -- what I'd like to do is switch gears and talk about risks of maladies and things other than cancer, and my first question for you on that is: Did you read Doctor Werntz's expert report, the report where he laid out the medical monitoring program that he thought was appropriate here and the risks, the noncancer risks, that he thought were -- he thought was an issue?

A. Yes.

Q. And did you notice in his report that in his report, where he was talking about what medical monitoring there should be here, that he talked about the decreased renal function and lead poisoning as the two main issues?

A. I think that's a fair summary.

Q. Now, yesterday Doctor Werntz talked about a whole lot of things that -- that might come up with cadmium or arsenic, things like Raynaud's disease and black foot disease. Are you prepared to talk about the things that Doctor Werntz identified as the reason for having medical monitoring in this case?

A. Yes.

Q. So let me ask you, as a doctor, considering whether there's a significant increased risk of kidney -- renal failure or lead poisoning, what's the first thing you would want to see in your analysis? What would you -- what would you look for first?

A. I'd want to look right away at whether these people had elevated blood lead levels, elevated blood cadmium levels, elevated excretion of cadmium and arsenic. I mean, that's the best index of whether people have been overexposed.

And it's the best way of starting to determine whether in fact they're likely to have had any damage or whether they're at any risk of damage.

Q. Before we get to that topic, let me ask you: In your teaching, do you teach risk assessment?
A. Yes, I do.
Q. And are you familiar with the concept of a risk assessment for determining whether people are at a significantly increased risk of disease?
A. Yeah, sure.
Q. When it came to the cancer risks, did Doctor Werntz rely on somebody to perform a risk assessment for him?
A. Doctor Brown did a risk assessment for cancer risks.
Q. When it comes to the noncancer risks, things other than cancer, like the Raynaud's disease and black foot disease that he mentioned yesterday, can you do a similar risk assessment for those things?
A. You certainly can.
Q. Did Doctor Brown do a risk assessment for those things?
A. No.
Q. Did Doctor Werntz?
A. No.

Q. Now, would it be appropriate, as a scientist or a doctor, to take a risk assessment for cancer and draw conclusions about whether there was a risk of noncancer diseases like renal failure?
A. No, you can't do that.
Q. Why -- why can't you do it that way?
A. Well, the calculation of risks has to do with some very specific data on what the risks are, and the risks for cancer are typically calculated using cancer slope factors, whereas these other things have different mechan -- these other health effects have different mechanisms and they typically involve calculations based on ideas that there is a threshold or a dose above which you get damage and below which you don't get damage.
So it's one of the principles in risk assessment foremost noncarcinogens that you have to identify a no-observed adverse effect level, and that exposures below that don't have any adverse effects. Exposures above, then you calculate the risk. And that wasn't done here.
You can't take a cancer risk assessment

and say, "Well, that applies to these other things" that Doctor Werntz has talked about.
Q. You mentioned a couple concepts that I'd like to have you explain for us a little in detail. The first was you mentioned dose. Is there a difference between the exposure you have in your environment, what might be in your soil or in your dust, and the dose that you get in your body?
A. Yes.
Q. What is the difference between those two things?
A. That's an important concept. We typically think of exposure as the concentration of things outside of our body, so the concentrations of arsenic in air, the concentration of cadmium in water, in other words, the things in the environment around us, that's exposure.
Dose refers to the amount in your body, and the concept is, it has to have crossed the barrier between me and the outside world, whether that barrier is the skin or the lining of my respiratory tract or the lining of my GI tract.

It has to cross the barrier to become dose.
And we measure dose routinely by measuring blood levels, measured blood leads, blood cadmium or blood arsenic, or by measuring how much we excrete, so you can measure urinary cadmium or arsenic. Sometimes we measure dose in the tissues, so you can actually measure -- for example, the lead -- you can measure lead in bone.
Okay, you can actually use a technique that shines a beam of neutrons through bone and measure how much lead is in the bone. Those are measures of dose, because they relate to what's in your body, not what's in the outside world.
And as a physician, we routinely measure internal dose of chemicals. That's part of what we're trained to do.
Q. Now, the other concept you mentioned was the concept of a threshold, and when you talk about things like the risk of renal failure due to exposure to arsenic and cadmium or -- is there some threshold below which the exposure, the dose, is not an issue, and above which you start to see problems?
A. Yeah. For example, for cadmium, there's

a very solid and substantial body of literature that establishes that there are concentrations of cadmium in the kidney above which you start to see damage and below which you do not, and that those correlate with levels of cadmium in blood.

So there's clearly a threshold for kidney damage, and it's a good thing there is, because we all get cadmium in our diet in very small amounts, and it doesn't do any damage. And so the body is able to handle small amounts without any damage, and if you get to a certain dose, then you can have damage.

Q. Now, with respect to the noncancer risks that Doctor Werntz identified, can you reach a conclusion about whether people are at a significantly increased risk of those things without knowing if you are at an exposure that puts you above that threshold?

A. No, you -- you have to have some scientific evidence that either there's exposure adequate to put you at increased risk or there's internal dose adequate to put you at increased risk. And of the two, I'd strongly prefer the internal dose, because it's much closer to the

issue of whether the tissue has been damaged.

Q. Now, on the question of decreased renal function and renal failure, one of the things that Doctor Werntz wants to monitor for here, have you considered whether the plaintiffs, the class members, are at a significantly increased risk of those things as a result of their living in the class area?

A. Of -- of decreased renal function and renal failure?

Q. Yes.

A. Well, there's no evidence that would support that conclusion.

Q. What kind of evidence would you need to see before you could reach that conclusion?

A. Well, I -- I would want to see a blood cadmium and a urine cadmium level, and ideally, I'd want to see -- when cadmium damages the kidney, it damages the part of the kidney that's responsible for concentrating the urine, and when those cells get damaged, low molecular weight protein, small proteins, are excreted in the urine, and one of those is called Beta-2 microglobulin.

A couple of others are called retinol-binding protein, and the third is N-acetylglucosamine, so RBP and NAG and Beta-2 microglobulin. It would be very easy to look for those proteins in the urine to establish whether anybody had evidence of early kidney dysfunction.

But the earliest signs of kidney damage can be detected by looking for those proteins in the urine.

Q. Is that another way of saying that you could test and get evidence before drawing the conclusion that there was a significant increased risk?

A. Well, it's not only that you can; you should.

Q. Based on the evidence -- well, let me ask it this way: Have you seen any evidence in this case of tests showing increased levels that approach the threshold and would put these people in the class area at a significantly increased risk of decreased renal function or renal failure?

A. No, I've seen no evidence of blood cadmium, urinary cadmium or urinary excretion of Beta-2 microglobulin or anything else.

Q. Now, the other area in which Doctor Werntz has proposed medical monitoring for noncancer risks is in the area of lead poisoning. Do you have a conclusion -- an opinion whether people in the area, the class area, are at a significantly increased risk of lead poisoning or effects from exposure to lead?

A. There's no evidence that they're at increased risk of lead poisoning, none at all. And that's very easy to assess.

Q. Is this again an area where before drawing the conclusion that there was a significantly increased risk where you would want to see evidence in the form of testing of levels in the bodies?

A. Yeah, sure, you'd do a blood lead test. We do them routinely.

Q. Doctor Werntz was asked yesterday, "You would expect, wouldn't you, if you took blood lead tests of people who lived in the class area and there was ongoing exposure that you say is happening, the blood leads would be elevated."

And he said, "Yes, you'd expect to see that." Do you agree that if there were elevated

levels of exposure in the class area, that you would see that in blood tests of people who lived in the class area?
A. Yes.
Q. Have you seen any evidence in this case that people who live in the class area have elevated blood lead levels?
A. No, I have not. ATSDR did a study in 1996 of children. They tested 25 children, and the average blood lead levels were entirely normal. And I think ATSDR's conclusion was there was no evidence of excessive lead exposure.
Q. So let me go back to the cancer risks and ask you, Doctor, based on your review of all the evidence, do you think the people living in the class area are at a significantly increased risk of any cancer as a result of exposure to arsenic, cadmium or lead?
A. No.
Q. And do you think the people living in the class area are at a significantly increased risk of any noncancer diseases as a result of their exposure to arsenic, cadmium or lead?
A. You know, the answer is again no. But

the real answer is that there's simply no basis for drawing that conclusion. You know, and as a scientist, if you're going to draw a conclusion, you have to have something to base it on.
And that's simply not here.
MR. GALLAGHER: Thank you, your Honor, I have nothing further.
THE COURT: Mr. -- Mr. Barr, you may inquire, if you'll fetch the microphone.
Mr. Barr, you may inquire. And if you'll be mindful of high noon, I'm sure the jury would appreciate it.
MR. BARR: Thank you, your Honor, I'll try to be fairly quick with this first step.
CROSS EXAMINATION
BY MR. BARR:
Q. Now, Doctor Garabrant, we've heard a lot about your testimony and the opinions you have to offer in this case and your review of the scientific evidence. Would you agree with me that reasonable unbiased scientists can look at a body of evidence and come to different conclusions about what that evidence shows?
A. You know, certainly there is a range of

opinions, and as we've seen for cadmium, IARC says it's a carcinogen, and other agencies don't. There is some range for disagreement.
However, I think it's important to realize that the disagreements are usually relatively small, as to whether cadmium is Category 1 or a 2A, is -- is basically everybody saying, "Well, there's some evidence, there's reason to be concerned, but it doesn't meet the standard for proof in humans," and that's sort of the range of disagreement.
Q. Well, we're going to get into this in a little bit. You testify a lot in trial. That's fair to say, you're a fairly experienced professional witness, aren't you?
A. I have testified in trials in the past.
Q. And you understand that in a trial setting, it's very common for one expert to say one thing and another expert to say something totally different.
A. I think that different people testify as to different opinions.
Q. And when a jury has to decide which one is right, issues like credibility are very

important, aren't they?
A. Well, I think the key issue for scientific testimony is whether testimony has a scientific foundation.
Q. So things like the scientists' bias or possible outside influences could affect the weight a jury may give to that testimony, couldn't it?
A. Well, I think the key issue is whether there's scientific evidence that underlies the testimony.
Q. Now, are you aware of any reason Doctor Werntz would come into this courtroom and mislead this jury about what the scientific evidence shows about arsenic, cadmium and lead?
A. I do not know Doctor Werntz, and I have no knowledge about his background.
Q. So you're not aware of any reason he would come in and do that, are you?
A. Like I said, I don't know Doctor Werntz, I don't know anything about his background.
Q. Well, you know he's a -- he's with -- affiliated with West Virginia University, right? Do you know that?

A. I do know that.
Q. He's an actual practicing physician that sees patients every day. You understand that, don't you?
A. I am aware that he's on the faculty of West Virginia as, I believe, an assistant professor or assistant clinical professor.
Q. Okay. And again, you don't have any reason to suspect that he would come in and mislead this jury because of his background or outside influences, do you?
A. Well, I've already said, I don't know him, and I don't have any information on his background.
Q. Okay. Well, let's talk a little bit about your background, while we're talking about backgrounds. Now, you already told me that you testify a lot in trial. I mean, this is something you very commonly do, isn't it?
A. I didn't say that. But I have testified in trial, and I think courts need scientists to explain complicated science so that it can be understood. And yes, I do that.
Q. I'm going to put this up on the Elmo. If

you want a copy of this, I can bring it up to you, but I think you'll be able to see it from there.
Can you read that, Doctor?
A. Yes.
Q. Or do you want your own copy?
A. No, I can read it.
Q. Okay. Now, this is a list that you prepared of legal testimony you have presented since 2002, is it not?
A. Yes.
Q. And there are, what -- how many cases, you think, on here? 40 maybe? 40 some depositions and trial testimony?
A. I don't know the number. I think it's -- it shows that I testify either in deposition -- well, I testify in deposition probably six times a year, eight times a year, and I testify in trial maybe twice a year for the past four years.
Q. Well, it's actually the past ten years that you testified in trial two to three times a year, isn't it?
A. I don't think I've been in trial twice a year for ten years, but I testify typically two to three times a year in trials.

Q. Okay. Well, hold on one second, since you -- you don't, you know, necessarily believe me on this --
A. It's not that I don't believe you, it's that I don't recall.
Q. If I show you your deposition testimony, would that maybe refresh your recollection on it?
A. If I testified to it, it's true.
Q. Okay. So you don't -- you don't disagree that if you testified in your deposition that was taken in this case that you testified two to three times a year in trial for the past ten years, you don't disagree with that, do you?
A. If that's what my testimony is, yes, I agree with it.
Q. Now, you've been testifying as an expert what, since the mid 1980s? Is that fair?
A. No, I testified once in, I think it was, 1986, and to the best of my recollection, no, I didn't testify in any consistent pattern in the next few years. But certainly for the past ten years, I have been asked to come into court to explain epidemiology to juries.
Q. Okay. And we're going to talk about that

first case you testified in in just a little bit. But would you agree with me that legal testimony or outside consulting away from your job -- legal testimony comprises 50 percent of your income?
A. That's a reasonable estimate, yes.
Q. So 50 percent of the money you take home every year is from testifying as an expert for primarily defendants, isn't it?
A. I do work as an expert consultant in litigation matters, and it does provide probably half of my total income.
Q. And excluding Workers' Compensation matters, you've testified that only once in your career have you ever testified for a plaintiff.
A. Why would you exclude the Workers' Compensation? I testified on behalf of plaintiffs many times.
Q. In other than Workers' Compensation, have you ever, in a trial setting, testified for a plaintiff other than the one time that we're going to discuss here today?
A. I've testified on behalf of plaintiffs in Workers' Compensation many, many times, and on behalf of defendants many times. If you want to

break it down into Workers' Comp versus nonWorkers' Comp, I have testified on behalf of defendants in nonWorkers' Comp almost exclusively.

Q. Well, except for one case.

A. That's correct.

Q. Now, would it be fair to say that you've testified before that you make $300,000 to $350,000 a year as an expert witness?

A. It would be fair to say that that has been my income for my consulting work related to litigation, not always as an expert witness.

Q. But it provides significant income to you, doesn't it?

A. Yes, it does.

Q. Now, you told the jury that you're board certified in, I believe it was, occupational and environmental medicine and internal medicine; isn't that right?

A. Yes.

Q. Now, isn't it true, Doctor, that in your current practice, you see less than one patient per week?

A. That would be correct. I spend most of my time now doing research.

Q. Okay. So you aren't out there as a clinician practicing medicine, treating people primarily, are you?

A. Well, I do complex evaluations. I see people who are referred because they have very complicated issues related to chemical exposures, and they take a lot of time. I typically spend two hours with a patient, and then I have to research the issue and write a report.

So I'll typically spend a half a day on a patient.

Q. Now, I want to talk to you a little bit about the first case you were ever hired as an expert, the asbestos case. Do you remember that case?

A. I do.

Q. That was a case about a gentleman who was claiming he had colorectal cancer because of his exposure to asbestos, was it not?

A. I believe that's correct.

Q. And you testified in that case that there was adequate scientific evidence that the asbestos fibers caused his colorectal cancer, didn't you?

A. Yes, I did. And that case caused me to look carefully at the evidence, and we undertook a series of research studies to see whether asbestos exposure was associated with colorectal cancer, and in fact, we published in 1992 a case control study, very nicely done case control study, where we couldn't find an association between asbestos exposure and risk of colorectal cancer.

That was published in the American Journal of Epidemiology. And then we followed that up with a meta-analysis, a summary analysis like we've been talking about that IARC does, looking at all of the published literature on asbestos exposure and colorectal cancer in which, when you added the studies up carefully, it really didn't bear out that there was clear evidence of any increased risk.

Q. So when you testified in 1986, '87, that time frame -- is that about right?

A. Somewhere in the mid '80s.

Q. -- you testified that there was adequate scientific evidence, and now you're saying you were wrong, there wasn't adequate scientific evidence?

A. At the time I testified, I believe the

evidence supported the conclusion that asbestos was associated with increased risk of colorectal cancer. After we did our own research and additional studies were published and we summarized them, I felt that the scientific evidence had changed, and I published it.

Q. And in 1986/'87, you were sworn under oath to testify to the truth, weren't you? I mean, that -- you -- you swore to tell the truth, didn't you?

A. I certainly was, and I did.

Q. But now you're saying when you testified that there was no -- that there was adequate scientific evidence, you just didn't know? And you testified to that anyway? I mean, I'm a little confused as to what you're telling the jury.

A. No, I did what good scientists do: I based my opinions on evidence. The evidence in 1986, I felt, was adequate to say it's a risk factor. Six years later -- especially after we had done what is now regarded as one of the best studies on that issue -- I felt that the evidence was not adequate to support that conclusion.

That's my whole point. Scientists base their opinions on scientific evidence. When the evidence changed, my opinion changed because the evidence had changed.

Q. And let's talk about what else changed. Then you started testifying for the defendants in asbestos cases, didn't you?

A. I was asked to work as an expert on behalf of a number of defendants in the late 1980s.

Q. You've testified for a lot -- I mean, let's go through kind of the list of defendants you've testified -- you've served as an expert for. You've served as an expert for Ford Motor Company, haven't you?

A. I have.

Q. General Motors?

A. I have.

Q. Chrysler?

A. I have.

Q. Various different asbestos manufacturers.

A. Only on the issue of colon cancer.

Q. Only on the issue of colon cancer, which is the exact thing you said there was adequate

scientific evidence of until you changed your mind.

A. Well, until the evidence changed.

Q. Okay. You've testified for Dow Chemical Company.

A. A long time ago, yes.

Q. You don't still have any relationship with Dow?

A. I have a large research study -- or I should say the University of Michigan is doing a large research study -- I'm the principal investigator on that study -- and Dow is funding that study.

Q. You don't have any other relationships with Dow other than that study?

A. No.

Q. You've never done any published articles funded by Dow on Dow products where you came out and said, "This product doesn't cause the injury that people were claiming it causes?" You've never done that?

A. I did a research study with colleagues looking at the risk of neurologic disease from a pesticide called chlorpyritos, a widely-used

pesticide, looking for effects of neurologic disease, looking at the workers who were quite heavily exposed, and we did not find evidence of neurologic damage. We published four or five articles from that research study.

Q. And correct me if I'm -- that's what's commonly known as Dursban.

A. Dursban is one of the commercial labels on it.

Q. And the EPA has banned that product because of neurotoxicity, haven't they?

A. They have restricted it from residential use; it is still widely used in agriculture.

Q. So the EPA disagreed with your study funded by Dow on a Dow product, that you know, your study that it does cause neurotoxicity.

A. No, EPA didn't disagree with our study.

Q. Well, they banned it, didn't they?

A. They restricted it so it wouldn't be used in homes. It's still widely used in agriculture.

Q. Let's talk about some others. You've testified for manufacturers of welding products, haven't you?

A. On the issue of whether welding causes

Parkinson's disease, which it does not.

Q. I'm actually very familiar with that, which you might be shocked to know, and we may get into that in a little bit. You've testified for BP Petroleum, haven't you?

A. I have on occasion, yes.

Q. BASF?

A. I don't recall. I may have on issues of diisocyanate.

Q. I'll show you that one in just a minute. And Baxter Health Care, you remember that one, don't you?

A. I remember quite clearly claims that people made -- health care workers, claiming that they had crippling allergies from wearing latex gloves and could no longer work as a result of latex glove.

Q. And your opinion was that there's no adequate scientific evidence that latex gloves causes, what, some sort of latex allergy?

A. No, that wasn't it. It's actually much more important than that. It's clear that wearing gloves causes contact dermatitis. Lots of people get glove rashes.

What the claims were about was the gloves were causing asthma and anaphylactic responses. Anaphylaxis is like when you get a bee sting and you go into cardiovascular collapse and die. And the claims was that gloves were causing those types of reactions, and our work research showed -- and in fact, we published our own analysis of data from the National Center for Health Statistics -- showing that health care workers were not at any increased risk of sensitization to the allergens in natural latex rubber and that there was simply not evidence that health care workers were suffering allergic responses to latex rubber gloves.

Q. What --

A. It was clear that they had contact dermatitis. Nobody's arguing with that point.

Q. But your essential opinion was "There's no adequate scientific evidence," correct? On whatever the issue was, there's no adequate scientific evidence. That was your opinion.

A. Well, the issue, sir, was that claims were being made that the science had proven that wearing gloves -- particularly in the health care

setting -- was causing all these serious diseases and allergic responses, and we looked carefully at the evidence, and there was not evidence to support that claim.

Q. And when -- we're probably not going to have time to get into this immediately, but you're -- the most common theme, if we can use a theme, of the testimony you give in trial when you testify for these manufacturing defendants like you've done in this case, is that there's no adequate scientific evidence, that whatever the product is or whatever the chemical is, causes the alleged injury. Isn't that true?

A. Only when it's true.

Q. Only --

A. Remember I testify on behalf of plaintiffs when it is true.

Q. You testified on behalf of a plaintiff once.

A. No, no, I've testified on behalf of plaintiffs many, many times.

Q. Okay. Well, I think the jury heard your testimony on that. So defendants can rely upon you -- I mean, it's fair to say, your testimony is

pretty predictable, isn't it? I mean, you're going to look at a situation and you're going to say, "No scientific evidence here" and you're going to come into court, and you're going to testify to a jury, "There's no adequate scientific evidence," is there?

A. Only when there's no scientific evidence.

Q. So you're saying there's no scientific evidence whatsoever that cadmium, arsenic and lead are associated with the health effects being claimed by the members of this class.

A. Well, my testimony is exactly what I gave. It's not -- can't be boiled down to that simple a statement.

Q. Well --

A. But it's quite clear that there is not evidence that the people who live in this area are at increased risk of these diseases from that smelter.

MR. BARR: Hey, Matt, can you pull up the chart real quick?

Q. Can you see that all right, Doctor?

A. Yes.

MR. BARR: Matt, can you pull up the

first one?

Q. Now, you remember this case? Duke Power case? This is the first case where you came in on an asbestos situation, and you said, "There's no adequate evidence to show asbestos causes colorectal cancer," isn't that right?

A. Of course I do, yes. The evidence had change. You could put up the one before that in 1986 where I felt the early evidence supported it, and it changed.

Q. But, you've since changed your opinion on this. Your current opinion today is "No adequate scientific evidence," correct?

A. That's what the evidence says, yes. I'm not alone in that.

Q. All right. That's fine.

MR. BARR: Will you go to the next one, Matt?

Q. You remember this case, Miles and Tri-Continental Industries about benzene?

A. I don't recall the case, but I would certainly agree with the opinion, chronic lymphocytic leukemia and chronic myelogenous leukemia are not known to be caused by benzene.

Acute myelogenous leukemia is, but this is an instance where people are putting the wrong diagnosis in.

Q. That wasn't the issue in this case, was it, it was chronic myelogenous leukemia -- it was chronic myelogenous leukemia, right?

A. CLL and CML are not known to be caused by benzene; AML is.

Q. You were paid $350.00 an hour to provide that opinion with regard to that? Is that about right?

A. I don't recall, but that's probably right.

MR. BARR: Let's go to the next case, Matt.

Q. Remember, this is a case, Dow. This is about TCA. What is TCA?

A. Trichloroethane, a commonly used degreasing solvent back in the 1980s and 1990s.

Q. Do you remember testifying for Dow in this case and saying that there's no adequate scientific evidence to show that TCA caused neurological injury?

A. To cause the neurological injuries that

were claimed. This was a case in 1991, 16 years ago.

MR. BARR: Can you go to the next one, Matt?

Q. This is the one where you testified for BASF. Do you remember when you testified about BASF about methacrylate?

A. Methacrylate.

Q. Methacrylate, I'll trust you on how to say it. Do you remember that testimony, and you said there was no adequate scientific evidence to show methacrylate caused multiple chemical sensitivity?

A. This is a fascinating issue. Multiple chemical sensitivity is a disease that doesn't exist. It doesn't exist. There was a big brouhaha about how trace levels of chemicals could be causing people to have all sorts of multi-system symptoms, and the scientific community has largely concluded that MCS is not a valid diagnosis, and it does not meet the criteria for saying that that's a real definable disease.

And back at the time methacrylate -- methacrylate is the stuff that's in typically

clear Lucite ceiling panels, clear plastic products that are very shock-resistant. It's a very durable plastic. And yeah, it was true, there was no scientific evidence that that material caused multiple chemical sensitivity.

Q. Well, I think people's stomachs are rumbling. We'll pick up with this chart as soon as we get back from lunch. Okay?

THE COURT: Thank you, sir. Ladies and gentlemen of the jury, hopefully your lunches are here, and you may go with your bailiff. We'll shoot for about 1:00 o'clock to return.

(The jury exited the courtroom.)

THE COURT: Doctor, you may step down, but again let me ask that you not discuss you testimony with anyone.

Counsel, why don't we reconvene at 12:45, please.

(A recess was taken for lunch after which the proceedings continued as follows:)

THE COURT: Be seated, please. Mr. Bailiff, you may return the jury to its box, sir.

(The jury returned to the courtroom.)

THE COURT: You may be seated.

Good afternoon, ladies and gentlemen of the jury. Let's note that all 11 of our jurors are again present before the Court and the parties and the respective counsel.

Mr. Barr, I believe the questioning is still yours, so you may continue.

MR. BARR: Thank you, your Honor.

Matt, could you put the chart back up?

BY MR. BARR:

Q. Now, Doctor Garabrant, we left talking about the testimony you gave for BASF, methacrylate? You remember where we were?

A. Yes.

Q. This is Baxter Healthcare. You remember this case. We talked about this a little by, this is dealing with latex gloves, right?

A. Yes, but that is not what my opinion was.

Q. Okay. But your whole opinion, even if it wasn't to show latex gloves caused dermatitis, your opinion was that there was no adequate evidence to show whatever the injury was that was being claimed, wasn't it?

A. The injury that was being claimed wasn't

caused by the gloves. What else could I say?
Q. So you said there was no adequate evidence, correct?
A. Well, it wasn't -- there wasn't evidence. It hadn't been caused by it.
Q. The same opinion you've given in this case, right?
A. No.
Q. No, it's not? In this case, you're saying there's adequate evidence that cadmium, arsenic and lead have affected the health of the class members?
A. I've made it very clear there's adequate evidence that arsenic causes lung cancer by inhalation; I've made it very clear that arsenic causes lung, bladder, kidney and skin when you have drinking water contaminated.
There's no question about that. I've also made it clear that cadmium can cause lung cancer by inhalation.
Q. So are you saying that the members of this class are at risk for lung cancer because of their exposure to arsenic? Is that what your testimony is?

A. No.
Q. That's what I thought. And you said there's no adequate evidence, didn't you?
A. My testimony is there's -- I have not seen evidence in this case that would establish that they're at increased risk at all.
Q. Okay. And in the Baxter Healthcare case, you were paid $400.00 an hour for your testimony; isn't that right?
A. I believe that's correct.
Q. Okay.
MR. BARR: Let's go to the next slide.
Q. You remember testifying for Crown Central Petroleum? It dealt with ground water contamination.
A. What was the name of the case? I don't remember the company.
Q. Let me pull that out for you real quick. I have Stiley (Phonetic) versus Crown Central Petroleum. You remember that?
A. I think so, yes.
Q. You remember it dealt with ground water contamination, right?
A. Well, if this was the case I remember,

this was a family that lived next door to a gas station, and I think they claimed that gasoline that had leaked in a plume from the gas station on to their property was causing things like heart disease and lung disease and skin rashes, and that's -- there's just no scientific basis for those claims.
Q. Okay. And you were again paid, what, in the neighborhood of $400.00 an hour for that testimony?
A. I don't recall, but that's probably right.
MR. BARR: Let's go to the next case.
Q. Do you remember testifying on behalf of Cooper Industries on a case dealing with community contamination?
A. I do, yes.
Q. And again, in that case, you said there was no adequate scientific evidence to show the contamination caused a cancer cluster, didn't it?
A. Well, if you're going to put my opinions up, I think we should actually get the opinions, because I don't think that what you're putting up accurately reflects my opinions. I think you're

changing them a little bit.
Q. Well, what was your opinion? Your opinion was that there was adequate scientific evidence? Was that your opinion?
A. The Cooper case, to the best of my recollection, was in 1996, 11 years ago. I -- I would have to look at the transcript to see exactly what I said. But I don't think that what you've put up is an accurate reflection of it.
Q. Well, would your opinion be that the handful of cancers that were observed in the adjacent community were not caused by the repair facility that was near them? Would that be an accurate reflection of your opinion?
A. Could I see what you're reading from?
Q. Certainly.
MR. BARR: Could I approach, your Honor?
THE COURT: Yes, sir.
Q. This is your deposition in the Baxter Healthcare cases.
A. I thought we were on Cooper.
Q. You were talking about Cooper in the case -- in this deposition, you were talking about Cooper basically.

A. So you don't have my opinions in the Cooper case.
Q. In this deposition, if you'll look at it, Doctor, you'll see you stated your opinion in this Cooper case.
A. Well, this is not what you put up. You've misstated my testimony.
Q. You said -- what was your opinion in the Cooper case?
A. I'll read it. My opinions were that the handful of cancers that were observed in the adjacent community were not caused by the repair facility that was near them.
Q. Okay.
A. And that was correct.
Q. Okay. So that's your testimony. And that accurately reflects your opinion.
A. Well, to the extent I recall that in a deposition years later, I believe it did.
Q. Okay. And if there had been adequate scientific evidence to show that that cancer cluster was caused by their exposures to contaminants from the repair facility, your opinion would have been different, wouldn't it?

A. If the facts were different, my opinion would have been different, of course.
Q. Okay.
MR. BARR: Let's go to the next case.
Q. You remember testifying on behalf of CSX dealing with degreasing solvents?
A. And the claim that they were causing irreparable brain damage, yes, I do.
Q. Okay. Is that an accurate -- is the "No adequate scientific evidence to show solvents caused central nervous damage," is that an accurate reflection of your opinion?
A. If you could show me the opinion, I'd be happy to look. I don't believe that that is.
MR. BARR: May I approach, your Honor?
THE COURT: Yes, sir.
Q. It was Trexlar versus CSX. You were also talking about that in the Baxter deposition.
A. So you don't have my opinion in the case.
Q. Were you under oath when you were talking about this -- under oath in the Baxter deposition?
A. What you've handed me was a deposition given in January of 2000 about cases that occurred years before.

Q. So that testimony is not true; is that what you're saying?
A. I was asked to recall something I had said years earlier.
Q. Okay.
A. I testified truthfully to the best of my recollection.
Q. Fair enough.
A. That I was being asked about something that I had said years earlier.
Q. Okay.
A. And I will stand by my testimony. Now let's read it.
Yeah, let's read this. This is important.
Q. Go ahead.
A. Okay. "I've next listed Smith versus Arco, a deposition in Houston, Texas. Do you recall the case?"
"I don't recall it specifically. I -- to the best of my recollection, it had to do with claims of disease of the hemologic system, in other words, either leukemia or lymphoma or myelodysplasia."

Question: "And you were retained by?"
Answer: "A defendant in that case."
Question: "And the substance of your testimony?"
"I don't recall specifically."
"All right. You've next listed Trexlar versus CSX in Cumberland, Maryland."
Q. That's the case we're talking about on the chart here, right?
A. Yes.
-- "a deposition. Do you recall that case, sir?"
Answer: "I don't recall Trexlar specifically, no."
"Do you recall by whom you were retained?"
Answer: "I believe I was retained by the law firm Whiteford, Taylor and Preston."
"And did they represent a defendant?"
"I believe they represented CSX."
Q. Okay.
A. So now you're asking me if I recall what I said about a case when I testified under oath I didn't recall exactly what I said.

Q. Go to the next page.
A. Let's keep reading then. "Do you recall the substance of opinions that you expressed in your deposition?"
Answer: "There were a couple of similar cases. The issue in those cases was the men who had worked in the CSX repair facility in Cumberland, Maryland believed that the solvents they used to remove grease and oil from the diesel electric locomotive engines had caused them to suffer central nervous system damage."
Question: "Did you agree?"
Answer: "I did not agree with that."
Question: "And what was the substance of your testimony?"
Answer: "That the solvent exposures that they had at CSX did not damage their central nervous system."
That's not what you put up there.
Q. Well, did you believe that the solvents you were exposed to, there's adequate scientific evidence to show that those solvents caused central nerve damage?
A. My opinion is exactly what I read, I

believed it did not cause it. You're not putting up what the testimony is.
Q. Let's go to the next case. You remember testifying for BP Petroleum --
A. Could you --
Q. -- in this case?
A. Could you show me the case and show me my testimony, please?
Q. Sure, we can do that. It was Rogerio versus BP America. Do you remember that?
A. I don't recall offhand. What year was it, please?
Q. You didn't tell us in your sworn testimony.
A. Was I asked?
Q. It doesn't -- it doesn't reflect it. It says you were retained by BP Petroleum. Do you remember that?
A. Well, I don't recall the case. Do you have the testimony from the case?
Q. Just answer my question.
A. I did.
Q. Do you remember being retained by BP Petroleum?

A. I do not recall -- the case was Rogerio?
Q. Yes.
A. I do not recall it. When was it?
Q. Mr. Rogerio died of multiple myeloma. You remember that?
A. I don't.
Q. And you didn't feel that that multiple myeloma was associated with his work in the petroleum injury. You don't remember that?
A. Well, I don't remember what I said in a case that was many years ago. I don't remember when the case was. I can tell you my opinion today.
Q. Okay.
A. Multiple myeloma is not caused by petroleum exposures.
Q. Based on what?
A. Based on the scientific evidence.
Q. That there's no adequate scientific evidence, right?
A. I gave you my opinion, sir.
Q. Let's go to the next case. Do you remember testifying on behalf of Ford Motor Company, GM, Chrysler, NAPA, Abex, certain other

people in the asbestos brakes litigation? Certainly you remember those cases.
A. I remember them well.
Q. And your testimony in that case was that there was no adequate scientific evidence to show asbestos-containing brakes causes mesothelioma.
A. That's not my testimony. My testimony is that there's no evidence.
Q. No evidence at all.
A. No evidence. The epidemiology show 15 things that show risks at or below 1.0 for mesothelioma among people that do brake repair work and vehicle repair work, and I'd be happy to go into that in detail with you.
Q. So that's your -- your standard testimony that you give?
A. I don't have standard testimony. I'm very proud of the work I do. I work very hard to be sure that what I say in court is based on science and it is a truthful and fair representation of the science.
Q. I mean, you have to agree that the testimony you give at trial on behalf of these large corporations is pretty much always the same.

A. No.
Q. Have you ever testified on behalf of one of these defendants that there was adequate scientific evidence to show that the injury alleged was caused by the product or the chemical? Have you ever done that?
A. I have testified truthfully on every occasion, and I have testified in cases where the claims were not supported by the science.
Q. So the answer is "No." You -- you've never offered that opinion.
A. I have testified on behalf of plaintiffs many times when I felt that plaintiffs had been injured by exposures.
Q. In the Workers' Compensation setting.
A. Yes, they're Workers' Compensation; they were injured at work.
Q. Would you agree with me that all this evidence, all this testimony we've been going through is essentially the same testimony you're offering here, that there's no adequate scientific evidence to show that arsenic, cadmium and lead are related to lung cancer, stomach cancer, kidney cancer, cognitive effects in the members of this

class?
A. You know, it's not about my opinions; it's about the science. We've looked at IARC, we looked at EPA, we looked at NTP. This is what the body of scientists around the world largely agrees to. And the fact that I happen to agree with mainstream science, I think says something about my ability to analyze the science honestly and fairly.
Q. So did I get you -- just said this is not about your opinions; is that what you just said?
A. I said it's about the science, it's not about me.
Q. So your opinions don't matter; is that what you're telling this jury?
A. I said the science matters.
Q. Okay. Now, you understand -- and I kind of want to reference back to your -- your first case that you ever testified in, the asbestos case, where you said there was adequate scientific evidence. You remember that? We talked about that.
A. Yes.
Q. And then science changed, according to

you, and you changed your mind. Remember that?
A. I did some of the original research that was published in the peer-review literature that raised issues about whether asbestos causes colon cancer.
Q. And is it your testimony to this jury that the science is not going to change -- even if your opinions are true, that the science is not going to change and it's never going to be shown that arsenic, cadmium and lead can cause the health effects being alleged in front of this jury?
A. Let me make it clear, science changes endlessly.
Q. Okay.
A. That's the whole point. That's why science succeeds so well, because it is based on evidence, and good scientists base their opinions on the evidence, and as the evidence grows and changes, good scientists revise their opinions.
So we talked about cadmium was linked to prostate cancer back when I was in public health school, and the evidence changed. Well, it's just not supported by the science.

We talked about kidney cancer and cadmium, that the science really doesn't support it. Even though there were some positive studies early on. And so that is the nature of science. It does change.
Q. So it's your testimony that this jury should just accept your and DuPont's word for it that the members of this class aren't exposed to significant risks from their exposures to arsenic, cadmium and lead, mind you, a company that's already been found negligent for exposing these people to these substances. That's your testimony, is that they should just trust you?
A. No, it's my testimony that the jury should look very carefully and very critically at the evidence, and I hope that they will do that, and I hope that they will come to a thoughtful and fair decision.
Q. Now, do you think DuPont looked at your body of testimony before they went out and hired you to come in and testify in this case?
A. I have no idea.
Q. You have no idea? You don't think that's something they would do?

A. I don't work for DuPont; I don't know.
Q. Okay. Well, let's talk a little bit about your background and what DuPont may have been able to find out about you before they hired you to come talk to this jury. You want to do that? Okay. I thought -- I'll take that as a yes.
A. I don't think my answer is going to matter on that one.
Q. You're probably right. Now, wouldn't you agree with me that one of the most significant studies you've done in your career was that dioxin study -- you've already talked about it a little bit -- in Michigan.
A. Well, we're still working on that study.
Q. Okay, so it's still ongoing.
A. I have a team of 15 people working on that. We just gave 28 papers at the dioxin conference, the international conference in Tokyo in early September.
Q. Now, is dioxin --
A. Gave 28 papers. I took 15 people to -- 14 people to Tokyo.
Q. Is dioxin a suspected carcinogen?

A. One of the dioxin compounds, 2,3,7,8 tetrachlorodibenzo-dioxin, is regarded by IARC as a Class 1 carcinogen to cause cancer in humans, or suspect.
Q. And the Dow Chemical Company paid your University 15 million dollars to go and conduct that study, didn't they?
A. 11, I believe. Not 15.
Q. So 11. So I disagree with you on that, but we'll just -- we'll take 11 for the purposes of this. Would you agree that the reason the study had to occur at all was that there was a Dow plant in the community that was emitting dioxin out into the -- and exposing the people that lived around this plant to dioxins?
A. I think it's fair to say that the study was done because of pollution in Midland at Saginaw that came from Dow, yes.
Q. Okay. And you went out and you tested blood, soil and house dust. You remember that?
A. Yes.
Q. Okay. Now, just remind me, do you know if DuPont ever did that for the members of this class? Did they ever go out and test soil, blood

and house dust?
A. I'm not aware that they ever had any opportunity to do that. I'm not aware that they did.
Q. You don't think they ever had an opportunity to go out and study the soil and the house dust of the people that lived in the area they contaminated? You don't think they ever had an opportunity to go do that?
A. I do not know. I assume that your firm has the opportunity to do that, and that would be compelling evidence if it had been done.
Q. And -- and you understand that was done in this case by the members of the class. They hired scientists to go out and study the soil, study the house dust.
A. I haven't seen the blood tests. I haven't seen the urine tests. Do they exist?
Q. So you think that the members of this class have an obligation to go out and get their blood tested before this company who negligently contaminated their community has to medically monitor them? You think they have an obligation to go out and pay for tests that this company has

caused them to go have to have?
A. If you think that they have received doses that have put them in at risk, as a physician, it is absolutely appropriate to do the blood tests and the urine tests.
Q. Well, do you --
A. They will provide the foundation for determining whether people were or were not at increased risk.
Q. Okay. You understand that's what they're asking for in this case, don't you?
A. I believe that Doctor Werntz has asked for some elements of that, and has stated -- and I can quote it from his report where he said, "Doing those tests now wouldn't help." That's wrong.
Q. And you think that's wrong.
A. I do.
Q. But he still -- his program calls for urine testing, does it not?
A. Let's read what he says.
Q. Doctor, I'm asking a very simple question. His program calls for urine testing, does it not?
A. It calls for testing for some things in

urine, but not some of the ones that would be most useful.
Q. Does it call for blood lead testing?
A. It calls for blood lead -- blood lead testing.
Q. And that's what you think these people have an obligation to go out and do before this company -- I mean, why should these people -- why should the members of the class have to come out of their own pocket for being exposed to something that they had nothing to do with?
This company put those chemicals in their community, not them. Why should they have to go out and pay for that?
A. I think that if you're going to claim that somebody's harmed you and you have the opportunity to show whether they've harmed you, it's a reasonable thing to do. A blood lead test is not a very expensive test.
Q. So you think they had that obligation, it's on them. That's your testimony.
A. I think it's a reasonable foundation, if you want to make a case that somebody's hurt you.
Q. Okay. Well, let's go back to the dioxin

thing. Let's finish your study with Dow. You found in your study high levels of dioxin in homes, soil and people, didn't you? Wasn't that one of the results of your study?
A. You'd have to bring up the results. You could go right to the web site and find it. Everything we had found is on our web site.
Q. You don't remember that? Weren't you the principal investigator?
A. I remember in exquisite detail what we found. Do you want me to go through it?
Q. I just asked you a very simple question. You found high levels of dioxins in the homes, the soil and people's blood, didn't you?
A. Okay.
Q. I mean, that's "yes" or "no." You either did or you didn't.
A. Well, a "yes" or "no" answer wouldn't characterize it accurately. Let me answer, please. Okay. We found that there was extensive soil contamination in Midland and Saginaw, and it occurred in two patterns. One pattern was to the north and northeast of the plant, and likely represented aerosol deposition from air emissions

that settled back on the community.
And we found very high levels of dioxins in the soil in that part of the city.
There was a second pattern in the river -- there's a river that runs through the plant. It's the Tittabawassee River, and the Tittabawassee River had a different pattern of contamination.
It was heavily loaded with polychlorinated dibenzofurans, not heavily loaded with the dioxins, and it's not clear where those furans came from, but they probably came from Dow's operations in the early part of the 20th century, maybe 80, 70, 60 years ago, and the soils in the flood plain of the river were highly variable, but many of them were heavily contaminated with those furans.
The household dust -- so we actually vacuum cleaned -- or vacuumed people's homes. The household dust showed elevated levels of dioxins, and they correlated with what was in the soil outside the home.
We tested people's blood. The blood levels showed virtually no relationship to what

was in the soil or what was in the household dust. So even though the soil was contaminated and the dust was contaminated, there did not seem to be any direct pathway from the contaminated soil and contaminated dust into people's blood.
I can go on --
Q. I mean --
A. -- for as many hours as you'd like to hear.
Q. If you want to waste the jury's time, that's up to you.
A. I don't, sir.
Q. I asked you a very simple "yes" or "no" question, and the answer is "yes." You found high levels in the soil, you found high levels in the dust.
A. But not in the blood.
Q. Fine. Now, in your study, did you do any research on the potential health effects of dioxin and the high levels in the soil? Was that part of your study?
A. No. And this is the critical issue. Because before you're going to study health effects, you have to establish that exposure has

led to an increased dose. In other words, it doesn't make sense to go out and do a health effects study until you've established that the chemical in the environment is actually getting into the people's bodies. It doesn't make sense to do it. It's nonsense.

And so our study was designed to see whether the chemicals in the environment got into people's bodies, and by what pathways they got in. Because that's the critical issue.

If you want to do something about the exposure, you have to figure out how it gets from the environment into people's bodies. And then you can take effective action to stop the exposure.

What you don't do is say, "Oh, we'll go out and study health effects" before you can establish that something in the environment even gets into people's bodies. Because it's nonsense.

You've got to go step by step, and you have to establish the first thing first, and then if you find that the body burdens are increased and you've figured out which pathways, then it makes sense to think about health studies.

And of course that's what we did.

Q. In this case that we're presently here for today, did DuPont do any of these tests that you just talked about, any? One? Just one?

A. I'm not aware that DuPont or anyone else has done tests except for the ATSDR who came in and tested children and found nothing.

Q. You're not aware that plaintiffs -- that the scientists that worked for the members of this class went out, tested the soil, house dust and air in the class area. You're not aware of that.

A. I am aware of the data that underlies Doctor Brown's risk assessment model, and I'm aware that there's been one blood test of a plaintiff in the class.

MR. BARR: Your Honor, can we approach real quick?

THE COURT: Yes, please.

(Counsel approached the bench and the following proceedings were had out of the hearing of the jury:)

MR. BARR: He was specifically instructed not to get into that blood test, and he's --

MR. GALLAGHER: He challenged him by

saying, there has been no testing in this class on blood. He asked him.

THE COURT: But the witness was instructed by the Court, through counsel, not to answer that, so I'm going to instruct the jury that they're to disregard it, that the witness answered inappropriately, contrary to the Court's ruling, and they're not to consider.

MR. GALLAGHER: I think it was invited, but if that's the Court's ruling --

(Counsel returned to counsel table and the proceedings continued in the presence and hearing of the jury as follows:)

THE COURT: Ladies and gentlemen of the jury, there's been an appropriate objection as to the last answer of the witness which violated a prior Court order, so the Court is striking that answer from the record and instructing the jury not to consider it in any respect in arriving at their decision.

So you may continue, sir.

BY MR. BARR:

Q. Going back to the dioxin study, would it be fair to say that you went so far as

discouraging people that lived around the Dow plant from going to other doctors and getting their blood tested because you thought the Dow study would be conclusive?

A. No.

Q. You did not do that.

A. I did not.

Q. Now Doctor, it's fairly common for you to do corporate sponsored research, isn't it?

A. Well, the sponsors of my research include the National Cancer Institute, the National Institutes of Health, the National Institute for Environmental Health Sciences, NIOSH, the State of California, the American Cancer Society, the United Auto Workers, Ford Motor Company National Joint Committee, the Dow Chemical Company, the Rohm and Haas Chemical Company, private foundations. That's a few.

Q. I want to show you a few of the studies you've done, just a couple. Let's start with --

MR. BARR: Matt, can you pull up -- I don't know that you have this. I'll just use the Elmo.

Can I approach, your Honor?

THE COURT: Yes, sir.
Q. Do you recognize this study, Doctor Garabrant?
A. I do.
Q. This is a study that you conducted, is it not? See your name there?
A. I was a co-investigator on this, yes.
Q. And this was about scleroderma and solvent exposure among women. You remember that?
A. I do.
Q. Do you know who this was -- who paid for this study?
A. Yes, the Dow Corning Corporation.
Q. So that's just one study you've done that was on behalf of Dow Corning, right?
A. That was a study done on behalf of the Dow Corning -- actually, you know what? That's not true. It says right in the study, "Supported by grants from the Halogenated Solvents Industry Alliance, the Dow Corning Corporation and the National Institutes of Health."
Q. So this is one example of studies you've done on behalf of the Dow Chemical Corporation, similar to your -- not in the same realm, but

similar to the dioxin study, where Dow went out and paid you or -- you know, your University, to go out and conduct a study, didn't it?
A. No, you've mixed up two companies. This is the Dow Corning Corporation; that is not the Dow Chemical Company. It's a different company.
Q. So they're not related at all?
A. I'm not sure of the relationship. To the extent I know, I believe Dow Corning was formed during or just after World War II as a joint interest between Dow Chemical and the Corning Glass Company to explore new findings in the silica (Phonetic) chemistry which had evolved during World War II.
And I'm not an expert in corporate finance, but I'm under the impression that this was created as a separate corporate entity.
Q. Okay. The next study I want to show that you've done, this was the study on Dursban that we previously talked about.
MR. BARR: May I approach, your Honor?
THE COURT: Yes.
Q. Do you recognize this study, Doctor Garabrant?

A. I sure do.
Q. This is the study we talked about earlier?
A. Yes, we did a study of chlorpyrifos, which is Dursban, and we did this with funding from the Dow Chemical Company which is stated on page 366.
Q. There's your name, right? You see that right on the front.
A. Yes.
Q. And this is "The Effects of Occupational Exposure to" -- how do you say that?
A. Chlorpyrifos.
Q. And that's the same as Dursban, isn't it?
A. Chlorpyrifos is the chemical name; Dursban is the market name.
Q. And what was your conclusion in this study?
A. It's written right on the first page.
Q. Where's it at? I see it. It was your conclusion that -- here, I'll read it. Was your conclusion that "Chronic chlorpyrifos exposure produced no clinical evidence of cortical, pyramidal tract, extrapyramidal, or other CNS

dysfunction among chlorpyrifos subjects compared with referents, either at baseline or after one year of additional exposure."
A. That's exactly what we wrote, yes.
Q. And this is a study you did for the Dow Chemical Company, correct?
A. That's correct.
Q. And in this study, you even listed -- you see down there at the acknowledgements? It says, "Some of the authors have at times been retained as consultants or served as expert witnesses in litigation for firms or companies including Dow Chemical, Agrosciences, concerned the manufacture or use of insecticides." Do you see that?
A. Yes. That's absolutely appropriate to put in, a full disclosure.
Q. So you published a study having to do with Dursban, saying that there are no central nervous system effects, and that study was paid for by Dow Chemical, right?
A. We studied Dow employees who made Dursban and who were heavily exposed, and the study was funded by Dow, and they gave us access to their employees. The study was done by the University

of Michigan with all of the appropriate protections for human subjects in research and protections of confidentiality as well, and I'm very proud of the work. Yes, it's good science.

MR. BARR: May I approach, your Honor?

THE COURT: Yes, sir.

Q. Do you recognize this, Doctor Garabrant?

A. I don't know that I've seen this exact document in the past, but I'm certainly aware of the EPA's actions to restrict use of Dursban in homes and consumer use in gardens.

Q. And they did that despite the fact that you published a study that said there are no central nervous system effects, right?

A. Well, I think you've got your dates out of order.

Q. Your study actually came after this.

A. Right. Our study came out in 2004, so it's hard to say that the EPA knew the results of our study before we'd done it.

Q. Well, did the EPA -- have they since lifted the ban?

A. I believe they have not.

Q. Now, Doctor Garabrant -- give me one

second here to get organized. You also work with research centers that are heavily funded by corporations, don't you?

A. I don't know what you're referring to. I'd have to say not to my knowledge, no.

Q. You don't -- you don't serve as an advisor to the Mickey Leland National Air Toxic Research Center?

A. The Mickey Leland Center is a Congressionally-mandated center to study air pollution and it is funded by the EPA. It has a line item in the EPA budget. It's named after representative Mickey Leland who was a strong advocate for control of urban air pollution and who was killed in that airline flight -- oh, I want to say -- back in Serbia during the Clinton administration.

The Center was named after him because of his strong advocacy on behalf of cleaning up urban air.

Q. And are you telling this jury that there aren't corporate sponsors to the Mickey Leland National Urban Air Toxic Research Center?

A. No, I'm not telling them that. The

Mickey Leland Center, which is correctly called the National Urban Air Toxics Research Center, gets its money from the EPA, and they also get donations from industries that are interested in and concerned about urban air pollution.

MR. BARR: May I approach, your Honor?

THE COURT: Yes, sir.

Q. Not -- let me put this here so the jury can see it. You see that this is about the center, the Mickey Leland National Urban Air Toxics Research Center. Do you see that?

A. Yes. Could you move it up a little bit?

Q. Is that better? Let me try and zoom out, see if that helps.

A. That's too far. Let's go back in.

Q. You tell me where you want me to stop.

A. About right there -- whoa, that's good.

Q. Now, if you look -- obviously you're going to want to point me to the part where this is authorized by the U.S. Congress.

A. And funded by the Congress, too.

Q. Well, let's go to the third page -- well, let's start with the second page. You see there Under Current Scientific Advisory Panel Members?

A. I do, yes.

Q. It lists your name, doesn't it, David H. Garabrant. That's you, isn't it?

A. Yes.

Q. And then if we go to the third page, it says as the -- that's a short form for the center?

A. The National Urban Air Toxics Research Center, that's correct.

Q. "Moves towards implementing its mission and achieving its important goals, substantial additional public and private support will be needed." Do you see that?

A. Yes, I do. Let's look up at the first paragraph at its mission.

Q. We'll get there -- I'll go where you want to go once I get through with this. Okay? Is that fair?

A. Yeah, sure.

Q. And it says, "More industrial firms, as well as foundations and other sources, will be asked to take part in the funding of the Center's complex research." Do you see that?

A. Yeah, I think this is great, get industries to pay for air pollution research.

Q. It says, "Past and present corporate and other sponsors are: Ashland Chemical." Do you see that?
A. I see it, yes.
Q. You see DuPont down there?
A. I do.
Q. Okay. You see companies like Goodyear, Georgia-Pacific, Exxon Chemical, Sunoco, Union Carbide --
A. Yeah.
Q. So this study -- this research center is heavily funded by industry, isn't it?
A. Well, it's heavily funded by the EPA, and they're asking companies to assist in paying for air pollution research. I think that's -- that's a worthy mission.
Q. I'm going to move on a little bit, Doctor. Would you agree with me that -- I'm not the first person to criticize you for your ties to the industries, am I -- to ties to corporate industry, am I?
A. I've been criticized for doing work for the UAW too. I mean, I work in a realm that is highly contentious. When you start to research

the health effects of chemicals, it's highly contentious, and people criticize whatever you do.
Yes, I've been criticized.
I've also been the recipient of research awards for outstanding research too.
Q. Okay. You remember being nominated to the EPA Science Advisory Board on ethylene oxide?
A. Yes, I do.
Q. And you remember a group of public health advocates objecting to you being on the panel?
A. I'm not sure that would characterize correctly who they were, but --
Q. You don't think that would -- okay, well, I'll show you that in a second. But they were concerned that you were a corporate insider, weren't they?
A. Why don't we read exactly what they said?
Q. So you don't remember if they were concerned that you had financial conflicts that would prevent you from being an unbiased member of the science advisory panel on ethylene oxide? You don't remember that? I mean, seems like to me that that's something that people would remember.
A. I saw their letter approximately two

years ago. I didn't memorize it. But if we could look at it, I would be pleased.
Q. We'll -- I'll show it to you in a minute here.
A. Okay.
Q. Would you agree with me that the Dow Chemical Company is the number one producer of ethylene oxide?
A. I have no idea.
Q. You have no idea.
A. -- what chemical company is the number one producer of ethylene oxide.
Q. Well, let's show this to you.
MR. BARR: May I approach, your Honor?
THE COURT: Yes, sir.
Q. You recognize this, Doctor Garabrant?
A. Yes.
Q. Let's read what this says. This is June 16, 2006, right? You see that on the front?
A. I do.
Q. And it says, "Re," regarding, "Invitation for comments on the short list candidates for the Ethylene Oxide Review Panel, EPA Science Advisory Board." Do you see that?

A. Yes.
Q. And it says, "This letter is supported by the following worker and public health advocates." Do you see that?
A. I see that's what it says, yes.
Q. And it's supported by Jennifer Sass with the Natural Resources Defense Council. You see that?
A. Yes.
Q. By an Amanda Hawes from the Toxics Chair WORKSAFE. You see that?
A. Yes, and my experience is Ms. Hawes -- she's an attorney in the Bay area.
Q. Okay. You see that it's supported by Kathleen Burns, the director of Science Core. You see that?
A. I can read it very well. I have no idea who some of them people are, to be honest.
Q. Well, they knew who you were, didn't they?
A. I'm sorry?
Q. They knew who you were.
A. I have no idea --
Q. Well, we'll get --

A. -- what they knew about me.
Q. We'll get to that in a minute. It's sponsored by a Bill Borwegen, Occupational Health and Safety Director, Service Employees International Union. You see that?
A. Yes.
Q. By Michael Jacobson, Center for Science in the Public Interest?
A. Yes.
Q. By Mikal Harbut, Chief Center for Occupational and Environmental Medicine at Wayne State University. You see that?
A. I see that. Although what does "For ID only" mean? I don't know what that applies to.
Q. Well, he certainly signed his name, didn't he? He allowed them to use his name on there, didn't he?
A. I don't know.
Q. You see it says, "Jane Houlihan," there's a Joe DiGangi, Environmental Health Fund, David Michaels, Department of Environmental Health at George Washington University, David LeGrande? You see that -- see all the people listed here?
A. I see those names, yes.

Q. Okay. Now, let's go to the second page. It says, "Overview of ethylene oxide toxicity: Ethylene oxide is a known carcinogen." Do you see that?
A. I do.
Q. Now, let's go here when they start talking about their comments. "Five of the nominees" -- I'm on the third page, if you can follow. "Five of the nominees have financial conflicts: Gargas, Garabrant." You see that?
A. I see it.
Q. Then it lists four more nominees, and it says, "These nine individuals should not be selected for expert committee since several represent a coordinated perspective, several have overlapping expertise, and since at least five, to our knowledge have a direct financial relationship with the ethylene oxide and petrochemical industry."
Do you see that?
A. I see it.
Q. You had such a tie with Dow Chemical Company, didn't you?
A. Well, we've already discussed that I have

done research under contract between the University of Michigan and Dow. I need to make it clear to the jury, I don't get any money from doing that. I'm a tenured professor. My salary is paid no matter what I do.
If I bring in a research grant, that doesn't change my salary, I don't get anything. So it doesn't matter to me if it comes from the National Cancer Institute, United Auto Workers, Ford Motor Company National Committee or the Dow Chemical Company, if it's a good research project, I will pursue it, but I get no financial reward for doing that.
Q. Well, the only financial reward you get, as the jury's already seen, is it comprises 50 percent of your total income.
A. No, no --
Q. Testifying for people like Dow Chemical.
A. Unh-unh, unh-unh, you just mixed up two separate things. The research money that comes to the University of Michigan does not come to me, not a penny of it.
Q. Okay.
A. Okay?

Q. Well, let's go to page 5 of this, and let's see what they wrote about your financial conflicts. You see that where it says "Garabrant and Schnatter" -- is that how you say that, do you know?
A. I do not know Doctor Schnatter. I have seen his name in the peer-review literature. I've never met him. It says, "Garabrant and Schnatter have financial conflicts. David Garabrant has a history of industry consulting and has served as an expert witness to corporate defendants.
Between 2001-2005, Garabrant was personally paid over $264,800 for his consulting for Ford and General Motors in asbestos litigation. He is now conducting a 15 million dollars stud on dioxin hazards funded by Dow Chemical. Since Dow Chemical is the world's largest producer of ethylene oxide, this constitutes a direct financial conflict." See that?
A. I see it.
Q. And then it goes on to say, "Garabrant's financial relationship is not disclosed on the EPA web site, an apparent violation of the law." Do

you see that?
A. That is -- first off, I have no knowledge of how much or whether the Dow Chemical Company produces ethylene oxide. I was surprised to read that. And the fact that I didn't disclose it, I don't know how I could have disclosed something I didn't know.
Q. You didn't know that you had a financial relationship?
A. Well, I don't receive money from the Dow Chemical Company. I didn't know I had a financial relationship with them.
Q. And this group thought that your failure to disclose that was a violation of the law.
A. Since I don't get money from them, I didn't know I had a financial relationship.
Q. Now, ultimately, Doctor, you weren't appointed to that advisory board, were you?
A. I was not.
Q. Now, I want to move now into your opinions regarding this case and the class area and what your opinions are, okay? Now, you hold the opinion, if I'm correct, that there's been no significant exposure to the members of the class

area from the DuPont plant. Is that your opinion?
A. I hold the opinion that Doctor Brown's models show relatively low level mild exposures --
Q. Now --
A. -- the opinion that there is no evidence of internal dose to the plaintiffs.
Q. Other than Doctor Brown's data, which the members of the class paid for, not DuPont -- other than his data, what do you know about the exposures of these members of the class to cadmium, arsenic and lead? You don't know anything, do you?
A. I know they live in a community in which there is a former smelter, and I know that studies of similar communities around the world have not demonstrated excess risks of cancer.
Q. Okay. Do you -- and the jury's heard this, so I don't want to go through all the documents on this, but you apparently don't know this -- are you aware that this smelter released 5600 pounds of dust a day? Is that something you knew?
A. I do not recall those numbers. I don't know the source of that. Is there a document you

want to show me?
Q. I'm just asking you if that's something you knew before you came in here and told this jury that the members of this class have not been significantly exposed to cadmium and arsenic and lead. Did you know that?
A. I -- I've already answered the question. I'm not aware of that number, but -- what -- what document are we referring to? Could I see it?
Q. I'm not going to waste this jury's time with it. They've seen this stuff a lot. The important point is, there's a lot of stuff you don't know, isn't there?
A. Well, sir, if you're going to ask me questions about documents I haven't seen, I think it would be fair to let me see them.
Q. Well, I'm going to show you some documents here in a little bit. When did you actually arrive in town to testify in this trial?
A. Early in the morning of Tuesday morning, in the wee hours.
Q. Okay. And it's now Wednesday, right?
A. Yes.
Q. Okay. So you've been here today. Is

this the first time you've ever come to Clarksburg?
A. To the best of my recollection, yes.
Q. So you've never gone out and looked at the facility and the class area, have you?
A. I'd actually very much like to do that.
Q. Well, you're saying you haven't had the chance or the opportunity to go do that?
A. I have not had the opportunity to do that.
Q. How long have you --
A. I'll do that, actually, when we finish today. I'd very much like to see it.
Q. Oh, after you testify and tell this jury that there's no significant exposure, then you'll go look?
A. I'd still like to see the site.
Q. Well, how long have you been retained as an expert in this case?
A. I'm not sure I recall. I think perhaps nine months, could be a year. I'm not sure.
Q. And in that nine months to a year, you've never had the opportunity to come to Clarksburg and talk to the members of this class about their

exposures to cadmium and arsenic and lead from the smelter.
A. I don't know how I could have spoken with them. I don't know that I'm allowed to talk with plaintiffs in litigation when I've been retained by the other side. Am I allowed to do that?
MR. BARR: Your Honor, I don't have any further questions for Doctor Garabrant.
THE COURT: Any follow-up, counsel?
MR. GALLAGHER: Yes, your Honor.
THE COURT: And if you'll share the microphone, please.
MR. GALLAGHER: Your Honor, there is one thing that I need to approach about.
THE COURT: Please.
(Counsel approached the bench and the following proceedings were had out of the hearing of the jury:)
MR. GALLAGHER: Your Honor, we're back to the topic of Lenora Perrine's blood. He asked him, "What do you know about exposures in the class area to arsenic, cadmium and lead? You don't know anything, do you?"
He -- he impeached him with his lack of

knowledge, when he has knowledge. You can't do that. He's -- you can't move in limine to keep something out and then ask a question that is designed to impeach somebody when they can't answer it. He's opened the door to that, and I should be allowed to --
THE COURT: But didn't you ask the exact same question? "There isn't any evidence that you've seen other than the ATSDR study concerning this," and I think it's the same question. So the ruling of the Court remains the same.
So you may continue.
(Counsel returned to counsel table and the proceedings continued in the presence and hearing of the jury as follows:)
MR. GALLAGHER: Your Honor, there's nothing I need to ask.
THE COURT: May this witness be excused?
MR. BARR: Yes, your Honor.
THE COURT: Sir, you are excused. Thank you for your time.
THE WITNESS: Thank you, your Honor.
THE COURT: Mr. Thomas, you may call your next witness.

MR. THOMAS: Thank you, your Honor. DuPont calls Kelly Nelson.
THE COURT: If you'll come forward, sir, please. Could counsel approach, please?
(Counsel approached the bench and the following proceedings were had out of the hearing of the jury:)
THE COURT: I'm just asking -- it's my general understanding -- you correct me -- that Doctor Nelson's being called as a fact witness. Is that correct?
MR. THOMAS: No, that's not correct. He's going to be called as an expert in primary care, treatment of patients in the area.
THE COURT: Okay. I just kind of remember our prior conversations, so -- okay, that's fine. Thank you.
(Counsel returned to counsel table and the proceedings continued in the presence and hearing of the jury as follows:)
THE COURT: Sir, if you'll raise your right hand.
(The witness was sworn.)
THE COURT: You may have a seat up in the

witness stand, please, sir.

K E L L Y N E L S O N

was called as a witness by the Defendant, and having been first duly sworn, testified as follows:

DIRECT EXAMINATION

BY MR. THOMAS:
Q. State your name, please.
A. Kelly R. Nelson.
Q. And it's Doctor Nelson, isn't it?
A. That's correct, yes, sir.
Q. Doctor, where do you live?
A. 46 Juniper Lane, Bridgeport, West Virginia, right here in Harrison County.
Q. And where do you work?
A. A place called Medbrook.
Q. Would you tell the jury what Medbrook is?
A. Medbrook is a large group practice located in Bridgeport, 1370 Johnson Avenue. We're a group of family physicians. We do primary care, continuity care, do some occupational medicine. We also have a large urgent care practice.
Q. And what's your role currently at Medbrook?