

IN THE CIRCUIT COURT OF HARRISON COUNTY
WEST VIRGINIA

LENORA PERRINE, CAROLYN HOLBERT,
WAUNONA MESSINGER, REBECCA 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

Page 4192 1 2 3 4 5 6 Proceedings had in the above-entitled 7 action before the Honorable Thomas S. Bedell, and 8 a jury, in the Harrison County Courthouse, 9 Clarksburg, West Virginia, on the 3rd day of 10 October, 2007. 11 12 13 14 15 REALTIME REPORTERS, LLC TERESA S. EVANS, RMR, CRR 16 108 Cedar Ridge Ripley, WV 25271 17 (304) 372-8069 1-800-805-8079 18 19 20 21 22 23 24	Page 4194 1 APPEARANCES: 2 3 APPEARING FOR THE DEFENDANT DuPONT: 4 David Thomas, Esquire Stephanie D. Thacker, Esquire ALLEN GUTHRIE McHUGH & THOMAS 5 P. O. Box 3394 Charleston, WV 25333-3394 6 Jeffrey A. Hall, Esquire 7 Andrew C. Baak, Esquire John S. Phillips, Esquire 8 BARTLIT BECK HERMAN PALENCHAR & SCOTT Courthouse Place 9 54 West Hubbard Street Chicago, IL 60610 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24
Page 4193 1 APPEARANCES: 2 3 APPEARING FOR THE PLAINTIFFS: 4 Farrest J. Taylor, Esquire Keith Givens, Esquire Angela Mason, Esquire 5 THE COCHRAN FIRM 163 W. Main Street 6 Dothan, AL 36302 Mike Papantonio, Esquire 7 Steven A. Medina, Esquire Neil E. McWilliams, Esquire 8 Brian Barr, Esquire 9 LEVIN PAPANTONIO THOMAS MITCHELL ECHSNER & PROCTOR 10 316 S. Baylen Street, Suite 400 Pensacola, FL 32502 11 Gerald E. Jones, Esquire 12 Perry Jones, Esquire WEST & JONES 13 360 Washington Avenue Clarksburg, WV 26302 14 Gary W. Rich, Esquire 15 Brock, Reed & Wade Building 212 High Street, Suite 223 16 Morgantown, WV 26505 17 18 19 20 21 22 23 24	Page 4195 1 I N D E X 2 3 DEFENDANT'S WITNESSES DIRECT CROSS REDIRECT 4 David Garabrant, M.D. 4208 4301 5 Kelly Nelson, M.D. 4375 4434 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24

Page 4196 1 PROCEEDINGS 2 THE COURT: Be seated, please. Let's go 3 on the record. Comes on for proceedings at the 4 present time a matter encaptioned Lenora Perrine, 5 et al versus E.I. DuPont DeNemours & Company, et 6 al, Defendants. The matter bears Case No. 7 04-C-296-2. 8 The Court would note that today is 9 Wednesday, the 3rd day of October, 2007. These 10 matters come on by way of further proceedings of 11 the jury proceedings which recessed at the close 12 of the day on the 2nd day of October, 2007. 13 Will counsel be so kind as to note their 14 appearance for the record and further note the 15 presence or the absence of their respective 16 clients or others whose attendance they care to 17 note. 18 Mr. Taylor? 19 MR. TAYLOR: Thank you, your Honor. With 20 me today is Joseph Lane, Gary Rich, Angela Mason, 21 our client, Lenora Perrine, and Brian Barr. 22 THE COURT: Okay. Mr. Hall? 23 MR. HALL: Good morning, your Honor, Jeff 24 Hall for DuPont. With me is Brian Prestes, also	Page 4198 1 like to focus on three points, and with respect to 2 the others, rest on the paper that we have filed 3 this morning. 4 Under the first element of Bower, the 5 plaintiff must show significant exposure. Bower 6 makes clear that a significant exposure is what is 7 required, not mere exposure. Doctor Werntz 8 yesterday testified that he merely assumed 9 exposure. He did no tests. 10 In fact, he testified he had never met a 11 single member of the class and didn't even know 12 how many class representatives there were. Doctor 13 Werntz testified that he relied on Doctor Brown, 14 who is a soil scientist, not a medical doctor. 15 Doctor Brown is not an expert in cancer 16 causation, and Doctor Brown has no basis for 17 opining on the levels of exposure that are 18 sufficient to justify or necessitate medical 19 monitoring. 20 With respect to Doctor Brown's testimony 21 and the insufficiency of that testimony, we'd rely 22 and incorporate our Phase 1 judgment as a matter 23 of law briefing. 24 But just to provide two specific
Page 4197 1 Shawn Gallagher, who will be handling the 2 examination of our first witness, David Thomas and 3 Mr. Yalvigi. 4 THE COURT: Counsel, let me note that 5 appropriate, as far as the timing goes, that the 6 Defendant DuPont moved for judgment as a matter of 7 law at the close of the plaintiffs' case yesterday 8 on the medical monitoring phase and further has 9 submitted their written motion for judgment as a 10 matter of law on medical monitoring or, in the 11 alternative, to decertify the class. 12 Let me ask counsel who will again address 13 that to approach the bench and we'll argue it at 14 side bar. 15 (Counsel approached the bench and the 16 following proceedings were had out of the hearing 17 of the audience:) 18 MR. PRESTES: Good morning, Judge, Brian 19 Prestes on behalf of DuPont. DuPont moves for 20 judgment as a matter of law, or in the 21 alternative, to decertify the class. 22 There is no sufficient evidentiary basis 23 for the jury to find for the plaintiffs on a 24 number of the critical elements of Bower. I'd	Page 4199 1 examples, Doctor Brown conducted no risk 2 assessment on lead, and his contour maps on lead 3 show extremely low levels of lead, below the 4 levels that Doctor Brown himself identified as 5 clean. 6 Moreover, the only tests of presence of 7 lead in the bodies of anyone then introduced in 8 this case -- the ATSDR tests show no significant 9 exposure to lead. So on that point, we believe 10 that DuPont is entitled to judgment as a matter of 11 law. 12 Two other points quickly, Judge. The 13 first is that we believe the plaintiffs have 14 failed to show an increased risk of disease. 15 That's Bower Element 4. The plaintiffs must show 16 a significantly increased risk of disease caused 17 by the exposure they've alleged. 18 Again on this point, Doctor Werntz, the 19 plaintiffs' only Phase 2 witness, provided only 20 nonspecific testimony, not testimony relating to 21 anything having to do with this class. 22 Again he relied on Brown, and I'd 23 incorporate the argument I just made about the 24 insufficiency of Doctor Brown's testimony to

Page 4200 1 support the opinion that the plaintiffs are at an 2 increased risk of disease, especially as it 3 relates to lead for which Doctor Brown conducted 4 no risk assessment whatsoever. 5 My final point relates to the fifth 6 element of Bower, which is that the plaintiffs 7 must show medical testing is reasonably 8 necessary. And the way Bower has defined that is 9 reasonably necessary testing is testing that a 10 qualified physician would prescribe. 11 First off, many of the diseases that 12 Doctor Werntz described in his testimony yesterday 13 are diseases for which Doctor Werntz himself 14 concedes medical monitoring is not appropriate. 15 Doctor Werntz went through a laundry list 16 of perhaps 20 diseases, but in the end, with the 17 assistance of one of plaintiffs' demonstratives, 18 testified there were only five cancers and three 19 noncancer diseases, and so DuPont submits that to 20 the extent any of those other diseases are in 21 play, they cannot form the basis of a medical 22 monitoring claim. 23 They may go to show - if plaintiffs' 24 evidence is taken as correct - that lead or	Page 4202 1 experimental procedure based on a, quote, 2 "uncorroborated premise." 3 And finally, one other specific example, 4 which is kidney and stomach cancer screening. 5 Again, Doctor Werntz conceded that, quote, "No 6 organization recommends screening for kidney 7 cancer and stomach cancer." And to the extent 8 that Doctor Werntz's opinion itself is not 9 supported by any facts and to the extent that it's 10 contradicted by the vast weight of science -- of 11 the medical consensus on Doctor Werntz's own view, 12 we believe that DuPont is entitled to a judgment 13 as a matter of law on those points. 14 With that, your Honor, and with respect 15 to the other points in our paper, DuPont is 16 content to rest on its papers. Thank you. 17 THE COURT: Ms. Mason? 18 MS. MASON: The plaintiffs have 19 established every one of the elements that is 20 required in Bower through Doctor Werntz's 21 testimony. The defendant's motion is based 22 primarily on trying to relitigate Doctor Brown's 23 conclusion and the jury's acceptance of those 24 conclusions concerning his risk assessment.
Page 4201 1 arsenic or cadmium have the potential to be 2 harmful, but they certainly cannot be the basis of 3 a medical monitoring program. 4 Two other quick examples. First, Doctor 5 Werntz testified -- we think Doctor Werntz's 6 testimony shows that CT scans are not reasonably 7 necessary. He testified that those scans are not 8 recommended by the United States Preventive 9 Services Task Force, which Doctor Werntz conceded 10 himself is the consensus. 11 Although Doctor Werntz himself does 12 recommend those CT scans, the bare unsupported 13 opinion of Doctor Werntz, in the face of his own 14 testimony that the consensus of qualified 15 physicians does not recommend those tests, we 16 believe entitles DuPont to judgment as a matter of 17 law, at least as it relates to the CT scan element 18 of plaintiffs' proposed medical monitoring plan. 19 Indeed -- now, Doctor Werntz relies on 20 only a single article for his CT scan 21 recommendation. It is an article from the Journal 22 of the American Medical Association, which itself 23 concluded - as I believe was shown on cross 24 examination - that these CT scans are an	Page 4203 1 It is true that Doctor Brown did not 2 perform a risk assessment on lead itself; however, 3 he did measure lead levels and -- not in humans, 4 and Doctor Werntz testified that lead has additive 5 effect, and because of that additive effect, it is 6 reasonably necessary to consider that a 7 significant risk, and increased -- and increasing 8 the risk of contracting a serious latent disease. 9 Doctor Werntz, in addition to relying on 10 Doctor Brown, performed his own qualitative 11 analysis that he testified to yesterday. Doctor 12 Werntz established the significant risk of 1 in 13 100,000, and confirmed that Doctor Brown's risk 14 assessment is three times that, I believe, in the 15 furthest-away zone, Zone 3. 16 So it exceeds Doctor Werntz's 17 determinations -- actually exceed the levels that 18 even Doctor Brown found in his risk assessment. 19 In other words, Doctor Werntz reviewed it himself 20 and made his own conclusions about what 21 constitutes significant risk. 22 It would be in -- we -- Doctor Werntz 23 presented testimony on various diseases that can 24 be caused by all three of the elements at issue.

4 (Pages 4200 to 4203)

Page 4204 1 Those three diseases -- goes to show that they are 2 hazardous, that he restricted his medical 3 monitoring to what he would consider to be, as a 4 practicing physician in West Virginia, reasonably 5 necessary to screen for and practical as well. 6 Are there any other issues that you'd 7 want us to specifically address? 8 THE COURT: Counsel, anything in 9 conclusion? 10 MR. PRESTES: Yes, your Honor, just three 11 quick points. First of all, Ms. Mason has 12 suggested that we are trying to relitigate the 13 jury's acceptance of Doctor Brown's conclusions. 14 We don't think that's accurate. Though 15 it is true that in the Phase 1 verdict, the jury 16 did conclude that there had been exposure, as I 17 previously suggested, the relevant question here 18 is whether there has been significant exposure. 19 In addition, the Phase 1 verdict found 20 exposure to persons or property, and thus there is 21 no Phase 1 verdict making -- accepting the 22 conclusion that there has been significant 23 exposure to persons. 24 Moreover, to the extent there is any such	Page 4206 1 instead of Doctor Hall, that counsel ought to be 2 making the closing summation. 3 Most, if not everything, counsel's 4 introduced really goes to the weight of the 5 evidence, which is really an issue for the trier 6 of fact. 7 The Court cannot say as a matter of law 8 that the plaintiffs failed in any one of the 9 Bowers factors or in any of the subarguments that 10 have been made. Seems to me that those are -- 11 they may very well be compelling arguments, but 12 one that must be offered to the triers of fact, 13 and the Court - even accepting those in a light 14 most favorable - cannot say as a matter of law 15 that the plaintiffs failed to meet its burden at 16 this point. 17 So for those reasons and all other 18 reasons appearing of record, the motion for 19 judgment as a matter of law - or in the 20 alternative, to decertify class - will be denied 21 and the defendant's exceptions preserved. 22 MS. MASON: Thank you, your Honor. 23 MR. PRESTES: Thank you. 24 (Counsel returned to counsel table and
Page 4205 1 conclusion, we believe that there is no sufficient 2 evidence to support that conclusion. 3 Second, I'm somewhat confused by 4 counsel's reference to Doctor Brown's risk 5 assessment and how they provide a basis for Doctor 6 Wertz's opinion, because as is clear from the 7 record, Doctor Brown conducted no risk assessment 8 on lead, and hence, there is no basis in Doctor 9 Brown's testimony or in the evidence that has come 10 in to this case so far for Doctor Wertz to 11 testify that any class members have been exposed 12 to a risk relating to the exposure to lead that 13 would justify medical monitoring. 14 Finally, it sounds like we are in 15 agreement on the issue relating to the laundry 16 list of diseases that Doctor Wertz testified to 17 and the fact that although he testified to, say, 18 20 or more diseases; only a small number of those 19 five cancers and three noncancers would even 20 conceivably form the basis of the medical 21 monitoring program that the plaintiffs urge. 22 THE COURT: Counsel, let me indicate 23 although counsel for DuPont makes a compelling 24 argument and if -- if his client was smart,	Page 4207 1 the proceedings continued in the presence and 2 hearing of the audience as follows:) 3 THE COURT: We have about ten minutes, 4 counsel. They're deliberating lunch. 5 (A recess was taken after which the 6 proceedings continued as follows:) 7 THE COURT: Be seated, please. 8 Mr. Bailiff, you may return the jury to its box, 9 sir, please. 10 (The jury returned to the courtroom.) 11 THE COURT: You may be seated. Good 12 morning, ladies and gentlemen of the jury. Let me 13 note the appearance of all 11 of our jurors who 14 are again present before the Court and the parties 15 and their respective counsel. 16 I believe the plaintiff now having 17 rested, it's the Defendant DuPont's presentation 18 of evidence. Mr. Hall, DuPont may call its first 19 witness, sir. 20 MR. HALL: Yes, your Honor, DuPont calls 21 David Garabrant. Mr. Gallagher will handle the 22 examination, your Honor. 23 THE COURT: If you'll come forward, sir, 24 and be sworn, please.

5 (Pages 4204 to 4207)

Page 4208 1 (The witness was sworn.) 2 THE COURT: You may have a seat up here 3 in the witness stand, please. 4 DAVID GARABRANT 5 was called as a witness by the Defendant, and 6 having been first duly sworn, testified as 7 follows: 8 DIRECT EXAMINATION 9 BY MR. GALLAGHER: 10 Q. Good morning. Would you tell us your 11 name? 12 A. Yes, David Hay Garabrant. 13 Q. Are you a medical doctor? 14 A. Yes. 15 Q. What is your specialty? 16 A. Occupational and environmental medicine 17 and also internal medicine. 18 Q. Do you study the human health effects of 19 exposure to chemicals in the environment? 20 A. That's what I've spent my career doing. 21 Q. Have you been asked to provide expert 22 opinions in this case concerning the medical 23 monitoring program suggested by the plaintiffs' 24 expert, Doctor Werntz?	Page 4210 1 Harvard. 2 In 1980 to 1981, I completed my senior 3 residency in internal medicine at Boston 4 University medical center in Boston. And so by -- 5 in 1981, I finished my training. 6 Q. Are you currently licensed to practice 7 medicine? 8 A. I am currently licensed to practice 9 medicine in Michigan, I have an active license. 10 I'm also licensed to practice in Massachusetts, 11 Washington, D.C., Maryland and California. Those 12 licenses are inactive because I don't reside in 13 those states. 14 Q. Are you board certified to practice in 15 any specialties in medicine, special areas? 16 A. Yes. I'm board certified in preventive 17 medicine, and within preventive medicine, the 18 field of occupational medicine. I'm also board 19 certified in internal medicine. 20 Q. How long have you been practicing 21 occupational and internal medicine? 22 A. Well, since I finished my residency in 23 1981, so that's 26 years. 24 Q. Are you still practicing medicine today?
Page 4209 1 A. Yes, I have. 2 Q. And in particular, have you been asked to 3 consider whether Doctor Werntz's conclusions about 4 the risk of cancer and other diseases are 5 warranted in light of the evidence? 6 A. Yes. 7 Q. Before we get to your opinions on that, 8 let's talk about your background. Could you 9 describe for the jury your education? 10 A. Sure. I got my undergraduate degree, my 11 college degree, in chemical engineering. I went 12 to Tufts University outside of Boston, graduated 13 in 1972. Then I went directly to medical school, 14 also in Boston, at Tufts University School of 15 Medicine, received my M.D. degree in 1976. 16 I interned at Georgetown University 17 Hospital in Washington, D.C. in internal medicine 18 in 1976/'77, then I did a fellowship in medical 19 ambulatory care also at Georgetown, 1977 to 1978. 20 Then from 1978 to 1980, I did a residency 21 in occupational medicine at the Harvard School of 22 Public Health in Boston. At that same time, I 23 also received two graduate degrees, one in public 24 health and a master of science in physiology from	Page 4211 1 A. Yes. 2 Q. As -- over the course of your career 3 since 1981, has the nature of your practice 4 changed? 5 A. Yes. I'm on the faculty at the 6 University of Michigan, and been there for -- it 7 will be 19 years this fall. And over that 19 8 years, my time has shifted much more towards doing 9 research and much less in the clinic caring for 10 patients. 11 Q. Do you still see patients, though? 12 A. I do. 13 Q. At the University of Michigan, do you 14 teach classes to students in the medical school 15 and graduate schools? 16 A. I teach in the School of Public Health 17 principally, and also in the School of Medicine. 18 Q. What subjects do you teach? 19 A. I teach risk assessment, I teach 20 occupational environmental diseases. I teach 21 research methods in occupational and environmental 22 epidemiology, and I run a number of different 23 seminar courses. They vary from year to year. 24 Q. You mentioned that at the University, you

Page 4212 1 do research. Is there a particular area that you 2 focus on in your research? 3 A. Yes. I've spent my whole career doing 4 epidemiology, studies of people who handle 5 chemicals and who are exposed to chemicals. So 6 I've spent my whole career out in work places, in 7 factories, in the auto industry, in mines, various 8 industrial facilities, chemical plants, looking at 9 cancer risks among people who are exposed to 10 chemicals, sometimes looking at risks of lung 11 disease, sometimes looking at risks of nervous 12 system or neurologic disease. 13 That's what I've spent my career doing. 14 And also looking at environmental exposures and 15 disease risks related to those exposures. 16 Q. Now, does that research that you do, does 17 that involve preparing studies that summarize your 18 findings? 19 A. Well, it certainly involves writing the 20 findings up and writing them for publication in 21 the peer-reviewed literature. I do that 22 routinely. 23 Q. Are studies that are performed in your 24 area typically published in a scholarly journal of	Page 4214 1 going to accept this manuscript," or "We're not 2 going to accept it." And typically when they 3 don't accept it, they write you back and say, 4 "We're sorry, your manuscript is not accepted, 5 comments from the reviewers are attached. If you 6 are willing to respond to those comments, we would 7 be happy to reconsider your manuscript." 8 And so you read the comments and you say, 9 "Yeah, you know, I can do those things," and good 10 reviews often suggest things that make your 11 manuscript better. So they say, "You know, you 12 really should have presented this data," or "We'd 13 like to see this table" or whatever, and so then 14 you revise your manuscript and you resubmit it, 15 and hopefully it gets accepted. 16 Sometimes it doesn't. Sometimes you 17 submit it to a different journal; sometimes you 18 start over again. 19 And that process -- so then if it gets 20 accepted, then it gets published in that peer- 21 reviewed journal, such as the Journal of 22 Occupational Environmental Medicine or 23 Environmental Health Perspectives or one of the 24 professional journals that publishes original
Page 4213 1 some sort? 2 A. Certainly try to get them published in 3 scholarly journals, yeah. 4 Q. Can you describe that process for us? 5 What do you have to do to get your research 6 published in a journal? 7 A. Yeah. So you have to do the research. 8 You have to have written up what the results are 9 and what it means, and you typically put that in a 10 manuscript form. Every journal has its own 11 format. You have to look at the journal format. 12 And then you mail it to the journal, or now you 13 submit them electronically. 14 The journal receives your manuscript, 15 they send you now an e-mail saying, "Yeah, we 16 received it," and then the editorial board of the 17 journal sends it out to other scientists who are 18 qualified in the area of your research to read 19 your manuscript and review it and write a review, 20 and they write comments and criticisms about the 21 manuscript. 22 They send those back to the editorial 23 board in the journal, and the journal looks at 24 them, and they say, "Okay, well, we're either	Page 4215 1 research. 2 Q. Is that process called peer review? 3 A. That's called peer review, yes. 4 Q. And is it called peer review because you 5 have other scientists that work in the field, 6 people who know the area, look at your research 7 and pass judgment on whether it meets the criteria 8 for your science? 9 A. Yeah, the idea of having peers is that 10 people that are qualified in the area that you are 11 working in are reading and critiquing your work, 12 and so that's peer review. 13 Q. How many of your studies have gone 14 through this peer review process and been 15 published in scholarly journals? 16 A. Oh, I don't know the exact count. It's 17 over 100 at the moment. 18 Q. Do you participate in the peer review 19 process as one of the people who reviews the 20 studies when they come in to the journals? 21 A. Oh, sure, yeah. 22 Q. What journals do you perform peer review 23 for? 24 A. I review for the Journal of Occupational

7 (Pages 4212 to 4215)

Page 4216 1 Environmental Medicine, Environmental Health 2 Perspectives, American Journal of Industrial 3 Medicine, Cancer Epidemiology, Biomarkers and 4 Prevention, Occupational and Environmental 5 Medicine. There are more. I'm trying to think -- 6 ones I haven't done recently -- 7 Q. It's a bunch of journals? 8 A. Yeah, there are probably six or seven 9 journals that send me manuscripts and ask me to 10 review them. 11 Q. Does that take time out of your workday 12 to do that? 13 A. It sure does. 14 Q. Do you get paid to do that? 15 A. No. No, everybody does that because it's 16 part of being a scientist. It's part of 17 participating as a scientist. You know, the 18 reward is that you get to see what's being done, 19 you get to see what people are working on, you get 20 to see what they're thinking about, and it's very 21 engaging work. 22 Q. You mentioned earlier -- you used the 23 term "epidemiology" to explain research that you 24 do. Can you give us a very short -- we'll get	Page 4218 1 Q. In your research as an occupational -- in 2 occupational medicine since 1980, have you 3 conducted research into the causes of cancer? 4 A. Oh, yeah, I've -- a considerable part of 5 my career has been spent doing cancer 6 epidemiology. 7 Q. Without going over the entire body of 8 what you've done, are there any studies or 9 projects that you've worked on that you are 10 particularly proud of, what you think is your most 11 significant contribution to the body of scientific 12 knowledge on the causes of cancer? 13 A. I think the one that I like the most is a 14 paper that my colleagues and I published in 1984 15 that showed that people who have physically active 16 occupations are at lower risk of colon cancer than 17 people who have sedentary occupations. 18 And we published that in the American 19 Journal of Epidemiology in 1984. It's been 23 20 years. And that -- that study has been confirmed 21 now over 50 times, and now that finding is in all 22 the textbooks, that physical activity reduces your 23 risk of colon cancer. 24 And I see it -- you know, I see it in the
Page 4217 1 into it more later. But can you give us a very 2 short explanation of what that is? What is it, 3 epidemiology? 4 A. Epidemiology, the word comes from the 5 same word "epidemic," and epidemic is an unusual 6 occurrence of disease. 7 So there's some background rate of 8 typhoid or cholera or tuberculosis and then all of 9 the sudden, there's a big surge. That's an 10 epidemic, okay. Epidemiology is the study of 11 patterns of diseases in the human population, and 12 it has its routes in studying infectious diseases 13 like tuberculosis, cholera, typhoid, those types 14 of diseases, and now it includes studying patterns 15 of cancer risk, patterns of lung disease, patterns 16 of cardiovascular disease and trying to uncover 17 why people get these diseases and why people are 18 at increased risk. 19 Q. Is epidemiology one of the ways that 20 scientists study the causes of cancer? 21 A. Oh, absolutely. In the human population, 22 it's probably the principle area of study to try 23 and uncover what puts people at increased risk of 24 cancer.	Page 4219 1 news, I see it in, you know, Living Well and Good 2 Housekeeping and all the things you buy in the 3 grocery store. It's basically accepted now as an 4 established fact, that physical activity reduces 5 your risk of colon cancer. 6 And we were the first people to report 7 that. So I'm -- I'm proud of that one, because I 8 think it had some impact, and maybe it's prevented 9 some cancer. 10 Q. Have you done any research into the 11 incidence of cancer in the workplace? 12 A. Oh, sure, yeah. We've -- actually 13 another one I'm very proud of, we did a study for 14 a chemical company, the Rohm and Haas Corporation 15 in Philadelphia. Rohm and Haas had been doing -- 16 had been following their cohort of workers that 17 worked for us in one of their plants in 18 Philadelphia for years looking at cancer risk, and 19 they received an excess of pancreas cancer in 20 their work force, so they came to me, because I 21 had some interest in pancreas cancer, and they 22 said, "What should we do? We think we've an 23 excess?" 24 And I said, "Well, we really ought to do

8 (Pages 4216 to 4219)

Page 4220 1 a case control study to try to see whether the 2 people with pancreas cancer have been handling 3 chemicals differently or different chemicals than 4 the workers who don't have pancreas cancer." That 5 approach would be a case control study. 6 And because I was a chemical engineer in 7 college, I actually had some familiarity with 8 industrial processes and industrial chemistry, so 9 I actually did the study. I worked on-site in the 10 plant interviewing workers for months, and we 11 found that people who had handled DDT and two 12 pesticides derived from DDT that were made in that 13 plant were at very high risk of pancreas cancer, 14 and we published that study in the journal of the 15 National Cancer Institute, the JNCI, and it was 16 actually a lead article. 17 It was a very important article. And it 18 said, "You know, we've got a seven-fold risk among 19 pancreas cancer among people who handle DDT and 20 two very similar chemicals made from DDT." 21 Q. You talk about some of your research. In 22 your practice of medicine, have you been involved 23 in medical monitoring programs? 24 A. Yes.	Page 4222 1 medical surveillance programs for the City of 2 Pasadena, California. Pasadena had a large number 3 of people who required medical surveillance, had 4 police officers, firefighters, emergency 5 responders. Pasadena has its own electrical power 6 plant, so they have power plant workers who are 7 potentially exposed to a number of hazardous 8 materials, and they also have their own parks 9 department that handles pesticides, they have 10 water and sewer workers, so there are confined 11 space entry issues. 12 So we did all the medical monitoring for 13 all of the City of Pasadena employees. 14 Q. Through your work in research and medical 15 monitoring, have you become familiar with the 16 human health effects and the exposure to 17 substances like arsenic, cadmium and lead? 18 A. Yes, I have. 19 MR. GALLAGHER: Your Honor, we tender 20 Doctor Garabrant as an expert in occupational and 21 environmental health, in epidemiology and in 22 particular, the study of the human health effects 23 from exposure to substances in the environment. 24 THE COURT: Any objection or voir dire as
Page 4221 1 Q. Can you describe your experience with 2 medical monitoring? 3 A. Yeah. We've got to back up a step. 4 After I finished my residency in 1981, I was asked 5 to join the faculty of the School of Medicine at 6 the University of Southern California. USC had - 7 and still has - a very fine department of 8 preventive medicine that does Cancer Epidemiology 9 studies, so it's a department of 23 or 24 10 physicians and statisticians who do Cancer 11 Epidemiology, so I joined the faculty at USC, and 12 I was at USC for seven years. 13 While I was there, I did medical 14 monitoring for a number of different populations 15 of people. I did medical monitoring for two brass 16 foundries. When you make brass, one of the 17 ingredients is lead, and because lead boils off at 18 a lower temperature than the other metals in 19 brass, pouring molten brass generates lead fume, 20 so you have to monitor brass workers typically for 21 lead exposure. 22 So I sat up and ran medical monitoring 23 for lead exposure on these brass foundry workers. 24 I also, with two colleagues, ran the	Page 4223 1 to qualifications, Mr. Barr? 2 MR. BARR: Your Honor, we'll do our 3 questions on cross. 4 THE COURT: Let me so qualify the 5 witness, and you may proceed. 6 MR. GALLAGHER: Your Honor, is it okay if 7 I go over there and put a board up on the easel? 8 THE COURT: Yes, sir. 9 Q. Doctor Garabrant, did you review the 10 expert report that Doctor Werntz prepared to 11 describe his proposed medical monitoring program? 12 A. Yes, I did. 13 Q. And did you -- do you understand that 14 Doctor Werntz has testified that there -- in the 15 class area, there is a significant exposure that 16 leads to a significantly increased risk of certain 17 types of cancer and certain noncancer ailments? 18 You understand that from reading his report? 19 A. Yes, I do. 20 Q. Specifically with respect to that 21 question, whether there is a significant exposure 22 leading to a significantly increased risk of 23 cancer or noncancer risks in the class area, do 24 you have an opinion as to whether Doctor Werntz's

Page 4224 1 conclusions are well-founded? 2 A. Yeah, I do. 3 Q. What is your opinion on that? 4 A. Well, I don't think they're well-founded. 5 I don't think he's gone through the process of 6 putting together the scientific evidence that 7 would adequately provide a foundation for his 8 opinions. And I don't think that scientific 9 evidence actually exists that would provide that 10 foundation. 11 Q. I'd like to talk about the cancer risks 12 and the noncancer risks separately and start with 13 the cancer risks. With respect to the risk of 14 cancer, how do scientists make a decision about 15 whether an exposure to something like arsenic, 16 cadmium or lead is likely or is known to cause 17 cancer? 18 A. Let me just go through the steps. First, 19 there has to be research that looks at populations 20 of people who are exposed to that chemical, and 21 that research has to be published, and it has to 22 show that people who are exposed are at increased 23 risk of that chemical. 24 And that evidence has to include	Page 4226 1 one coherent picture?" 2 That's what we try to do. 3 Q. And you prepared a series of slides to 4 help us understand what epidemiological studies 5 look like, the studies you're talking about that 6 you had collected and look at. 7 A. Yeah, sure. 8 Q. Is this the presentation you prepared? 9 A. Yes. 10 Q. So tell us what -- if you could introduce 11 to us your presentation on studies. What are we 12 going to talk about here? 13 A. Before I can talk about what the 14 epidemiology says about cancer, I've got to talk a 15 little bit about what epidemiology does and how we 16 do it. 17 Okay. So as I've already said, 18 epidemiology is that field of science that studies 19 the distribution of diseases and their causes or 20 the exposures in human populations. Epidemiology 21 is not about laboratory animals; it's about people 22 who live in society and do what people do and who 23 go to work and have exposures at work and come 24 home and eat foods and smoke cigarettes and do
Page 4225 1 estimates of the exposure, and you have to know 2 about the exposure pathway, in other words, 3 whether it's a setting where people are handling 4 the chemical because there's dust or fume or vapor 5 in the air, or whether they're getting that 6 chemical through drinking water, so ingestion. So 7 the exposure pathway matters, and the exposure 8 level matters. 9 And is there an association of increased 10 cancer risk with that exposure? When a study like 11 that is done, we often -- in fact, we almost 12 always expect to see it replicated. In other 13 words, we expect to see that other people have 14 studied the same issue and have found similar or 15 identical results. 16 You know, and results do vary, you know, 17 from chance a little bit. But we expect to see 18 replication. And so a scientist who says that a 19 chemical causes a disease like cancer has to go 20 back and find that scientific evidence and has to 21 assemble it and has to try and figure out, "Okay, 22 there's 10 or 15 or 20 studies that have looked at 23 that issue. What does that body of literature 24 actually say when you try to put it together into	Page 4227 1 what people normally do. 2 And the purpose is to understand the 3 causes of disease in human -- in the human 4 population. 5 Q. Are there different types of 6 epidemiological studies that have been performed 7 to get at that question? 8 A. There are two principal types of studies, 9 cohort studies and case control studies. Okay -- 10 Q. Can you tell us what a cohort study is? 11 A. A cohort study is, I think, the one 12 that's intuitively clearest. To do a cohort 13 study, you find a group of people who are exposed 14 to the chemical. Okay? And let's use an example 15 like coffee drinking. 16 So you would identify a population of 17 people who drink coffee, and then you'd want to 18 compare them to people who don't drink coffee, 19 right? Because you're interested in, "Well, does 20 coffee cause -- we'll use the example of pancreas 21 cancer, because it's a famous one. 22 So you want to know, does coffee drinking 23 cause pancreas cancer? You say, "Let's get 24 together a cohort of coffee drinkers, let's get

Page 4228 1 together a cohort of people who don't drink 2 coffee." And so you follow both groups for years 3 - because cancer is a very slow disease - you 4 follow both of these groups for 10, 20, 30, 40 5 years, and as time passes, you identify who got 6 pancreas cancer in both groups. 7 Okay, now we can go to the next slide. 8 Q. Just so it's clear, when you're doing 9 this cohort study and comparing the groups, you 10 have the exposed group that you've illustrated 11 here, and then you have the nonexposed group, 12 right? 13 A. Exactly. 14 Q. The exposed group is the group that has 15 been exposed to the thing you're studying, right? 16 A. Right. 17 Q. And the nonexposed group is the group 18 that's never been exposed to what you're looking 19 for. 20 A. Right. And we could use arsenic as an 21 identical example, in other words, a group of 22 people exposed to arsenic and a group of people 23 not exposed to arsenic. 24 Q. Okay. So what do you do next?	Page 4230 1 four out of twelve on the right in the nonexposed 2 group, so they have a halving of the risk, in 3 other words, one half. Which is equally as big as 4 two-fold. Like one half and two-fold are equally 5 large ratios. 6 Q. Okay. So just so it's clear, the three 7 basic possibilities are the two groups will be 8 roughly the same or that one or the other will 9 show a greater incidence of the disease. 10 A. That's correct. 11 Q. All right. So then what do you do with 12 the results to provide a conclusion about this? 13 A. Okay, we calculate what's called a risk 14 ratio or a rate ratio, which is very simply taking 15 what happens among the exposed group and dividing 16 it by what happens among the nonexposed group. 17 So in this example, in the exposed, two 18 out of twelve got the disease. We put that in the 19 numerator, and then two out of twelve in the 20 nonexposed group got the disease, we put that in 21 the denominator, and the ratio is what matters. 22 Okay? If the ratio is 1, it says the two 23 groups are equal. If the ratio is above 1, it 24 says the exposed group is at increased risk. So
Page 4229 1 A. You follow them, as I said, in cohort 2 studies for cancer, typically for decades, okay? 3 And you see who got the disease under study. And 4 I've marked here with purple boxes that in both 5 groups, you get cancer. Because there is a 6 background of cancer in every population. 7 And so the issue now is whether the rate 8 of cancer is higher in the exposed group than it 9 is in the nonexposed group. All right? And so 10 you have to compare exposed to nonexposed, and you 11 want to see what the rates are different, okay? 12 Q. So how do you do that? 13 A. Well, essentially you count them up, all 14 right? And so in the top diagram, I've got the 15 two groups have equal rates of disease because in 16 both groups, you've got two out of twelve with the 17 disease. 18 In the middle, I've got the exposed group 19 has a higher rate of disease, you've got four out 20 of twelve compared to two out of twelve, so that's 21 a doubling of the risk. 22 And in the bottom diagram, the exposed 23 group has a lower rate of disease, okay? So two 24 out of twelve on the left in the exposed group,	Page 4231 1 the risk ratio is above 1, there's increased risk 2 in the exposed, and if the risk ratio is below 1, 3 it says the exposed group has less disease. 4 Sometimes -- you'll say, "Well, how could 5 you have less disease?" It goes back to my 6 physical activity study. We found that the people 7 who exercised had lower risks than the people who 8 didn't, so the exposure prevented cancer, so it 9 can go either way. Or you can have no association 10 at all. Okay, so -- 11 Q. What -- 12 A. We covered this. The positive 13 association, so here would be a risk ratio of 14 four-fold, and the next one, here would be a risk 15 ratio of one quarter or .25. Okay, so you -- you 16 know, that's how we calculate these relative risks 17 or risk ratios, and we call this a measure of 18 association. 19 Is the exposure associated with disease 20 risk? Okay. 21 Q. So in this case, you've got a negative 22 association. 23 A. You can have a negative association. 24 Q. And does take mean -- if you see a

Page 4232 1 negative association, that's less than that 1 2 value that was the value of them being the same. 3 A. Right, if it's below 1, it's a negative 4 association. 5 Q. And if you have a negative association, 6 does that mean that people who were not exposed to 7 the chemical actually got more cancer than the 8 people who were? 9 A. That's correct, the exposed group has 10 lessor the nonexposed group has more. 11 Q. So that's a cohort study. Can you 12 explain what a case control study is, the second 13 type? 14 A. Yeah. I mentioned that pancreas cancer 15 study I did for the chemical company. We did a 16 case control study. Case control study is just a 17 little different design. What you do is, you go 18 out and find people who've got the disease you're 19 interested in. 20 So here we were interested in pancreas 21 cancer. So you've got to go out and find them, 22 typically by working with the hospitals, working 23 with the tumor registries in the area, working 24 with the state registrar of death information to	Page 4234 1 assemble their work records and find out what 2 their past exposures were, and we do that for both 3 groups, right? And so here I've -- I've 4 highlighted now in dark green among the cases, 5 four of the twelve had exposure, or the way we 6 actually calculate it is, among the cases, four 7 had exposure, eight did not, and we calculate it 8 as odds. 9 Anybody who's ever bet on horses or a dog 10 race knows what odds are. It's the number of 11 times something will occur if divided by the times 12 it doesn't occur. So we get the exposure odds 13 among the cases. We get the exposure odds among 14 the controls, and then we just compare them, put 15 the odds of the cases, the odds over the cases 16 over the odds on the controls, and we call that an 17 odds ratio, and it tells us the same thing as that 18 cohort study tells us. 19 It tells us whether exposure is 20 associated with disease. All right? If the cases 21 have had more exposure than the controls, the odds 22 ratio is above 1. If the cases and controls have 23 had the same amount of exposure, the odds ratio 24 equals 1.
Page 4233 1 get death certificates, so you assemble 50 or 100 2 or 300 people who've got pancreas cancer, and then 3 you want to compare them to people who do not have 4 pancreas cancer. 5 And the people who don't have pancreas 6 cancer, we call controls. So the cases are people 7 with the disease; the controls are people who 8 don't have it. And the controls are supposed to 9 come from -- to do the study right, they are 10 supposed to be drawn from the population, the same 11 population from which the pancreas cancer cases 12 came. 13 So the issue is, okay, we've got people 14 who got cancer, people who don't. What's 15 different about them? All right? What's 16 different about them? If we did that study for 17 lung cancer and we looked at smoking habits, we 18 would realize very quickly that the people with 19 lung cancer had smoked much more than the people 20 who don't have lung cancer, and of course, we have 21 to match them on age and sex and -- 22 Okay. And so what we do then - if I can 23 have the next slide - is that we typically 24 interview them or interview their next of kin or	Page 4235 1 All right? So it's the same idea that 2 you're looking to see whether exposure is more 3 common among people with disease, and we calculate 4 a relative risk. In this instance, it's called an 5 odds ratio, but it's the same idea as we saw 6 before. And if it's greater than 1, it's a 7 positive association. 8 Okay. 9 Q. So can you explain -- summarizing that, 10 what do the studies - cohort studies and the case 11 control studies - at the end of the day, what do 12 you get? 13 A. Okay. In both types of studies, we look 14 for this measure of relative risk, and that is 15 called an association between exposure and 16 disease. If it's 1.0, it means there's no 17 association at all, the two groups are the same. 18 If it's greater than 1, it's a positive 19 association; if it's less than 1, it's a negative 20 association. 21 So it doesn't matter what the study 22 design is, we measure it the same way. 23 Q. Now, if you have a relative risk greater 24 than 1, does that mean, as a matter of science,

Page 4236 1 that the thing you are studying causes cancer? 2 A. Sometimes, but sometimes not. 3 Q. Can you explain why it can sometimes not 4 mean cancer? 5 THE WITNESS: Next slide, if you would. 6 A. Okay. So associations occur for a number 7 of reasons. The first is that it represents a 8 causal -- a causal relationship, the exposure 9 causes the disease. But sometimes it's due to a 10 badly-designed study, that there's some bias in 11 the study, so you have to look carefully. 12 Sometimes it's due to what we call 13 "confounding," which means there's some other 14 factor that you didn't include in the study that 15 is causing an apparent association. For example, 16 if I study lung cancer and coffee drinking, I 17 would find that they're associated. But they're 18 associated because people who drink a lot of 19 coffee tend to smoke more. 20 And so that's confounding due to smoking. 21 The true cause is something that I didn't include 22 in my study - it's the smoking - and it produces 23 this apparent association between coffee drinking 24 and lung cancer, and so you have to deal with	Page 4238 1 taken a poll of how many people approve of the 2 president's performance and, you know, let's say 3 they find 32 percent plus or minus 4 percent, that 4 plus or minus 4 percent is the confidence 5 interval. 6 That is, what they're saying is, for a 7 sample as big as we took - and these surveys often 8 include 800 or 900 people - we are 95 percent 9 confident that the true answer falls between 32 10 plus or minus 4, so between 28 and 36. That's a 11 confidence interval. 12 And so you see it all the time, and you 13 always see it in scientific studies, because it 14 gives you a sense for, "Okay, is it possible that 15 chance explains this?" Now, the important point 16 -- 17 THE WITNESS: If I can have the next 18 slide. 19 A. So -- often we see graphs like this where 20 the relative risk is presented as a little box or 21 a little circle or something in the middle of a 22 range, and then a bar on either side represents 23 the confidence interval. 24 If that confidence interval includes 1.0,
Page 4237 1 that, or when you -- when you read a study, you 2 have to think, "You know, is there some 3 confounding factor that they didn't deal with?" 4 Okay, because that can -- that can give 5 you a -- an erroneous conclusion. Okay, and then 6 I think the most important one, at the bottom, is 7 chance, okay? Associations occur by chance, and 8 we have to evaluate the role of chance, and 9 scientists always do that. They always calculate 10 "P" values or confidence intervals to evaluate, 11 "Well, what's the role of chance in these 12 findings?" 13 Q. The confidence interval, can you explain 14 what that means? 15 A. Sure. We calculate this measure of 16 association called relative risk, and then we 17 calculate the confidence interval around it, and 18 conventionally, it's a 95 percent confidence 19 interval, and that confidence interval is the 20 range within which the relative risk would fall 95 21 percent of the time if we did the study over and 22 over and over again. 23 And you know, we see these in the 24 newspaper all the time, when you read that they've	Page 4239 1 what it means is, "Well, okay, these results are 2 reasonably compatible with the idea that there's 3 really no association." In other words, we saw a 4 relative risk of 1.38, but it's reasonably likely 5 that the truth could be 1. 6 "We saw 1.38, but it could be 1." On the 7 other hand, if that confidence interval -- if the 8 relative risk is up there way high and the 9 confidence interval doesn't include 1, then you 10 say, "You know, this is unlikely to be a chance 11 finding," so chance is effectively not the 12 explanation for it. 13 So we always evaluate the role of chance 14 in assessing these relative risks. 15 Q. In any one study that you've looked at, 16 any one epidemiological study about cancer, would 17 you see a relative risk reported with a confidence 18 interval? 19 A. You typically do. Older studies didn't 20 used to do it, but for the past 20 years, it's 21 been typical to do it that way. 22 Q. Now, as a scientist, would you draw a 23 conclusion about causation based on one study? 24 A. I -- I would be very reluctant to do

Page 4240 1 that, and I would not do that if there were other 2 studies that were properly conducted. I would 3 look at all of the evidence. I wouldn't rely on a 4 single study. I'd look at all the evidence. 5 Q. When it comes to studying the causes of 6 cancer, what substances cause cancer and under 7 what circumstances, are there bodies -- are there 8 groups of scientists who compile all the evidence 9 and put it all in one place for you to look at, 10 summarize it for you? 11 A. Yes. 12 Q. Is the International Agency for Research 13 on Cancer one of them? 14 A. Sure, yeah. The International Agency for 15 Research on Cancer is actually part of the United 16 Nations. Within the UN, there's the World Health 17 Organization. We all remember UNICEF when we were 18 kids, you know, collecting money in grammar school 19 for UNICEF, vaccinating kids abroad. That's part 20 of the World Health Organization. 21 World Health Organization also includes 22 an agency called IARC, or the International Agency 23 for Research on Cancer, and its mission is to do 24 cancer research and particularly to evaluate which	Page 4242 1 animal carcinogen, it might be carcinogenic to 2 humans. 3 Group 3 is chemicals for which there is 4 just not enough evidence to make any decision, we 5 can't classify it. And then Group 4 - and you can 6 see that there's only one chemical that's ever 7 been put in Group 4 - the evidence is strong and 8 adequate to say, "This thing probably doesn't 9 cause cancer." 10 So -- so the -- the plan is to figure out 11 which chemicals cause cancer and to assess the 12 strength of the evidence. 13 Q. Are there other agencies like IARC that 14 perform a similar function of classifying 15 substances by what the evidence shows about their 16 cancer risk? 17 A. Yes. And I put together a little slide. 18 Across the top row, you can see the U.S. 19 Environmental Protection Agency does this, IARC 20 does this, the American Conference of Governmental 21 Industrial Hygienists, the ACGIH, does it, and the 22 National Toxicology Program, or the NTP, does it. 23 And you can see that they have different letters 24 and numbers, which makes it very confusing.
Page 4241 1 chemicals and industries and occupations and foods 2 and pharmaceuticals cause cancer in humans. It's 3 a very important function. 4 And so IARC does that, and they use a -- 5 a grouping system, Groups 1, 2A, 2B, 3 and 4. 6 Agents that are put into Group 1 are ones 7 where the scientists who are convened feel that 8 the evidence is adequate to say "this chemical 9 causes cancer in humans." Okay? 10 2A means the animal evidence is 11 sufficient to say it's an animal carcinogen, but 12 the human evidence isn't good enough. There's 13 some human evidence, but it's just not good 14 enough. 15 And the -- the conclusion is, "Well, that 16 chemical is probably carcinogenic to humans," but 17 the evidence isn't good enough to say that we know 18 it causes cancer in humans. 19 And then for agents where there's little 20 human evidence or sometimes no human evidence, but 21 the animal evidence is still good, those are 22 typically put in 2B where it's "possibly 23 carcinogenic to humans." In other words, the 24 human evidence isn't there, but we know it's an	Page 4243 1 But the categorization system is very 2 similar across agencies. So a Group 1 or a Group 3 A or a Group A-1 means "This agent causes cancer 4 in humans, we have enough evidence to say that," 5 and then the ones below that mean, "Well, there's 6 limited evidence in humans, but some animal 7 evidence" or "No evidence in humans but some 8 evidence in animals," and it goes on from there. 9 Q. Have you looked at the classifications 10 that the organizations provide for arsenic, 11 cadmium and lead? 12 A. Yes, I have. 13 Q. Is there any difference in their -- in 14 the treatment that the EPA provides in terms of 15 where it classifies it versus IARC and the other 16 agencies? 17 A. Well, we've got to go through the agents. 18 Arsenic is a human carcinogen by everybody's 19 classification. There's no argument about that. 20 Cadmium is a human carcinogen according to IARC, 21 but is in the next category down according to two 22 of the classifications, and I don't have my own 23 list in front of me. 24 I think it's EPA and ACGIH. So there's

Page 4244 1 some minor disagreement about the strength of the 2 evidence for cadmium. 3 And then for lead, none of these agencies 4 classifies lead as a human carcinogen. 5 Q. You mentioned that these agencies 6 classify arsenic as a human carcinogen. Why isn't 7 -- the question here is, is arsenic in the 8 environment? Why isn't that classification the 9 end of the story? I mean, why not just stop right 10 there and say, "The EPA says this is known to 11 cause cancer?" Why don't we just stop with that? 12 A. Well, there's much more to the story than 13 that. 14 You know, if I could just get one of the 15 IARC books, these are -- these are complicated 16 decisions. The assessment of arsenic -- this is 17 the volume on arsenic in drinking water put out by 18 IARC, Volume 84, published in 2004. 19 This is the volume that includes cadmium, 20 Volume 58, published in 1993. This is the volume 21 on lead, inorganic and organic lead compounds. 22 It's published in 2006, just came out earlier this 23 year. I mean, it's a whole book on what the 24 evidence says, and a full and fair assessment of	Page 4246 1 personal experience with cancer, either through a 2 parent or a sibling or an aunt or uncle or 3 grandparent or children. And cancers are very 4 different. 5 So for example, nonmelanoma skin cancer 6 is almost always curable; lung cancer is rarely 7 curable. Pancreas cancer is one of the worst. I 8 mean, the five-year survival for pancreas cancer 9 is horrible. 10 So cancers have different presentations, 11 they have different treatment, and they have 12 different survival. They are different diseases. 13 Q. Do they also have different causes? 14 A. Yes, they have different causes as well. 15 Q. So just because something causes lung 16 cancer in certain circumstances through certain 17 exposure routes, that doesn't mean that it's going 18 to cause skin cancer through another route. 19 A. That's correct. I mean, the cancer risks 20 are specific to the chemical and the pathway of 21 exposure. And it differs, pathway by pathway. 22 Q. Now, does the designation that's given to 23 a -- to an element like arsenic by EPA or the 24 IARC, does that designation tell you what kind of
Page 4245 1 what that evidence means. 2 And when IARC does that, they consider 3 exposure levels, they consider whether the 4 exposure occurs by inhalation or whether it occurs 5 in drinking water or whether it occurs by skin 6 contact. They consider the dose. They consider 7 all of the human epidemiology. 8 They consider all of the animal 9 experiments in laboratories and rats and mice and 10 hamsters and other species, so it requires a lot 11 of work to consider the entire body of evidence, 12 and it's critically important to think about the 13 exposure pathway and the levels of exposure that 14 put people at risk as well as what types of cancer 15 occur from a certain exposure pathway. 16 Q. That's -- 17 A. Things you inhale may cause cancer in the 18 lungs but nowhere else, whereas things in drinking 19 water may cause cancer in different organs, 20 because the exposure pathway matters. 21 Q. That's a point I want you to elaborate 22 on. You said it's important to consider what type 23 of cancer. Are all cancers the same? 24 A. No, no. I think most people have had	Page 4247 1 cancer it's known to cause, through what -- by 2 what route of exposure or at what dose? 3 A. No. No, this is a -- a summary sentence 4 that says, "The evidence is adequate to say, this 5 is a carcinogen to humans" or "The evidence is not 6 adequate to say this is a carcinogen to humans, 7 but it's adequate to say it's probably or possibly 8 carcinogen to humans." 9 Q. Yesterday, Doctor Werntz suggested that 10 living in the area around a smelter necessarily 11 increases your risk of cancer. Do you agree with 12 that conclusion? 13 A. No, I do not. 14 Q. Why not? 15 A. Let's look at the evidence. 16 Q. What evidence do you need to look at? 17 A. Well, the epidemiologic evidence, that 18 studies that have looked at populations that live 19 around smelters. And these studies have been done 20 in many, many locations throughout the world. 21 Q. And do you need to look at that evidence 22 separately for arsenic and separately for cadmium 23 and separately for lead? 24 A. If you can, yes.

Page 4248 1 Q. Why -- 2 A. If the evidence exists, that's what you 3 should do. 4 Q. Why do you want to look at the evidence 5 separately for arsenic and separately for cadmium 6 and separately for lead? 7 A. Well, first off, our assessment of 8 whether these chemicals cause cancer in humans is 9 different for each of those. The types of cancer 10 they cause are different, and they differ in terms 11 of which pathways of exposure lead to cancer and 12 which cancers. 13 So you really have to be specific about 14 the agent and the route of exposure and the 15 chemicals that it's referring to. 16 Q. In your work, did you look separately at 17 arsenic, cadmium and lead? 18 A. Yes. 19 Q. So let's start with arsenic. Doctor 20 Wernitz includes monitoring in his medical 21 monitoring program for, I think, four different 22 types of cancer that he says are -- are at issue 23 here. He talks about lung cancer, he talks about 24 bladder and kidney and skin cancer.	Page 4250 1 Q. Can you explain what this chart is you 2 prepared? 3 A. Yeah. This is just a schematic to try 4 and make it clear that the risks of cancer are 5 different for inhalation than they are for 6 drinking water. Okay? So for inhalation, 7 particularly in occupational settings, lung cancer 8 is a risk. 9 Q. Is there any indication in the scientific 10 literature that inhalation of arsenic at any level 11 - whether it's in the environment or occupational 12 exposures - that that pathway, that route, 13 exposure, causes bladder, skin or kidney cancer? 14 A. I don't believe so. I don't believe so. 15 It's lung cancer in the inhalation literature. 16 Q. And then you have a separate route down 17 here for drinking water where you list some 18 additional cancers. Tell us why you provided 19 that. 20 A. There are a number of areas around the 21 world that have very high arsenic levels in their 22 drinking water. Taiwan, particularly the 23 southwest coast of Taiwan, Chile, and Bangladesh, 24 and these -- they have large populations who have
Page 4249 1 Have you considered whether there is a 2 significant increased risk of those four cancers 3 as a result of the exposure that we have in this 4 class area to arsenic? 5 A. I have. And we should look at the 6 evidence. 7 Q. What does the scientific literature say, 8 in general, about whether arsenic causes cancer or 9 under what circumstances it causes cancer? 10 A. Well, it's very clear that arsenic causes 11 lung cancer when you inhale it, and that evidence 12 comes from people who are in industrial operations 13 where there's arsenic exposure. So there's no 14 argument about that. 15 If you worked in arsenic smelting, 16 arsenic refining and there's high level of 17 exposure to arsenic by inhalation, it's associated 18 with an increased risk of lung cancer, and in 19 fact, that is much of the evidence upon which our 20 assessment that arsenic is a carcinogen is based. 21 It comes from the occupational studies. 22 Okay. So inhalation of arsenic, particularly in 23 occupational settings where the exposures are 24 high, is linked to increased risk of lung cancer.	Page 4251 1 drunk highly contaminated water for decades. 2 There have been an extensive number of 3 studies of cancer risks in Taiwan where people 4 have drunk arsenic-contaminated water from wells. 5 And those studies show very clearly that people 6 are at increased risk of lung cancer, bladder and 7 kidney cancer, and skin cancer. 8 And the evidence from Chile also shows 9 the same patterns. So I think there's no argument 10 that drinking water contaminated with arsenic at 11 high levels is associated with increased risk of 12 those four types of cancer: Lung, bladder, kidney 13 and skin. 14 Q. Now, in your chart, you indicate that 15 lung cancer is associated with exposure to arsenic 16 when you have inhalation and occupational 17 exposures. What does that mean? 18 A. It means that the -- the evidence that 19 inhalation of arsenic causes lung cancer really 20 comes from occupational studies. It doesn't come 21 from environmental studies. In fact, there have 22 been a lot of environmental studies that have 23 looked at that, and they have not given any clear 24 indication of increased risk.

Page 4252 1 And I'd like to review those. You know, 2 some are positive, and some are negative as well. 3 Q. And one last question on this side, and 4 then we'll go to those studies. When you indicate 5 that drinking water is a -- arsenic is associated 6 with these cancers when there's exposure in 7 drinking water, as the high levels that you 8 indicate there, has there been any indication in 9 this case that there are levels or exposures of 10 that type? 11 A. No. The studies in Taiwan involve 12 populations where the drinking water levels are -- 13 the majority of the wells are over 300 micrograms 14 per liter, and the levels are 300, 600, 800 and 15 above. 16 From the evidence I've seen in this case, 17 the highest value found in any reading in any well 18 here was 80. And it's my understanding that the 19 population's been on municipal water since 1964, 20 so even though there was a single well reading at 21 80, that the class area hasn't drunk that type of 22 water at all - or rarely - and even that is well 23 below the levels in Taiwan that are known to put 24 people at risk of these cancers.	Page 4254 1 did you include the studies that Doctor Werntz 2 relied on? 3 A. Yes. 4 Q. And did you include some additional 5 studies that you found through your own research? 6 A. Yes. 7 Q. Let's walk through this. And tell us 8 what the studies show about whether exposure to 9 arsenic in the environment like you would get 10 living around a smelter, whether that puts you at 11 an increased risk of lung cancer, if you could 12 walk us through. 13 A. Sure, yeah. So I've listed across the 14 top the name of the study. I should probably get 15 them out. Hold on a second. 16 Okay. So there's a lot of literature. 17 Okay. So I've listed the author, so the first one 18 is by Brown published in 1984, and then a brief 19 description of the population that was studied, 20 and so you can see in the Brown study, this is a 21 study of men whose usual residence was near a zinc 22 smelter, and then I've got the relative risk, and 23 in parentheses, the confidence interval. 24 And you can see in the Brown study, the
Page 4253 1 Q. Have you read Doctor Brown's analysis? 2 A. I have. 3 Q. Have you read Doctor Werntz's analysis? 4 A. I have. 5 Q. Did either of those experts identify 6 drinking water like the drinking water in Taiwan 7 or Bangladesh or these other areas as a source of 8 exposure to people in the class area? 9 A. No. 10 Q. So you said you wanted to talk about the 11 studies. Have you considered the studies that 12 exist on -- on whether living near a smelter or an 13 industrial site like that, whether that can 14 increase your risk of lung cancer? 15 A. I have. 16 Q. And does this slide include the studies 17 that you looked at? 18 A. It includes some of them. There are more 19 that I have reviewed in addition. There would be 20 a few more slides of those. But I think these 21 give the picture. And this slide includes the one 22 that Doctor Werntz considered, and others that he 23 didn't consider. 24 Q. So in your summary slide you've provided,	Page 4255 1 relative risk was 1.6, so it was -- it was above 2 1, slightly, and you can see that the confidence 3 interval included 1, went from .8 to 3.4. So 4 that's not a statistically significant finding. 5 I want to point out that the authors of 6 the Brown study calculated a 90 percent confidence 7 interval, which is a little narrower than the one 8 we usually calculate, which is 95. So I 9 recalculated the confidence interval, and it's a 10 little bit wider. Doesn't change the answer. 11 So Brown found the positive association. 12 But it was reasonably compatible with there being 13 no real association, that is, 1.0, and that the 14 results may be due to chance. Okay. 15 Q. Let me just ask you, based upon the Brown 16 study, could you conclude that people who lived in 17 the area of a zinc smelter and had exposures like 18 the exposures we're talking about here in this 19 case, could you conclude that they had -- were at 20 a significantly increased risk of lung cancer? 21 A. No, you would not conclude that. They do 22 not have significantly increased risk. They have 23 nonsignificantly increased risk. In other words, 24 it's possible that this is just chance.

Page 4256 1 Q. What's the next study you looked at that 2 you summarized in your table? 3 A. The next one is Pershagen, 1985, and that 4 study was updated recently and published again in 5 2003. 6 Q. What does it mean to update a study? 7 A. Well, they had followed that population 8 for more years, all right? So 18 years later, 9 we've got an update where they followed the 10 population. Remember I said in cohort studies, it 11 takes decades. So you've got a lot more evidence 12 - 18 more years - and much more data to work with 13 to try and measure the cancer rates. 14 Okay. So in the original Pershagen study 15 -- these are two parishes near a copper, lead and 16 zinc smelter in Sweden, and the original report 17 said it was a two-fold relative risk, and it was 18 statistically significant in men; the confidence 19 interval goes from 1.2 to 3.4. 20 But the update that came out just four 21 years ago gives the results now for men and for 22 women, and they've come down. So in men, it's 23 1.38 and it's no longer significant, confidence 24 interval goes from .89 to 2.14, and in women,	Page 4258 1 A. Well, that's a clear statement it does 2 not. It's -- you've got the rate that you would 3 expect among the rest of the state that's not 4 exposed. 5 Q. Now, the next study you have listed here 6 is the Tokudome study. Is that one of the studies 7 that Doctor Werntz relied upon in support of his 8 conclusion that there was a risk here? 9 A. Yes. 10 Q. Tell us about the Tokudome study. 11 A. Well, the Tokudome study was a study of 12 workers in a copper smelter. This is not a study 13 of people who lived near a smelter; it's a study 14 of the workers. 15 Q. Why is that an important distinction? 16 A. This is really important because these 17 are highly-exposed people who worked in and around 18 it. 19 Q. Not the people who lived around it. 20 A. This was not a study of people who lived 21 in the vicinity of a smelter, no. 22 Q. Was there anything about the Tokudome 23 study that you could look at and draw at least 24 some conclusions about whether people living
Page 4257 1 there's a negative association. It's .88. It's 2 not significantly negative. 3 So the point here is that it's a little 4 higher in men, a little lower in women, but on 5 balance, it's pretty close to 1.0. In other 6 words, it doesn't appear in the Pershagen study, 7 as updated recently, that there's evidence of 8 increased risk of lung cancer among people who 9 lived near a smelter. 10 Q. What is the next study you looked at? 11 A. The Bartlesville, Oklahoma study. This 12 was one done by ATSDR. It's part of the Centers 13 for Disease Control. This is residents near a 14 zinc smelter. They didn't give the relative risk, 15 at least the numbers aren't in the report. 16 But they did say the occurrence of lung 17 cancer was as expected from the state rates. So 18 what they're doing is, they're making a comparison 19 of the rates in the Bartlesville population to the 20 rates in the rest of the state. They said, "It's 21 the same; it's as expected." 22 Q. What does that tell you about whether 23 living near a zinc smelter puts you at a 24 significantly increased risk of lung cancer?	Page 4259 1 around a smelter are at increased risk of lung 2 cancer? 3 A. Well, I want to be cautious about making 4 leaps of faith. Let's talk exactly what the 5 Tokudome study says. In the workers in the 6 smelter, the refinery workers, there was an 11.89 7 or almost 12-fold risk. They didn't give the 8 confidence interval. 9 This is an old study. It goes back 30 10 years, 1976. But they did calculate a "P" value, 11 and they said this is statistically significant 12 with a "P" value of less than .01. Okay, so 13 12-fold risk in the workers. 14 They also included clerical workers who 15 worked in the smelter -- not out in the smelting 16 operation, but in offices on the site. Those 17 people were at no significantly increased risk, so 18 the relative risk was 1.75 and it was not 19 statistically significant. 20 Q. So can you extrapolate out to the 21 community around the smelter? 22 A. No, you cannot. 23 But what it does say is that even among 24 the workers on-site who were in office settings,

Page 4260 1 there was no significantly increased risk, and it 2 suggests that the risk comes from being out in the 3 plant under heavy exposures. 4 Q. Doctor Werntz relied upon that study to 5 support his conclusion that there was a risk of 6 lung cancer here. Do you have an opinion whether 7 you can draw that conclusion from the Tokudome 8 study? 9 A. I don't think that's fair. This is not a 10 study of people who live near a smelter. It's not 11 fair to draw that conclusion. It's not what the 12 study says. 13 Q. What's the next study you looked at? 14 A. Okay. Frost looked at females who lived 15 within three miles of a copper smelter and arsenic 16 refinery, found a relative risk of .82, so lightly 17 less than 1. Confidence interval included 1, so 18 not significantly different than 1. That study 19 basically says there's no association. 20 Q. And what about the Hinwood study you 21 reviewed? 22 A. Okay, Hinwood is a study from Australia 23 where they looked at areas that had high surface 24 soil or drinking water arsenic concentrations.	Page 4262 1 at more? 2 A. I looked at quite a few more in addition. 3 Q. Did you look at all the studies that 4 Doctor Werntz relied upon? 5 A. I certainly did. 6 Q. And based on your review of the studies 7 that Doctor Werntz relied upon and the wider body 8 of scientific knowledge, do you have an opinion as 9 to whether the people in the class area are at a 10 significantly increased risk of lung cancer as a 11 result of arsenic exposure? 12 A. Well, they're just not. The scientific 13 evidence would not support that conclusion. 14 Q. Now, in your slide you showed us before, 15 you talked about the pathways for arsenic, and 16 bladder cancer, skin cancer and kidney cancer. 17 Are those three cancers that Doctor Werntz 18 mentions in his report and about which he 19 concludes there's a significant risk here? 20 A. Yes. 21 Q. Did you look at the studies on those? 22 A. I did. 23 Q. And is this the slide you prepared to 24 summarize what the studies say about whether
Page 4261 1 Some of those came from mine tailings from mining 2 operations; some were naturally occurring, and the 3 Hinwood study found a relative risk of 1.0. 4 Q. So what does -- 5 A. Exactly on the "no association" mark. 6 Q. What does that tell you about our 7 situation here? 8 A. To the extent that it's applicable, it 9 talks about contaminated water, contaminated soil 10 not conferring any risk of lung cancer to people 11 who lived in those contaminated areas. 12 Q. What's the last study included on your 13 slide? 14 A. The Murray -- the Murray, Utah study, 15 also done by ATSDR. This was a study of residents 16 near a lead smelter. They didn't give the 17 relative risk, but they did conclude that they 18 considered the Murray smelter site to be no 19 apparent public health hazard. They did not feel 20 there was any reason to consider this a hazard. 21 Q. Now, on the question of whether the 22 exposure to arsenic in the class area puts people 23 at a significantly increased risk of lung cancer, 24 did you review just these studies, or do you look	Page 4263 1 people who live near a smelter are at a 2 significantly increased risk of kidney cancer, 3 bladder cancer and skin cancer? 4 A. Yes. 5 Q. Can you walk us through your analysis of 6 that question? 7 A. Yeah. The -- these studies, which are a 8 subset of the ones on the previous slide, were 9 studies in which the authors gathered information 10 on other cancers. In most cases, they didn't 11 report the results. 12 Q. What does that mean -- first let me just 13 ask: As we did with lung cancer, are you talking 14 about studies that are relevant to the question of 15 whether people living near a smelter are at risk 16 of these cancers? 17 A. Yeah. That -- yes. This isn't -- this 18 is an assembly of the evidence that tries to 19 answer that question. 20 Q. And when those studies say that the 21 cancers are not reported, what does that tell you 22 as an epidemiologist? 23 A. Okay. Well, let's -- let's just go back 24 one step. When you do a cohort study, you have a

Page 4264 1 population of people and you follow them over time 2 and you get their medical records together or 3 their death certificates together, you get all of 4 their records on every cause of death, and so it's 5 routine that you get lung cancer, kidney, bladder, 6 skin, pancreas, prostate, brain, you know, every 7 type of cancer, diabetes and lung disease and 8 heart disease, you name it. That's how you do a 9 cohort study. 10 Okay. So some of these were cohort 11 studies. They had the data for particularly 12 kidney cancer and bladder cancer. Now, they 13 typically don't have skin cancer, okay, because 14 skin cancer is rarely fatal and it doesn't come up 15 in tumor registries or death certificates. 16 But for bladder and kidney, they had the 17 data. Okay, for Bartlesville, the first one, they 18 didn't report the results, but they did say the 19 occurrence of kidney cancer was as expected from 20 the state rates. So there was no excess of kidney 21 cancer. 22 They did not report -- they didn't say 23 anything about bladder cancer, but they clearly 24 had the data. Okay.	Page 4266 1 excess; in fact, it's evidence of a deficit, a 2 negative association for skin cancer. 3 Q. So have you considered studies that 4 Doctor Werntz relied upon in reaching his 5 conclusion that there was a risk of bladder, 6 kidney and skin cancer here? 7 A. I certainly have. Many of these are the 8 studies that Doctor Werntz used. 9 Q. And have you considered additional 10 studies you found through your own research? 11 A. I have indeed, yes. 12 Q. And what is your conclusion as to whether 13 the people living in the area of the smelter are 14 at a significantly increased risk of bladder 15 cancer, kidney cancer or skin cancer? 16 A. Well, there's not scientific evidence 17 that would support that conclusion. It's that 18 simple. 19 Q. So the next element that Doctor Werntz 20 looked at - we've talked about arsenic - he looked 21 at cadmium. What does the scientific evidence say 22 about whether cadmium causes cancer and under what 23 circumstances? 24 MR. GALLAGHER: Your Honor, if -- I'm
Page 4265 1 Tokudome had the data, didn't report the 2 results for any other cancer sites. 3 Hinwood, the Australian study, did report 4 the risks for bladder cancer and kidney cancer and 5 you can see that there's a slight negative 6 association for bladder and a slight positive 7 association for kidney. Neither of them is 8 significant. Confidence interval would include 1. 9 The Murray, Utah study did not report any 10 other cancers, but they concluded that the site 11 was not an apparent public health hazard. If 12 there had been an excess of cancer, I doubt that 13 they would have concluded there's no public health 14 hazard. 15 And then the last one is Wong. That's a 16 study that I haven't presented before. That's 17 actually a study of skin cancer, and that's among 18 residents near the Anaconda copper smelter and 19 Anaconda mine where Doctor Wong did not give the 20 odds ratios; he simply presented the skin cancer 21 incidence rates and showed that they were 22 significantly lower around the copper smelter than 23 in nonexposed counties in the same state. 24 So again, that's not evidence of an	Page 4267 1 starting a new topic, so if you want to take a 2 mid-morning break, now would work, or we can -- 3 THE COURT: Why don't you do that. Do 4 you want the witness to answer your question, and 5 then we'll take our break? If he recalls the 6 question. 7 MR. GALLAGHER: I'll ask it again and so 8 -- 9 THE COURT: Okay. 10 Q. What does the scientific evidence -- what 11 does the literature say about cadmium and whether 12 it causes cancer and under what circumstances? 13 A. Well, IARC has classified cadmium as a 14 human carcinogen, and it is associated with lung 15 cancer under circumstances of occupational 16 inhalation exposure. So if you inhale cadmium 17 dust or fume in sufficient quantities, there is an 18 increased risk of lung cancer. 19 Now, some of the agencies disagree. They 20 put IARC -- or they put cadmium in the next 21 category down, "probably carcinogenic," by IARC 22 says it's a human carcinogen, and I would agree 23 with that. The evidence is sufficient -- 24 Q. Is --

Page 4268 1 A. -- lung cancer by inhalation. 2 Q. Is cadmium in the literature, including 3 the IARC, is cadmium associated with any other 4 cancers through any other route of exposure? 5 A. The simple answer is no. Cadmium is a 6 metal that's been investigated extensively since I 7 was in Public Health School -- before I was in 8 Public Health School in the late '70s. There was 9 an initial report that it was associated with 10 prostate cancer, and a lot of people tried to 11 investigate that. It didn't bear out. 12 I know Doctor Werntz claims it was 13 associated with kidney cancer. That doesn't bear 14 out. The only one that bears out is the lung 15 cancer association. 16 Q. Why don't we come back to that topic and 17 the evidence about the risk around this smelter 18 after our break. 19 A. Okay. 20 THE COURT: Ladies and gentlemen of the 21 jury, why don't you go with your bailiff about ten 22 or fifteen minutes, then we'll call for the 23 continuation of the testimony. 24 (The jury exited the courtroom.)	Page 4270 1 and he can discuss the ATSDR study which has been 2 talked about a lot. I think that Doctor Werntz 3 yesterday opened the door to further discussion 4 beyond the ATSDR study when he testified in 5 response to questioning that -- the question was, 6 "If you took blood lead tests of people who lived 7 in the class area in light of ongoing exposure," 8 so talking about today, "would their blood lead 9 test be elevated," and he answered "Yes," which is 10 the question, and he volunteered, "Yes, that was 11 what was found," the suggestion being -- 12 MR. BARR: Could you keep that open? 13 THE COURT: Didn't he also -- and then he 14 went on to talk about the ASTR -- whatever the 15 acronym is 16 MR. BARR: ATSDR. 17 THE COURT: Thank you. 18 -- and that, so -- 19 MR. GALLAGHER: He did, but that was with 20 respect to 1996. And the question where he 21 volunteered, "That was what was found" was the 22 question that was about "ongoing today." 23 So the suggestion I think he left with 24 the jury was that there are tests out there that
Page 4269 1 THE COURT: Counsel, why don't we take 2 about ten minutes, please. 3 (A recess was taken after which the 4 proceedings continued as follows:) 5 MR. BARR: Your Honor, may we approach 6 real quick? 7 THE COURT: Yes, please. 8 (Counsel approached the bench and the 9 following proceedings were had out of the hearing 10 of the audience:) 11 MR. GALLAGHER: There's one issue I 12 wanted to raise that may come up in the further 13 examination of Doctor Garabrant. I understand 14 there's been a ruling on Lenora Perrine's blood 15 lead level. 16 One of the topics we're addressing is 17 medical monitoring and whether there's sufficient 18 evidence of significant exposure, and one of the 19 things that he's going to talk about is that you 20 would see that in blood lead, you would see it if 21 it were there, and I'm prepared to stop right 22 there -- 23 THE COURT: Okay. 24 MR. GALLAGHER: -- but I think that --	Page 4271 1 they haven't heard about. 2 THE COURT: Now, it's -- I mean, he was 3 talking about the ATSDR study, so let me -- and I 4 appreciate you raising it in this manner, so -- 5 MR. GALLAGHER: Okay. 6 THE COURT: -- that's the exact way to do 7 it to keep from getting crabbed at by the judge. 8 I remember those days. So let me ask -- and if 9 you need to woodshed the witness -- 10 MR. GALLAGHER: He knows that there's a 11 ruling, your Honor, and he knows that he's not to 12 talk about it, and what I have prepared is a 13 question of, "Is there any evidence?" 14 And his answer to that is, "No," he's not 15 going to talk -- he's going to say, "I have not 16 seen any evidence of elevated blood levels," and 17 we can leave it at that, that and the ATSDR -- 18 THE COURT: Okay, thank you. 19 (Counsel returned to counsel table and 20 the proceedings continued in the presence and 21 hearing of the audience as follows:) 22 THE COURT: Mr. Bailiff, you may return 23 the jury to its box, please. 24 (The jury returned to the courtroom.)

Page 4272 1 THE COURT: All 11 of our jurors again 2 being present before the Court and the parties and 3 the respective counsel, you may continue your 4 examination, sir. 5 BY MR. GALLAGHER: 6 Q. Before we took our break, Doctor, you 7 were telling us now what the scientific literature 8 says about the risks of cancer and exposure to 9 cadmium. Have you prepared a slide that -- like 10 the one you did for arsenic that summarizes that 11 body of knowledge? 12 A. Yes. 13 Q. And is this the slide you prepared? 14 A. Yes. 15 Q. If you could, tell us what you're 16 illustrating here. 17 A. Okay. What the slide is showing is that 18 cadmium is associated with lung cancer in settings 19 where there is inhalation in occupational 20 settings. Okay? So the evidence that cadmium is 21 a carcinogen really comes from studies of workers 22 who have been heavily exposed in industry and they 23 are at increased risk of lung cancer. 24 I'm not aware of any evidence that links	Page 4274 1 Q. Now, with -- with arsenic, we went 2 through a list of studies, and you told us what 3 the studies showed about whether living near a 4 smelter puts you at an increased risk. Did you do 5 a similar analysis for cadmium and the risk of 6 lung cancer? 7 A. I did. 8 Q. And is this the slide you prepared to 9 summarize your review of the studies? 10 A. Yes. 11 Q. First let me ask you: What studies did 12 you include in your review on cadmium? 13 A. Okay. These are the same studies that I 14 had on the slide of arsenic in lung cancer because 15 the smelter studies all included arsenic and 16 cadmium exposure. So the fact of living near a 17 smelter relates to the issue of whether cadmium 18 carries risk of lung cancer as well as it did for 19 the issue of whether arsenic causes -- puts you at 20 increased risk of lung cancer, so it's the same 21 set of studies, and it's the same set of results 22 that we have already discussed. 23 And it's the same conclusion that the 24 evidence in populations who have lived near
Page 4273 1 cadmium to cancer via drinking water. I don't 2 believe that is an exposure pathway that carries 3 any risk of cancer. 4 You know, it should be mentioned that 5 cadmium is in the food supply, small amounts of it 6 occur in fruits and vegetables and grains, and we 7 ingest cadmium in small amounts every day, and 8 there's no evidence that that's a risk factor for 9 cancer. 10 Q. Doctor Werntz offered the opinion that we 11 should have medical monitoring for lung cancer and 12 a variety of other cancers, I think kidney cancer 13 was one of them based on the exposure to cadmium 14 in the class area. 15 Do you have an opinion as to whether 16 there is scientific evidence to support a finding 17 that people who live in the class area are at 18 significantly increased risk of any cancer due to 19 exposure to cadmium? 20 A. Well, I do have an opinion, and I think 21 that the materials I've just shown strongly 22 support an opinion that there's no reason to think 23 there's increased risk of cancer from cadmium 24 exposure resulting from living near the smelter.	Page 4275 1 smelters does not support a conclusion that they 2 are at increased risk of lung cancer as a result 3 of cadmium exposure. 4 Q. Same -- same studies, same conclusion as 5 you had with arsenic? 6 A. That's correct. 7 Q. Now, Doctor Werntz also talks about 8 kidney cancer and the potential for -- the risk of 9 kidney cancer here. Have you analyzed that 10 question by looking at the studies and the 11 scientific literature? 12 A. I have. 13 Q. Let's first talk about the studies you 14 looked at in particular. What did you look at 15 with respect to studies about people living near 16 or exposed near smelters and the risk of kidney 17 cancer? 18 A. There are really only the three, and we 19 discussed them all previously: The Bartlesville, 20 Oklahoma study of residents near a zinc smelter 21 where they said the occurrence of kidney cancer 22 was as expected from the State rates, which is a 23 round-about way of saying "No increased risk." 24 The Tokudome study which didn't report

Page 4276 1 the results, and my comment earlier is worth 2 saying again, the fact that they didn't report it, 3 it's reasonable -- it's reasonable to conclude 4 that there wasn't any significant excess. 5 And then the Murray, Utah study by ATSDR, 6 residents near a lead smelter, ATSDR considered 7 that smelter site to be no apparent public health 8 hazard. If there had been a significant excess of 9 kidney cancer, I think they would have said, and 10 they didn't. They said, "There's no apparent 11 public health hazard." 12 Q. Now, beyond the smelter studies, is there 13 a body of scientific knowledge, studies that have 14 been done, to test the question of whether cadmium 15 causes kidney cancer through an inhalation 16 pathway? Whether you -- whether it's near a 17 smelter or in some other scenario, is there 18 literature on that? 19 A. Oh, yeah. Yeah, there is literature on 20 cadmium and kidney cancer that has looked 21 carefully at that, and it is not supportive of the 22 conclusion that cadmium causes kidney cancer. 23 Q. Did you prepare a slide to summarize your 24 review of that -- another body of literature	Page 4278 1 the hypothesis that cadmium is associated with 2 kidney cancer doesn't bare out. It's not. 3 Q. Has this question of whether cadmium 4 causes kidney cancer, has it been extensively 5 studied by scientists looking for the causes of 6 cancer? 7 A. I believe that would be fair to say. 8 Q. And in that entire body of scientific 9 knowledge -- including the IARC report and 10 everything else -- is there a scientific basis for 11 concluding that people in the class area are at a 12 significantly increased risk of kidney cancer as a 13 result of exposure to cadmium? 14 A. No, there's not. There is not a 15 scientific basis for that conclusion, and I don't 16 think that's a valid conclusion. 17 Q. Now, earlier you showed us the summary of 18 the IARC monograph and the different 19 classifications for substances and whether they're 20 carcinogens. You recall that? 21 A. Yes. 22 Q. And you showed us some slides that show 23 that arsenic and cadmium can cause cancer in 24 certain circumstances, right?
Page 4277 1 beyond the smelter studies and what it has to say 2 about whether cadmium causes kidney cancer? 3 A. Yes. 4 Q. And this is your slide? 5 A. Yes, this is my slide. 6 Q. Tell us what you found when you looked at 7 the body of literature on this question of whether 8 cadmium causes kidney cancer. 9 A. Yeah. The agencies that have reviewed 10 the evidence of carcinogenicity of cadmium have 11 not concluded that cadmium is associated with 12 kidney cancer in humans. They haven't made that 13 conclusion. 14 And there have been a good number of 15 studies of cadmium exposure in industrial 16 settings, typically workers, battery workers 17 exposed to cadmium, you know, nickel cadmium, 18 NiCad batteries, lab workers exposed to cadmium, 19 cadmium alloy workers in the metal industry, 20 cadmium pigment workers. 21 Cadmium pigments have been used 22 extensively in paints. They produce beautiful 23 oranges and yellows and reds. And other cadmium- 24 exposed workers in a variety of industries, and	Page 4279 1 A. Yes. 2 Q. Are you saying that these substances -- 3 that arsenic and cadmium -- that they don't cause 4 cancer? 5 A. No. No. 6 Q. To sum it up for us, what is your opinion 7 with respect to arsenic and cadmium, and 8 specifically the question of whether there is a 9 significantly increased risk of cancer as a result 10 of exposure to arsenic or cadmium that you had in 11 this class area? 12 A. Okay. Arsenic causes lung cancer by 13 inhalation in industrial settings. There is not 14 evidence that arsenic puts people at increased 15 risk of lung cancer from living near smelters. 16 Arsenic causes lung, kidney, bladder and 17 skin cancer when it's in your drinking water. I'm 18 not aware that drinking water is an issue in this 19 case. It wasn't in Doctor Brown's report; it 20 wasn't in Doctor Werntz's report; and I haven't 21 seen any evidence that there was drinking water 22 contamination sufficient to have put anybody in 23 increased risk. 24 So I think the drinking water exposures

Page 4280 1 are not -- there's no basis to say that that's a 2 risk factor. 3 Cadmium causes lung cancer by inhalation 4 in industrial settings. I think that IARC has 5 that right, even though other agencies don't 6 agree, I think IARC has that right. 7 There is not evidence that people who 8 live downwind of smelter sites are at increased 9 risk of lung cancer if they inhaled or -- I should 10 say if there is cadmium exposure from those sites. 11 There's just not evidence that they're at an 12 increased risk. 13 So -- and I guess lastly, cadmium is not 14 known to cause kidney cancer, as Doctor Werntz 15 believes. The science doesn't support that 16 conclusion. 17 So when you add it all up, I don't think 18 that there is a scientific foundation that in any 19 way supports a conclusion that there's increased 20 risk of those cancers as a result of living near 21 this site. 22 There's simply no basis for it. 23 Q. The last thing that Doctor Werntz said on 24 the subject of cancer was that exposure to lead	Page 4282 1 present here. The compounds that may be present 2 here, I don't think there's even evidence in 3 animals to support it. 4 Q. Yesterday Doctor Werntz, in his 5 testimony, talked about the IARC monograph on lead 6 in Volume 23, and they put up on the screen this 7 quote from that monograph, and this quote says, 8 "There is sufficient evidence that lead subacetate 9 is carcinogenic to mice and rats and that lead 10 acetate and lead phosphate are carcinogenic to 11 rats." 12 Just so it's clear, do you believe that 13 this is sufficient evidence from which to conclude 14 that people in the class area are at a 15 significantly increased risk of any cancer as a 16 result of exposure to lead? 17 A. No. 18 Q. And can you explain for us, using the 19 monograph, this quote that Doctor Werntz pointed 20 to, can you explain to us why not? 21 A. Well, you know, the first is, you know, 22 lead acetate -- okay, vinegar is acidic acid, 23 okay, and so lead acetate is the lead salt of 24 vinegar. Now, I don't think that lead acetate is
Page 4281 1 puts you at an increased risk of cancer. What 2 does the scientific literature say about lead and 3 cancer? 4 A. We've already discussed that none of the 5 agencies rank lead as a human carcinogen. Most of 6 them rank it as a probable or possible carcinogen 7 based on animal evidence, recognizing that the 8 human evidence is not adequate to say that this is 9 a human carcinogen. 10 If you look a little more deeply into the 11 animal evidence, the evidence that lead compounds 12 cause cancer in animals -- and we're talking about 13 rodents, rats, mice -- is evidence derived from 14 lead compounds like lead acetate, lead subacetate, 15 lead phosphate. 16 It's not -- there is not evidence that 17 compounds like lead oxide, lead sulfide, lead 18 sulphate cause cancer even in animals. And those 19 compounds, lead acetate, lead subacetate, lead 20 phosphate, I don't even think are in issue in this 21 case, I don't think that they would be present in 22 a smelter. 23 So lead may cause cancer in animals, but 24 it involves lead compounds that really aren't	Page 4283 1 present in the smelting environment. I can't 2 imagine what it has to do with smelting. I was a 3 chemical engineer in college, it doesn't make 4 sense to me. 5 And the same thing with lead subacetate, 6 it's another salt -- it's another lead salt of 7 vinegar. It's not here. So the evidence that 8 Doctor Werntz is relying on is about lead 9 compounds that are not at issue here. 10 So that's -- so that's Issue No. 1. 11 Issue No. 2, I don't dispute that those 12 compounds are carcinogenic to rats or to mice, but 13 it's still a leap of faith to say that they're 14 carcinogenic to humans, and as you can see in this 15 quote, IARC does not make that leap of faith. 16 It says, "In the absence of adequate 17 human data, it's reasonable for practical purposes 18 to regard these compounds as if they presented a 19 carcinogenic risk to humans." 20 What that means is, "We don't have 21 evidence, but in order to be protective of humans, 22 we'll assume that the evidence -- that these could 23 be humans carcinogens." That's a reasonable 24 decision to make, but it's not evidence that they

Page 4284 1 cause cancer. 2 I think the most important point here is 3 that these are not -- these are not compounds that 4 would be present in a smelting environment. This 5 is not what's going on. 6 Q. So the first point we made, the materials 7 that are being talked about here are forms of lead 8 that are not at issue in this case? 9 A. As far as I understand. 10 Q. And the second point would be that what 11 this monograph shows is that those materials that 12 aren't at issue in this case cause cancer to mice 13 and rats, right? 14 A. Right. 15 Q. And is there any basis for extrapolating 16 from that to a conclusion that people who live in 17 this class area near the smelter are at a 18 substantially or significantly increased risk of 19 any kind of cancer? 20 A. No. That's not a reasonable conclusion 21 from this -- from this scientific evidence. 22 Q. Now, have people studied -- not rats and 23 mice, but have people looked at whether there is 24 human evidence that exposure to lead causes	Page 4286 1 evaluating Doctor Wermtz's opinion that lead 2 causes cancer in humans? 3 A. Yes. 4 Q. And what is this report? 5 A. Okay. Kyle Steenland, I know Doctor 6 Steenland, he's on the faculty at Emory now. He 7 used to be at NIOSH. He's a statistician, and 8 Paolo Boffetta is a physician. He's in charge of 9 the environmental epidemiology unit at IARC in 10 France, and a very talented scientist, they both 11 are. 12 They tried to summarize -- or did 13 summarize -- the epidemiologic evidence regarding 14 lead and cancer in humans, and this was published 15 in the American Journal of Industrial Medicine in 16 2000. 17 So it's seven years old. The IARC 18 publication is newer and seeks to do roughly the 19 same thing. But yeah, this is a well-written 20 summary of the evidence, in my opinion. 21 Q. And what was their conclusion about 22 whether there is a risk of cancer in humans from 23 exposure to lead? 24 A. Well, I don't know if you can blow it up,
Page 4285 1 cancer? 2 A. Oh, yeah, there's a big body of 3 literature on that. 4 Q. And has -- has anybody made an effort to 5 go through that body of literature on the human 6 evidence and summarize it and say, "Is this 7 something that causes cancer" or "Is it not?" 8 A. Well, yes. First off, IARC has done 9 that. There's a whole book. That's what this 10 book is about that just came out earlier this 11 year. It's exactly aimed at that, and IARC's 12 conclusion was "Evidence is not adequate in 13 humans, we're not going to put it in Category 1, 14 the evidence is adequate in animals, and it's 15 based on exactly what's on the slide here, and 16 we're going to put it in Category 2A." 17 So the evidence in humans is not 18 adequate. 19 Q. Have you looked at the literature, 20 including a study by -- report by a gentleman 21 named Kyle Steenland and Paolo Boffetta called 22 "Lead and Cancer in Humans, Where Are We Now?" 23 A. Yes. 24 Q. Is this one of the things you read in	Page 4287 1 but their conclusion is right on the screen. 2 THE WITNESS: Actually, stay on the first 3 page, if you would. 4 A. Can you make that larger? 5 Q. Sure. Where do you want -- 6 A. Where it says "Conclusion." Right down 7 -- right there. Yeah, that's it. Okay. So their 8 conclusion is: "Overall, there is only weak 9 evidence associating lead with cancer; the most 10 likely candidates are lung cancer, stomach cancer 11 and gliomas." Gliomas are brain tumors. 12 Okay, so it's not that there's no 13 evidence, but it's weak evidence. 14 Okay. And then if we could go to their 15 conclusion at the end of the paper, right there, 16 maybe you could blow that up. 17 Okay. And so here -- here's how they 18 conclude their paper: "Despite the fact that lead 19 is one of the earliest recognized occupational 20 toxins," and you know, lead's been around for 21 thousands of years, abdomen and we know a 22 tremendous amount about its toxicity. 23 "There exists relative few studies of 24 cancer among lead-exposed workers with well-

25 (Pages 4284 to 4287)

Page 4288 1 documented high exposures." And of course, these 2 are the most informative studies for identifying 3 lead causes cancer. 4 They identify eight principal studies 5 with well-documented high exposures. "The 6 evidence is suggestive of an association with lung 7 cancer and stomach cancer but remains limited." 8 One of the problems is "a possible confounding by 9 arsenic is a concern." 10 In other words -- we talked about 11 confounding -- it's possible that the associations 12 that are observed between lead and cancer are 13 really due to arsenic that hasn't been properly 14 controlled for in the analysis. 15 So they're saying possible confounding 16 by arsenic is a concern, in the study that the 17 highest lung cancer, relative risk. There's 18 weaker evidence of an association of kidney cancer 19 and the gliomas, the brain tumors. Most of the 20 studies did not have data on dose-response that 21 would provide a better basis for inference than 22 comparisons of exposed to non exposed people. 23 One of things that I haven't discussed is 24 when you have a carcinogen, you expect to see that	Page 4290 1 says, "Oh, lead would lead to an increased risk of 2 cancer." 3 It's the studies that we've discussed. 4 It's not clear that there's increased risk to 5 cancer no matter what the exposures are, whether 6 it's arsenic or lead or cadmium or combinations of 7 the three. 8 And some of those studies were lead 9 smelter operations. 10 Q. So we talked about cancer risks, and now 11 I -- what I'd like to do is switch gears and talk 12 about risks of maladies and things other than 13 cancer, and my first question for you on that is: 14 Did you read Doctor Werntz's expert report, the 15 report where he laid out the medical monitoring 16 program that he thought was appropriate here and 17 the risks, the noncancer risks, that he thought 18 were -- he thought was an issue? 19 A. Yes. 20 Q. And did you notice in his report that in 21 his report, where he was talking about what 22 medical monitoring there should be here, that he 23 talked about the decreased renal function and lead 24 poisoning as the two main issues?
Page 4289 1 as exposure increases, the risk of cancer 2 increases. That's called dose response, all 3 right? So people with low exposure, low risk; 4 people with more exposure, higher risk; people 5 with very high exposure, higher risks even. 6 We look for those responses in the 7 studies, it's not there for lead. 8 So -- not to read the whole thing, the 9 bottom line is, when you come down, their final 10 conclusion is, there's only weak evidence and it 11 is not clear that lead is a human carcinogen. 12 That, I think, was a fair assessment in 13 19 -- in 2000, and I think that's a fair 14 assessment today. 15 Q. And did you -- did you review the studies 16 about exposure in the area of smelters, the ones 17 we talked about earlier, for arsenic and cadmium, 18 and did you see in those any evidence that people 19 who live near a smelter are at risk of cancer or 20 -- any kind of cancer because of lead? 21 A. Well, it's -- it's the same set of 22 studies we've already discussed, and we've looked 23 at the cancer risks, and the direct answer to your 24 question is, no, there's nothing in them that	Page 4291 1 A. I think that's a fair summary. 2 Q. Now, yesterday Doctor Werntz talked about 3 a whole lot of things that -- that might come up 4 with cadmium or arsenic, things like Raynaud's 5 disease and black foot disease. Are you prepared 6 to talk about the things that Doctor Werntz 7 identified as the reason for having medical 8 monitoring in this case? 9 A. Yes. 10 Q. So let me ask you, as a doctor, 11 considering whether there's a significant 12 increased risk of kidney -- renal failure or lead 13 poisoning, what's the first thing you would want 14 to see in your analysis? What would you -- what 15 would you look for first? 16 A. I'd want to look right away at whether 17 these people had elevated blood lead levels, 18 elevated blood cadmium levels, elevated excretion 19 of cadmium and arsenic. I mean, that's the best 20 index of whether people have been overexposed. 21 And it's the best way of starting to 22 determine whether in fact they're likely to have 23 had any damage or whether they're at any risk of 24 damage.

Page 4292 1 Q. Before we get to that topic, let me ask 2 you: In your teaching, do you teach risk 3 assessment? 4 A. Yes, I do. 5 Q. And are you familiar with the concept of 6 a risk assessment for determining whether people 7 are at a significantly increased risk of disease? 8 A. Yeah, sure. 9 Q. When it came to the cancer risks, did 10 Doctor Werntz rely on somebody to perform a risk 11 assessment for him? 12 A. Doctor Brown did a risk assessment for 13 cancer risks. 14 Q. When it comes to the noncancer risks, 15 things other than cancer, like the Raynaud's 16 disease and black foot disease that he mentioned 17 yesterday, can you do a similar risk assessment 18 for those things? 19 A. You certainly can. 20 Q. Did Doctor Brown do a risk assessment for 21 those things? 22 A. No. 23 Q. Did Doctor Werntz? 24 A. No.	Page 4294 1 and say, "Well, that applies to these other 2 things" that Doctor Werntz has talked about. 3 Q. You mentioned a couple concepts that I'd 4 like to have you explain for us a little in 5 detail. The first was you mentioned dose. Is 6 there a difference between the exposure you have 7 in your environment, what might be in your soil or 8 in your dust, and the dose that you get in your 9 body? 10 A. Yes. 11 Q. What is the difference between those two 12 things? 13 A. That's an important concept. We 14 typically think of exposure as the concentration 15 of things outside of our body, so the 16 concentrations of arsenic in air, the 17 concentration of cadmium in water, in other words, 18 the things in the environment around us, that's 19 exposure. 20 Dose refers to the amount in your body, 21 and the concept is, it has to have crossed the 22 barrier between me and the outside world, whether 23 that barrier is the skin or the lining of my 24 respiratory tract or the lining of my GI tract.
Page 4293 1 Q. Now, would it be appropriate, as a 2 scientist or a doctor, to take a risk assessment 3 for cancer and draw conclusions about whether 4 there was a risk of noncancer diseases like renal 5 failure? 6 A. No, you can't do that. 7 Q. Why -- why can't you do it that way? 8 A. Well, the calculation of risks has to do 9 with some very specific data on what the risks 10 are, and the risks for cancer are typically 11 calculated using cancer slope factors, whereas 12 these other things have different mechan -- these 13 other health effects have different mechanisms and 14 they typically involve calculations based on ideas 15 that there is a threshold or a dose above which 16 you get damage and below which you don't get 17 damage. 18 So it's one of the principles in risk 19 assessment foremost noncarcinogens that you have 20 to identify a no-observed adverse effect level, 21 and that exposures below that don't have any 22 adverse effects. Exposures above, then you 23 calculate the risk. And that wasn't done here. 24 You can't take a cancer risk assessment	Page 4295 1 It has to cross the barrier to become dose. 2 And we measure dose routinely by 3 measuring blood levels, measured blood leads, 4 blood cadmium or blood arsenic, or by measuring 5 how much we excrete, so you can measure urinary 6 cadmium or arsenic. Sometimes we measure dose in 7 the tissues, so you can actually measure -- for 8 example, the lead -- you can measure lead in bone. 9 Okay, you can actually use a technique 10 that shines a beam of neutrons through bone and 11 measure how much lead is in the bone. Those are 12 measures of dose, because they relate to what's in 13 your body, not what's in the outside world. 14 And as a physician, we routinely measure 15 internal dose of chemicals. That's part of what 16 we're trained to do. 17 Q. Now, the other concept you mentioned was 18 the concept of a threshold, and when you talk 19 about things like the risk of renal failure due to 20 exposure to arsenic and cadmium or -- is there 21 some threshold below which the exposure, the dose, 22 is not an issue, and above which you start to see 23 problems? 24 A. Yeah. For example, for cadmium, there's

Page 4296 1 a very solid and substantial body of literature 2 that establishes that there are concentrations of 3 cadmium in the kidney above which you start to see 4 damage and below which you do not, and that those 5 correlate with levels of cadmium in blood. 6 So there's clearly a threshold for kidney 7 damage, and it's a good thing there is, because we 8 all get cadmium in our diet in very small amounts, 9 and it doesn't do any damage. And so the body is 10 able to handle small amounts without any damage, 11 and if you get to a certain dose, then you can 12 have damage. 13 Q. Now, with respect to the noncancer risks 14 that Doctor Werntz identified, can you reach a 15 conclusion about whether people are at a 16 significantly increased risk of those things 17 without knowing if you are at an exposure that 18 puts you above that threshold? 19 A. No, you -- you have to have some 20 scientific evidence that either there's exposure 21 adequate to put you at increased risk or there's 22 internal dose adequate to put you at increased 23 risk. And of the two, I'd strongly prefer the 24 internal dose, because it's much closer to the	Page 4298 1 A couple of others are called 2 retinol-binding protein, and the third is 3 N-acetylglucosamine, so RBP and NAG and Beta-2 4 microglobulin. It would be very easy to look for 5 those proteins in the urine to establish whether 6 anybody had evidence of early kidney dysfunction. 7 But the earliest signs of kidney damage 8 can be detected by looking for those proteins in 9 the urine. 10 Q. Is that another way of saying that you 11 could test and get evidence before drawing the 12 conclusion that there was a significant increased 13 risk? 14 A. Well, it's not only that you can; you 15 should. 16 Q. Based on the evidence -- well, let me ask 17 it this way: Have you seen any evidence in this 18 case of tests showing increased levels that 19 approach the threshold and would put these people 20 in the class area at a significantly increased 21 risk of decreased renal function or renal failure? 22 A. No, I've seen no evidence of blood 23 cadmium, urinary cadmium or urinary excretion of 24 Beta-2 microglobulin or anything else.
Page 4297 1 issue of whether the tissue has been damaged. 2 Q. Now, on the question of decreased renal 3 function and renal failure, one of the things that 4 Doctor Werntz wants to monitor for here, have you 5 considered whether the plaintiffs, the class 6 members, are at a significantly increased risk of 7 those things as a result of their living in the 8 class area? 9 A. Of -- of decreased renal function and 10 renal failure? 11 Q. Yes. 12 A. Well, there's no evidence that would 13 support that conclusion. 14 Q. What kind of evidence would you need to 15 see before you could reach that conclusion? 16 A. Well, I -- I would want to see a blood 17 cadmium and a urine cadmium level, and ideally, 18 I'd want to see -- when cadmium damages the 19 kidney, it damages the part of the kidney that's 20 responsible for concentrating the urine, and when 21 those cells get damaged, low molecular weight 22 protein, small proteins, are excreted in the 23 urine, and one of those is called Beta-2 24 microglobulin.	Page 4299 1 Q. Now, the other area in which Doctor 2 Werntz has proposed medical monitoring for 3 noncancer risks is in the area of lead poisoning. 4 Do you have a conclusion -- an opinion whether 5 people in the area, the class area, are at a 6 significantly increased risk of lead poisoning or 7 effects from exposure to lead? 8 A. There's no evidence that they're at 9 increased risk of lead poisoning, none at all. 10 And that's very easy to assess. 11 Q. Is this again an area where before 12 drawing the conclusion that there was a 13 significantly increased risk where you would want 14 to see evidence in the form of testing of levels 15 in the bodies? 16 A. Yeah, sure, you'd do a blood lead test. 17 We do them routinely. 18 Q. Doctor Werntz was asked yesterday, "You 19 would expect, wouldn't you, if you took blood lead 20 tests of people who lived in the class area and 21 there was ongoing exposure that you say is 22 happening, the blood leads would be elevated." 23 And he said, "Yes, you'd expect to see 24 that." Do you agree that if there were elevated

Page 4300 1 levels of exposure in the class area, that you 2 would see that in blood tests of people who lived 3 in the class area? 4 A. Yes. 5 Q. Have you seen any evidence in this case 6 that people who live in the class area have 7 elevated blood lead levels? 8 A. No, I have not. ATSDR did a study in 9 1996 of children. They tested 25 children, and 10 the average blood lead levels were entirely 11 normal. And I think ATSDR's conclusion was there 12 was no evidence of excessive lead exposure. 13 Q. So let me go back to the cancer risks and 14 ask you, Doctor, based on your review of all the 15 evidence, do you think the people living in the 16 class area are at a significantly increased risk 17 of any cancer as a result of exposure to arsenic, 18 cadmium or lead? 19 A. No. 20 Q. And do you think the people living in the 21 class area are at a significantly increased risk 22 of any noncancer diseases as a result of their 23 exposure to arsenic, cadmium or lead? 24 A. You know, the answer is again no. But	Page 4302 1 opinions, and as we've seen for cadmium, IARC says 2 it's a carcinogen, and other agencies don't. 3 There is some range for disagreement. 4 However, I think it's important to 5 realize that the disagreements are usually 6 relatively small, as to whether cadmium is 7 Category 1 or a 2A, is -- is basically everybody 8 saying, "Well, there's some evidence, there's 9 reason to be concerned, but it doesn't meet the 10 standard for proof in humans," and that's sort of 11 the range of disagreement. 12 Q. Well, we're going to get into this in a 13 little bit. You testify a lot in trial. That's 14 fair to say, you're a fairly experienced 15 professional witness, aren't you? 16 A. I have testified in trials in the past. 17 Q. And you understand that in a trial 18 setting, it's very common for one expert to say 19 one thing and another expert to say something 20 totally different. 21 A. I think that different people testify as 22 to different opinions. 23 Q. And when a jury has to decide which one 24 is right, issues like credibility are very
Page 4301 1 the real answer is that there's simply no basis 2 for drawing that conclusion. You know, and as a 3 scientist, if you're going to draw a conclusion, 4 you have to have something to base it on. 5 And that's simply not here. 6 MR. GALLAGHER: Thank you, your Honor, I 7 have nothing further. 8 THE COURT: Mr. -- Mr. Barr, you may 9 inquire, if you'll fetch the microphone. 10 Mr. Barr, you may inquire. And if you'll 11 be mindful of high noon, I'm sure the jury would 12 appreciate it. 13 MR. BARR: Thank you, your Honor, I'll 14 try to be fairly quick with this first step. 15 CROSS EXAMINATION 16 BY MR. BARR: 17 Q. Now, Doctor Garabrant, we've heard a lot 18 about your testimony and the opinions you have to 19 offer in this case and your review of the 20 scientific evidence. Would you agree with me that 21 reasonable unbiased scientists can look at a body 22 of evidence and come to different conclusions 23 about what that evidence shows? 24 A. You know, certainly there is a range of	Page 4303 1 important, aren't they? 2 A. Well, I think the key issue for 3 scientific testimony is whether testimony has a 4 scientific foundation. 5 Q. So things like the scientists' bias or 6 possible outside influences could affect the 7 weight a jury may give to that testimony, couldn't 8 it? 9 A. Well, I think the key issue is whether 10 there's scientific evidence that underlies the 11 testimony. 12 Q. Now, are you aware of any reason Doctor 13 Werntz would come into this courtroom and mislead 14 this jury about what the scientific evidence shows 15 about arsenic, cadmium and lead? 16 A. I do not know Doctor Werntz, and I have 17 no knowledge about his background. 18 Q. So you're not aware of any reason he 19 would come in and do that, are you? 20 A. Like I said, I don't know Doctor Werntz, 21 I don't know anything about his background. 22 Q. Well, you know he's a -- he's with -- 23 affiliated with West Virginia University, right? 24 Do you know that?

Page 4304 1 A. I do know that. 2 Q. He's an actual practicing physician that 3 sees patients every day. You understand that, 4 don't you? 5 A. I am aware that he's on the faculty of 6 West Virginia as, I believe, an assistant 7 professor or assistant clinical professor. 8 Q. Okay. And again, you don't have any 9 reason to suspect that he would come in and 10 mislead this jury because of his background or 11 outside influences, do you? 12 A. Well, I've already said, I don't know 13 him, and I don't have any information on his 14 background. 15 Q. Okay. Well, let's talk a little bit 16 about your background, while we're talking about 17 backgrounds. Now, you already told me that you 18 testify a lot in trial. I mean, this is something 19 you very commonly do, isn't it? 20 A. I didn't say that. But I have testified 21 in trial, and I think courts need scientists to 22 explain complicated science so that it can be 23 understood. And yes, I do that. 24 Q. I'm going to put this up on the Elmo. If	Page 4306 1 Q. Okay. Well, hold on one second, since 2 you -- you don't, you know, necessarily believe me 3 on this -- 4 A. It's not that I don't believe you, it's 5 that I don't recall. 6 Q. If I show you your deposition testimony, 7 would that maybe refresh your recollection on it? 8 A. If I testified to it, it's true. 9 Q. Okay. So you don't -- you don't disagree 10 that if you testified in your deposition that was 11 taken in this case that you testified two to three 12 times a year in trial for the past ten years, you 13 don't disagree with that, do you? 14 A. If that's what my testimony is, yes, I 15 agree with it. 16 Q. Now, you've been testifying as an expert 17 what, since the mid 1980s? Is that fair? 18 A. No, I testified once in, I think it was, 19 1986, and to the best of my recollection, no, I 20 didn't testify in any consistent pattern in the 21 next few years. But certainly for the past ten 22 years, I have been asked to come into court to 23 explain epidemiology to juries. 24 Q. Okay. And we're going to talk about that
Page 4305 1 you want a copy of this, I can bring it up to you, 2 but I think you'll be able to see it from there. 3 Can you read that, Doctor? 4 A. Yes. 5 Q. Or do you want your own copy? 6 A. No, I can read it. 7 Q. Okay. Now, this is a list that you 8 prepared of legal testimony you have presented 9 since 2002, is it not? 10 A. Yes. 11 Q. And there are, what -- how many cases, 12 you think, on here? 40 maybe? 40 some 13 depositions and trial testimony? 14 A. I don't know the number. I think it's -- 15 it shows that I testify either in deposition -- 16 well, I testify in deposition probably six times a 17 year, eight times a year, and I testify in trial 18 maybe twice a year for the past four years. 19 Q. Well, it's actually the past ten years 20 that you testified in trial two to three times a 21 year, isn't it? 22 A. I don't think I've been in trial twice a 23 year for ten years, but I testify typically two to 24 three times a year in trials.	Page 4307 1 first case you testified in in just a little bit. 2 But would you agree with me that legal testimony 3 or outside consulting away from your job -- legal 4 testimony comprises 50 percent of your income? 5 A. That's a reasonable estimate, yes. 6 Q. So 50 percent of the money you take home 7 every year is from testifying as an expert for 8 primarily defendants, isn't it? 9 A. I do work as an expert consultant in 10 litigation matters, and it does provide probably 11 half of my total income. 12 Q. And excluding Workers' Compensation 13 matters, you've testified that only once in your 14 career have you ever testified for a plaintiff. 15 A. Why would you exclude the Workers' 16 Compensation? I testified on behalf of plaintiffs 17 many times. 18 Q. In other than Workers' Compensation, have 19 you ever, in a trial setting, testified for a 20 plaintiff other than the one time that we're going 21 to discuss here today? 22 A. I've testified on behalf of plaintiffs in 23 Workers' Compensation many, many times, and on 24 behalf of defendants many times. If you want to

Page 4308 1 break it down into Workers' Comp versus 2 nonWorkers' Comp, I have testified on behalf of 3 defendants in nonWorkers' Comp almost exclusively. 4 Q. Well, except for one case. 5 A. That's correct. 6 Q. Now, would it be fair to say that you've 7 testified before that you make \$300,000 to 8 \$350,000 a year as an expert witness? 9 A. It would be fair to say that that has 10 been my income for my consulting work related to 11 litigation, not always as an expert witness. 12 Q. But it provides significant income to 13 you, doesn't it? 14 A. Yes, it does. 15 Q. Now, you told the jury that you're board 16 certified in, I believe it was, occupational and 17 environmental medicine and internal medicine; 18 isn't that right? 19 A. Yes. 20 Q. Now, isn't it true, Doctor, that in your 21 current practice, you see less than one patient 22 per week? 23 A. That would be correct. I spend most of 24 my time now doing research.	Page 4310 1 look carefully at the evidence, and we undertook a 2 series of research studies to see whether asbestos 3 exposure was associated with colorectal cancer, 4 and in fact, we published in 1992 a case control 5 study, very nicely done case control study, where 6 we couldn't find an association between asbestos 7 exposure and risk of colorectal cancer. 8 That was published in the American Journal of 9 Epidemiology. And then we followed that up with a 10 meta-analysis, a summary analysis like we've been 11 talking about that IARC does, looking at all of 12 the published literature on asbestos exposure and 13 colorectal cancer in which, when you added the 14 studies up carefully, it really didn't bear out 15 that there was clear evidence of any increased 16 risk. 17 Q. So when you testified in 1986, '87, that 18 time frame -- is that about right? 19 A. Somewhere in the mid '80s. 20 Q. -- you testified that there was adequate 21 scientific evidence, and now you're saying you 22 were wrong, there wasn't adequate scientific 23 evidence? 24 A. At the time I testified, I believe the
Page 4309 1 Q. Okay. So you aren't out there as a 2 clinician practicing medicine, treating people 3 primarily, are you? 4 A. Well, I do complex evaluations. I see 5 people who are referred because they have very 6 complicated issues related to chemical exposures, 7 and they take a lot of time. I typically spend 8 two hours with a patient, and then I have to 9 research the issue and write a report. 10 So I'll typically spend a half a day on a 11 patient. 12 Q. Now, I want to talk to you a little bit 13 about the first case you were ever hired as an 14 expert, the asbestos case. Do you remember that 15 case? 16 A. I do. 17 Q. That was a case about a gentleman who was 18 claiming he had colorectal cancer because of his 19 exposure to asbestos, was it not? 20 A. I believe that's correct. 21 Q. And you testified in that case that there 22 was adequate scientific evidence that the asbestos 23 fibers caused his colorectal cancer, didn't you? 24 A. Yes, I did. And that case caused me to	Page 4311 1 evidence supported the conclusion that asbestos 2 was associated with increased risk of colorectal 3 cancer. After we did our own research and 4 additional studies were published and we 5 summarized them, I felt that the scientific 6 evidence had changed, and I published it. 7 Q. And in 1986/'87, you were sworn under 8 oath to testify to the truth, weren't you? I 9 mean, that -- you -- you swore to tell the truth, 10 didn't you? 11 A. I certainly was, and I did. 12 Q. But now you're saying when you testified 13 that there was no -- that there was adequate 14 scientific evidence, you just didn't know? And 15 you testified to that anyway? I mean, I'm a 16 little confused as to what you're telling the 17 jury. 18 A. No, I did what good scientists do: I 19 based my opinions on evidence. The evidence in 20 1986, I felt, was adequate to say it's a risk 21 factor. Six years later -- especially after we 22 had done what is now regarded as one of the best 23 studies on that issue -- I felt that the evidence 24 was not adequate to support that conclusion.

Page 4312 1 That's my whole point. Scientists base 2 their opinions on scientific evidence. When the 3 evidence changed, my opinion changed because the 4 evidence had changed. 5 Q. And let's talk about what else changed. 6 Then you started testifying for the defendants in 7 asbestos cases, didn't you? 8 A. I was asked to work as an expert on 9 behalf of a number of defendants in the late 10 1980s. 11 Q. You've testified for a lot -- I mean, 12 let's go through kind of the list of defendants 13 you've testified -- you've served as an expert 14 for. You've served as an expert for Ford Motor 15 Company, haven't you? 16 A. I have. 17 Q. General Motors? 18 A. I have. 19 Q. Chrysler? 20 A. I have. 21 Q. Various different asbestos manufacturers. 22 A. Only on the issue of colon cancer. 23 Q. Only on the issue of colon cancer, which 24 is the exact thing you said there was adequate	Page 4314 1 pesticide, looking for effects of neurologic 2 disease, looking at the workers who were quite 3 heavily exposed, and we did not find evidence of 4 neurologic damage. We published four or five 5 articles from that research study. 6 Q. And correct me if I'm -- that's what's 7 commonly known as Dursban. 8 A. Dursban is one of the commercial labels 9 on it. 10 Q. And the EPA has banned that product 11 because of neurotoxicity, haven't they? 12 A. They have restricted it from residential 13 use; it is still widely used in agriculture. 14 Q. So the EPA disagreed with your study 15 funded by Dow on a Dow product, that you know, 16 your study that it does cause neurotoxicity. 17 A. No, EPA didn't disagree with our study. 18 Q. Well, they banned it, didn't they? 19 A. They restricted it so it wouldn't be used 20 in homes. It's still widely used in agriculture. 21 Q. Let's talk about some others. You've 22 testified for manufacturers of welding products, 23 haven't you? 24 A. On the issue of whether welding causes
Page 4313 1 scientific evidence of until you changed your 2 mind. 3 A. Well, until the evidence changed. 4 Q. Okay. You've testified for Dow Chemical 5 Company. 6 A. A long time ago, yes. 7 Q. You don't still have any relationship 8 with Dow? 9 A. I have a large research study -- or I 10 should say the University of Michigan is doing a 11 large research study -- I'm the principal 12 investigator on that study -- and Dow is funding 13 that study. 14 Q. You don't have any other relationships 15 with Dow other than that study? 16 A. No. 17 Q. You've never done any published articles 18 funded by Dow on Dow products where you came out 19 and said, "This product doesn't cause the injury 20 that people were claiming it causes?" You've 21 never done that? 22 A. I did a research study with colleagues 23 looking at the risk of neurologic disease from a 24 pesticide called chlorpyrifos, a widely-used	Page 4315 1 Parkinson's disease, which it does not. 2 Q. I'm actually very familiar with that, 3 which you might be shocked to know, and we may get 4 into that in a little bit. You've testified for 5 BP Petroleum, haven't you? 6 A. I have on occasion, yes. 7 Q. BASF? 8 A. I don't recall. I may have on issues of 9 diisocyanate. 10 Q. I'll show you that one in just a minute. 11 And Baxter Health Care, you remember that one, 12 don't you? 13 A. I remember quite clearly claims that 14 people made -- health care workers, claiming that 15 they had crippling allergies from wearing latex 16 gloves and could no longer work as a result of 17 latex glove. 18 Q. And your opinion was that there's no 19 adequate scientific evidence that latex gloves 20 causes, what, some sort of latex allergy? 21 A. No, that wasn't it. It's actually much 22 more important than that. It's clear that wearing 23 gloves causes contact dermatitis. Lots of people 24 get glove rashes.

Page 4316 1 What the claims were about was the gloves 2 were causing asthma and anaphylactic responses. 3 Anaphylaxis is like when you get a bee sting and 4 you go into cardiovascular collapse and die. And 5 the claims was that gloves were causing those 6 types of reactions, and our work research 7 showed -- and in fact, we published our own 8 analysis of data from the National Center for 9 Health Statistics -- showing that health care 10 workers were not at any increased risk of 11 sensitization to the allergens in natural latex 12 rubber and that there was simply not evidence that 13 health care workers were suffering allergic 14 responses to latex rubber gloves. 15 Q. What -- 16 A. It was clear that they had contact 17 dermatitis. Nobody's arguing with that point. 18 Q. But your essential opinion was "There's 19 no adequate scientific evidence," correct? On 20 whatever the issue was, there's no adequate 21 scientific evidence. That was your opinion. 22 A. Well, the issue, sir, was that claims 23 were being made that the science had proven that 24 wearing gloves -- particularly in the health care	Page 4318 1 pretty predictable, isn't it? I mean, you're 2 going to look at a situation and you're going to 3 say, "No scientific evidence here" and you're 4 going to come into court, and you're going to 5 testify to a jury, "There's no adequate scientific 6 evidence," is there? 7 A. Only when there's no scientific evidence. 8 Q. So you're saying there's no scientific 9 evidence whatsoever that cadmium, arsenic and lead 10 are associated with the health effects being 11 claimed by the members of this class. 12 A. Well, my testimony is exactly what I 13 gave. It's not -- can't be boiled down to that 14 simple a statement. 15 Q. Well -- 16 A. But it's quite clear that there is not 17 evidence that the people who live in this area are 18 at increased risk of these diseases from that 19 smelter. 20 MR. BARR: Hey, Matt, can you pull up the 21 chart real quick? 22 Q. Can you see that all right, Doctor? 23 A. Yes. 24 MR. BARR: Matt, can you pull up the
Page 4317 1 setting -- was causing all these serious diseases 2 and allergic responses, and we looked carefully at 3 the evidence, and there was not evidence to 4 support that claim. 5 Q. And when -- we're probably not going to 6 have time to get into this immediately, but you're 7 -- the most common theme, if we can use a theme, 8 of the testimony you give in trial when you 9 testify for these manufacturing defendants like 10 you've done in this case, is that there's no 11 adequate scientific evidence, that whatever the 12 product is or whatever the chemical is, causes the 13 alleged injury. Isn't that true? 14 A. Only when it's true. 15 Q. Only -- 16 A. Remember I testify on behalf of 17 plaintiffs when it is true. 18 Q. You testified on behalf of a plaintiff 19 once. 20 A. No, no, I've testified on behalf of 21 plaintiffs many, many times. 22 Q. Okay. Well, I think the jury heard your 23 testimony on that. So defendants can rely upon 24 you -- I mean, it's fair to say, your testimony is	Page 4319 1 first one? 2 Q. Now, you remember this case? Duke Power 3 case? This is the first case where you came in on 4 an asbestos situation, and you said, "There's no 5 adequate evidence to show asbestos causes 6 colorectal cancer," isn't that right? 7 A. Of course I do, yes. The evidence had 8 change. You could put up the one before that in 9 1986 where I felt the early evidence supported it, 10 and it changed. 11 Q. But, you've since changed your opinion on 12 this. Your current opinion today is "No adequate 13 scientific evidence," correct? 14 A. That's what the evidence says, yes. I'm 15 not alone in that. 16 Q. All right. That's fine. 17 MR. BARR: Will you go to the next one, 18 Matt? 19 Q. You remember this case, Miles and 20 Tri-Continental Industries about benzene? 21 A. I don't recall the case, but I would 22 certainly agree with the opinion, chronic 23 lymphocytic leukemia and chronic myelogenous 24 leukemia are not known to be caused by benzene.

Page 4320 1 Acute myelogenous leukemia is, but this 2 is an instance where people are putting the wrong 3 diagnosis in. 4 Q. That wasn't the issue in this case, was 5 it, it was chronic myelogenous leukemia -- it was 6 chronic myelogenous leukemia, right? 7 A. CLL and CML are not known to be caused by 8 benzene; AML is. 9 Q. You were paid \$350.00 an hour to provide 10 that opinion with regard to that? Is that about 11 right? 12 A. I don't recall, but that's probably 13 right. 14 MR. BARR: Let's go to the next case, 15 Matt. 16 Q. Remember, this is a case, Dow. This is 17 about TCA. What is TCA? 18 A. Trichloroethane, a commonly used 19 degreasing solvent back in the 1980s and 1990s. 20 Q. Do you remember testifying for Dow in 21 this case and saying that there's no adequate 22 scientific evidence to show that TCA caused 23 neurological injury? 24 A. To cause the neurological injuries that	Page 4322 1 clear Lucite ceiling panels, clear plastic 2 products that are very shock-resistant. It's a 3 very durable plastic. And yeah, it was true, 4 there was no scientific evidence that that 5 material caused multiple chemical sensitivity. 6 Q. Well, I think people's stomachs are 7 rumbling. We'll pick up with this chart as soon 8 as we get back from lunch. Okay? 9 THE COURT: Thank you, sir. Ladies and 10 gentlemen of the jury, hopefully your lunches are 11 here, and you may go with your bailiff. We'll 12 shoot for about 1:00 o'clock to return. 13 (The jury exited the courtroom.) 14 THE COURT: Doctor, you may step down, 15 but again let me ask that you not discuss you 16 testimony with anyone. 17 Counsel, why don't we reconvene at 12:45, 18 please. 19 (A recess was taken for lunch after which 20 the proceedings continued as follows:) 21 THE COURT: Be seated, please. 22 Mr. Bailiff, you may return the jury to its box, 23 sir. 24 (The jury returned to the courtroom.)
Page 4321 1 were claimed. This was a case in 1991, 16 years 2 ago. 3 MR. BARR: Can you go to the next one, 4 Matt? 5 Q. This is the one where you testified for 6 BASF. Do you remember when you testified about 7 BASF about methacrylate? 8 A. Methacrylate. 9 Q. Methacrylate, I'll trust you on how to 10 say it. Do you remember that testimony, and you 11 said there was no adequate scientific evidence to 12 show methacrylate caused multiple chemical 13 sensitivity? 14 A. This is a fascinating issue. Multiple 15 chemical sensitivity is a disease that doesn't 16 exist. It doesn't exist. There was a big 17 brouhaha about how trace levels of chemicals could 18 be causing people to have all sorts of multi- 19 system symptoms, and the scientific community has 20 largely concluded that MCS is not a valid 21 diagnosis, and it does not meet the criteria for 22 saying that that's a real definable disease. 23 And back at the time methacrylate -- 24 methacrylate is the stuff that's in typically	Page 4323 1 THE COURT: You may be seated. 2 Good afternoon, ladies and gentlemen of 3 the jury. Let's note that all 11 of our jurors 4 are again present before the Court and the parties 5 and the respective counsel. 6 Mr. Barr, I believe the questioning is 7 still yours, so you may continue. 8 MR. BARR: Thank you, your Honor. 9 Matt, could you put the chart back up? 10 BY MR. BARR: 11 Q. Now, Doctor Garabrant, we left talking 12 about the testimony you gave for BASF, 13 methacrylate? You remember where we were? 14 A. Yes. 15 Q. This is Baxter Healthcare. You remember 16 this case. We talked about this a little by, this 17 is dealing with latex gloves, right? 18 A. Yes, but that is not what my opinion was. 19 Q. Okay. But your whole opinion, even if it 20 wasn't to show latex gloves caused dermatitis, 21 your opinion was that there was no adequate 22 evidence to show whatever the injury was that was 23 being claimed, wasn't it? 24 A. The injury that was being claimed wasn't

Page 4324 1 caused by the gloves. What else could I say? 2 Q. So you said there was no adequate 3 evidence, correct? 4 A. Well, it wasn't -- there wasn't evidence. 5 It hadn't been caused by it. 6 Q. The same opinion you've given in this 7 case, right? 8 A. No. 9 Q. No, it's not? In this case, you're 10 saying there's adequate evidence that cadmium, 11 arsenic and lead have affected the health of the 12 class members? 13 A. I've made it very clear there's adequate 14 evidence that arsenic causes lung cancer by 15 inhalation; I've made it very clear that arsenic 16 causes lung, bladder, kidney and skin when you 17 have drinking water contaminated. 18 There's no question about that. I've 19 also made it clear that cadmium can cause lung 20 cancer by inhalation. 21 Q. So are you saying that the members of 22 this class are at risk for lung cancer because of 23 their exposure to arsenic? Is that what your 24 testimony is?	Page 4326 1 this was a family that lived next door to a gas 2 station, and I think they claimed that gasoline 3 that had leaked in a plume from the gas station on 4 to their property was causing things like heart 5 disease and lung disease and skin rashes, and 6 that's -- there's just no scientific basis for 7 those claims. 8 Q. Okay. And you were again paid, what, in 9 the neighborhood of \$400.00 an hour for that 10 testimony? 11 A. I don't recall, but that's probably 12 right. 13 MR. BARR: Let's go to the next case. 14 Q. Do you remember testifying on behalf of 15 Cooper Industries on a case dealing with community 16 contamination? 17 A. I do, yes. 18 Q. And again, in that case, you said there 19 was no adequate scientific evidence to show the 20 contamination caused a cancer cluster, didn't it? 21 A. Well, if you're going to put my opinions 22 up, I think we should actually get the opinions, 23 because I don't think that what you're putting up 24 accurately reflects my opinions. I think you're
Page 4325 1 A. No. 2 Q. That's what I thought. And you said 3 there's no adequate evidence, didn't you? 4 A. My testimony is there's -- I have not 5 seen evidence in this case that would establish 6 that they're at increased risk at all. 7 Q. Okay. And in the Baxter Healthcare case, 8 you were paid \$400.00 an hour for your testimony; 9 isn't that right? 10 A. I believe that's correct. 11 Q. Okay. 12 MR. BARR: Let's go to the next slide. 13 Q. You remember testifying for Crown Central 14 Petroleum? It dealt with ground water 15 contamination. 16 A. What was the name of the case? I don't 17 remember the company. 18 Q. Let me pull that out for you real quick. 19 I have Stiley (Phonetic) versus Crown Central 20 Petroleum. You remember that? 21 A. I think so, yes. 22 Q. You remember it dealt with ground water 23 contamination, right? 24 A. Well, if this was the case I remember,	Page 4327 1 changing them a little bit. 2 Q. Well, what was your opinion? Your 3 opinion was that there was adequate scientific 4 evidence? Was that your opinion? 5 A. The Cooper case, to the best of my 6 recollection, was in 1996, 11 years ago. I -- I 7 would have to look at the transcript to see 8 exactly what I said. But I don't think that what 9 you've put up is an accurate reflection of it. 10 Q. Well, would your opinion be that the 11 handful of cancers that were observed in the 12 adjacent community were not caused by the repair 13 facility that was near them? Would that be an 14 accurate reflection of your opinion? 15 A. Could I see what you're reading from? 16 Q. Certainly. 17 MR. BARR: Could I approach, your Honor? 18 THE COURT: Yes, sir. 19 Q. This is your deposition in the Baxter 20 Healthcare cases. 21 A. I thought we were on Cooper. 22 Q. You were talking about Cooper in the case 23 -- in this deposition, you were talking about 24 Cooper basically.

Page 4328 1 A. So you don't have my opinions in the 2 Cooper case. 3 Q. In this deposition, if you'll look at it, 4 Doctor, you'll see you stated your opinion in this 5 Cooper case. 6 A. Well, this is not what you put up. 7 You've misstated my testimony. 8 Q. You said -- what was your opinion in the 9 Cooper case? 10 A. I'll read it. My opinions were that the 11 handful of cancers that were observed in the 12 adjacent community were not caused by the repair 13 facility that was near them. 14 Q. Okay. 15 A. And that was correct. 16 Q. Okay. So that's your testimony. And 17 that accurately reflects your opinion. 18 A. Well, to the extent I recall that in a 19 deposition years later, I believe it did. 20 Q. Okay. And if there had been adequate 21 scientific evidence to show that that cancer 22 cluster was caused by their exposures to 23 contaminants from the repair facility, your 24 opinion would have been different, wouldn't it?	Page 4330 1 Q. So that testimony is not true; is that 2 what you're saying? 3 A. I was asked to recall something I had 4 said years earlier. 5 Q. Okay. 6 A. I testified truthfully to the best of my 7 recollection. 8 Q. Fair enough. 9 A. That I was being asked about something 10 that I had said years earlier. 11 Q. Okay. 12 A. And I will stand by my testimony. Now 13 let's read it. 14 Yeah, let's read this. This is 15 important. 16 Q. Go ahead. 17 A. Okay. "I've next listed Smith versus 18 Arco, a deposition in Houston, Texas. Do you 19 recall the case?" 20 "I don't recall it specifically. I -- to 21 the best of my recollection, it had to do with 22 claims of disease of the hemologic system, in 23 other words, either leukemia or lymphoma or 24 myelodysplasia."
Page 4329 1 A. If the facts were different, my opinion 2 would have been different, of course. 3 Q. Okay. 4 MR. BARR: Let's go to the next case. 5 Q. You remember testifying on behalf of CSX 6 dealing with degreasing solvents? 7 A. And the claim that they were causing 8 irreparable brain damage, yes, I do. 9 Q. Okay. Is that an accurate -- is the "No 10 adequate scientific evidence to show solvents 11 caused central nervous damage," is that an 12 accurate reflection of your opinion? 13 A. If you could show me the opinion, I'd be 14 happy to look. I don't believe that that is. 15 MR. BARR: May I approach, your Honor? 16 THE COURT: Yes, sir. 17 Q. It was Trexlar versus CSX. You were also 18 talking about that in the Baxter deposition. 19 A. So you don't have my opinion in the case. 20 Q. Were you under oath when you were talking 21 about this -- under oath in the Baxter deposition? 22 A. What you've handed me was a deposition 23 given in January of 2000 about cases that occurred 24 years before.	Page 4331 1 Question: "And you were retained by?" 2 Answer: "A defendant in that case." 3 Question: "And the substance of your 4 testimony?" 5 "I don't recall specifically." 6 "All right. You've next listed Trexlar 7 versus CSX in Cumberland, Maryland." 8 Q. That's the case we're talking about on 9 the chart here, right? 10 A. Yes. 11 -- "a deposition. Do you recall that 12 case, sir?" 13 Answer: "I don't recall Trexlar 14 specifically, no." 15 "Do you recall by whom you were 16 retained?" 17 Answer: "I believe I was retained by the 18 law firm Whiteford, Taylor and Preston." 19 "And did they represent a defendant?" 20 "I believe they represented CSX." 21 Q. Okay. 22 A. So now you're asking me if I recall what 23 I said about a case when I testified under oath I 24 didn't recall exactly what I said.

Page 4332 1 Q. Go to the next page. 2 A. Let's keep reading then. "Do you recall 3 the substance of opinions that you expressed in 4 your deposition?" 5 Answer: "There were a couple of similar 6 cases. The issue in those cases was the men who 7 had worked in the CSX repair facility in 8 Cumberland, Maryland believed that the solvents 9 they used to remove grease and oil from the diesel 10 electric locomotive engines had caused them to 11 suffer central nervous system damage." 12 Question: "Did you agree?" 13 Answer: "I did not agree with that." 14 Question: "And what was the substance of 15 your testimony?" 16 Answer: "That the solvent exposures that 17 they had at CSX did not damage their central 18 nervous system." 19 That's not what you put up there. 20 Q. Well, did you believe that the solvents 21 you were exposed to, there's adequate scientific 22 evidence to show that those solvents caused 23 central nerve damage? 24 A. My opinion is exactly what I read, I	Page 4334 1 A. I do not recall -- the case was Rogerio? 2 Q. Yes. 3 A. I do not recall it. When was it? 4 Q. Mr. Rogerio died of multiple myeloma. 5 You remember that? 6 A. I don't. 7 Q. And you didn't feel that that multiple 8 myeloma was associated with his work in the 9 petroleum injury. You don't remember that? 10 A. Well, I don't remember what I said in a 11 case that was many years ago. I don't remember 12 when the case was. I can tell you my opinion 13 today. 14 Q. Okay. 15 A. Multiple myeloma is not caused by 16 petroleum exposures. 17 Q. Based on what? 18 A. Based on the scientific evidence. 19 Q. That there's no adequate scientific 20 evidence, right? 21 A. I gave you my opinion, sir. 22 Q. Let's go to the next case. Do you 23 remember testifying on behalf of Ford Motor 24 Company, GM, Chrysler, NAPA, Abex, certain other
Page 4333 1 believed it did not cause it. You're not putting 2 up what the testimony is. 3 Q. Let's go to the next case. You remember 4 testifying for BP Petroleum -- 5 A. Could you -- 6 Q. -- in this case? 7 A. Could you show me the case and show me my 8 testimony, please? 9 Q. Sure, we can do that. It was Rogerio 10 versus BP America. Do you remember that? 11 A. I don't recall offhand. What year was 12 it, please? 13 Q. You didn't tell us in your sworn 14 testimony. 15 A. Was I asked? 16 Q. It doesn't -- it doesn't reflect it. It 17 says you were retained by BP Petroleum. Do you 18 remember that? 19 A. Well, I don't recall the case. Do you 20 have the testimony from the case? 21 Q. Just answer my question. 22 A. I did. 23 Q. Do you remember being retained by BP 24 Petroleum?	Page 4335 1 people in the asbestos brakes litigation? 2 Certainly you remember those cases. 3 A. I remember them well. 4 Q. And your testimony in that case was that 5 there was no adequate scientific evidence to show 6 asbestos-containing brakes causes mesothelioma. 7 A. That's not my testimony. My testimony is 8 that there's no evidence. 9 Q. No evidence at all. 10 A. No evidence. The epidemiology show 15 11 things that show risks at or below 1.0 for 12 mesothelioma among people that do brake repair 13 work and vehicle repair work, and I'd be happy to 14 go into that in detail with you. 15 Q. So that's your -- your standard testimony 16 that you give? 17 A. I don't have standard testimony. I'm 18 very proud of the work I do. I work very hard to 19 be sure that what I say in court is based on 20 science and it is a truthful and fair 21 representation of the science. 22 Q. I mean, you have to agree that the 23 testimony you give at trial on behalf of these 24 large corporations is pretty much always the same.

Page 4336 1 A. No. 2 Q. Have you ever testified on behalf of one 3 of these defendants that there was adequate 4 scientific evidence to show that the injury 5 alleged was caused by the product or the 6 chemical? Have you ever done that? 7 A. I have testified truthfully on every 8 occasion, and I have testified in cases where the 9 claims were not supported by the science. 10 Q. So the answer is "No." You -- you've 11 never offered that opinion. 12 A. I have testified on behalf of plaintiffs 13 many times when I felt that plaintiffs had been 14 injured by exposures. 15 Q. In the Workers' Compensation setting. 16 A. Yes, they're Workers' Compensation; they 17 were injured at work. 18 Q. Would you agree with me that all this 19 evidence, all this testimony we've been going 20 through is essentially the same testimony you're 21 offering here, that there's no adequate scientific 22 evidence to show that arsenic, cadmium and lead 23 are related to lung cancer, stomach cancer, kidney 24 cancer, cognitive effects in the members of this	Page 4338 1 you, and you changed your mind. Remember that? 2 A. I did some of the original research that 3 was published in the peer-review literature that 4 raised issues about whether asbestos causes colon 5 cancer. 6 Q. And is it your testimony to this jury 7 that the science is not going to change -- even if 8 your opinions are true, that the science is not 9 going to change and it's never going to be shown 10 that arsenic, cadmium and lead can cause the 11 health effects being alleged in front of this 12 jury? 13 A. Let me make it clear, science changes 14 endlessly. 15 Q. Okay. 16 A. That's the whole point. That's why 17 science succeeds so well, because it is based on 18 evidence, and good scientists base their opinions 19 on the evidence, and as the evidence grows and 20 changes, good scientists revise their opinions. 21 So we talked about cadmium was linked to 22 prostate cancer back when I was in public health 23 school, and the evidence changed. Well, it's just 24 not supported by the science.
Page 4337 1 class? 2 A. You know, it's not about my opinions; 3 it's about the science. We've looked at IARC, we 4 looked at EPA, we looked at NTP. This is what the 5 body of scientists around the world largely agrees 6 to. And the fact that I happen to agree with 7 mainstream science, I think says something about 8 my ability to analyze the science honestly and 9 fairly. 10 Q. So did I get you -- just said this is not 11 about your opinions; is that what you just said? 12 A. I said it's about the science, it's not 13 about me. 14 Q. So your opinions don't matter; is that 15 what you're telling this jury? 16 A. I said the science matters. 17 Q. Okay. Now, you understand -- and I kind 18 of want to reference back to your -- your first 19 case that you ever testified in, the asbestos 20 case, where you said there was adequate scientific 21 evidence. You remember that? We talked about 22 that. 23 A. Yes. 24 Q. And then science changed, according to	Page 4339 1 We talked about kidney cancer and 2 cadmium, that the science really doesn't support 3 it. Even though there were some positive studies 4 early on. And so that is the nature of science. 5 It does change. 6 Q. So it's your testimony that this jury 7 should just accept your and DuPont's word for it 8 that the members of this class aren't exposed to 9 significant risks from their exposures to arsenic, 10 cadmium and lead, mind you, a company that's 11 already been found negligent for exposing these 12 people to these substances. That's your 13 testimony, is that they should just trust you? 14 A. No, it's my testimony that the jury 15 should look very carefully and very critically at 16 the evidence, and I hope that they will do that, 17 and I hope that they will come to a thoughtful and 18 fair decision. 19 Q. Now, do you think DuPont looked at your 20 body of testimony before they went out and hired 21 you to come in and testify in this case? 22 A. I have no idea. 23 Q. You have no idea? You don't think that's 24 something they would do?

Page 4340 1 A. I don't work for DuPont; I don't know. 2 Q. Okay. Well, let's talk a little bit 3 about your background and what DuPont may have 4 been able to find out about you before they hired 5 you to come talk to this jury. You want to do 6 that? Okay. I thought -- I'll take that as a 7 yes. 8 A. I don't think my answer is going to 9 matter on that one. 10 Q. You're probably right. Now, wouldn't you 11 agree with me that one of the most significant 12 studies you've done in your career was that dioxin 13 study -- you've already talked about it a little 14 bit -- in Michigan. 15 A. Well, we're still working on that study. 16 Q. Okay, so it's still ongoing. 17 A. I have a team of 15 people working on 18 that. We just gave 28 papers at the dioxin 19 conference, the international conference in Tokyo 20 in early September. 21 Q. Now, is dioxin -- 22 A. Gave 28 papers. I took 15 people to -- 23 14 people to Tokyo. 24 Q. Is dioxin a suspected carcinogen?	Page 4342 1 and house dust? 2 A. I'm not aware that they ever had any 3 opportunity to do that. I'm not aware that they 4 did. 5 Q. You don't think they ever had an 6 opportunity to go out and study the soil and the 7 house dust of the people that lived in the area 8 they contaminated? You don't think they ever had 9 an opportunity to go do that? 10 A. I do not know. I assume that your firm 11 has the opportunity to do that, and that would be 12 compelling evidence if it had been done. 13 Q. And -- and you understand that was done 14 in this case by the members of the class. They 15 hired scientists to go out and study the soil, 16 study the house dust. 17 A. I haven't seen the blood tests. I 18 haven't seen the urine tests. Do they exist? 19 Q. So you think that the members of this 20 class have an obligation to go out and get their 21 blood tested before this company who negligently 22 contaminated their community has to medically 23 monitor them? You think they have an obligation 24 to go out and pay for tests that this company has
Page 4341 1 A. One of the dioxin compounds, 2,3,7,8 2 tetrachlorodibenzo-dioxin, is regarded by IARC as 3 a Class 1 carcinogen to cause cancer in humans, or 4 suspect. 5 Q. And the Dow Chemical Company paid your 6 University 15 million dollars to go and conduct 7 that study, didn't they? 8 A. 11, I believe. Not 15. 9 Q. So 11. So I disagree with you on that, 10 but we'll just -- we'll take 11 for the purposes 11 of this. Would you agree that the reason the 12 study had to occur at all was that there was a Dow 13 plant in the community that was emitting dioxin 14 out into the -- and exposing the people that lived 15 around this plant to dioxins? 16 A. I think it's fair to say that the study 17 was done because of pollution in Midland at 18 Saginaw that came from Dow, yes. 19 Q. Okay. And you went out and you tested 20 blood, soil and house dust. You remember that? 21 A. Yes. 22 Q. Okay. Now, just remind me, do you know 23 if DuPont ever did that for the members of this 24 class? Did they ever go out and test soil, blood	Page 4343 1 caused them to go have to have? 2 A. If you think that they have received 3 doses that have put them in at risk, as a 4 physician, it is absolutely appropriate to do the 5 blood tests and the urine tests. 6 Q. Well, do you -- 7 A. They will provide the foundation for 8 determining whether people were or were not at 9 increased risk. 10 Q. Okay. You understand that's what they're 11 asking for in this case, don't you? 12 A. I believe that Doctor Werntz has asked 13 for some elements of that, and has stated -- and I 14 can quote it from his report where he said, "Doing 15 those tests now wouldn't help." That's wrong. 16 Q. And you think that's wrong. 17 A. I do. 18 Q. But he still -- his program calls for 19 urine testing, does it not? 20 A. Let's read what he says. 21 Q. Doctor, I'm asking a very simple 22 question. His program calls for urine testing, 23 does it not? 24 A. It calls for testing for some things in

Page 4344 1 urine, but not some of the ones that would be most 2 useful. 3 Q. Does it call for blood lead testing? 4 A. It calls for blood lead -- blood lead 5 testing. 6 Q. And that's what you think these people 7 have an obligation to go out and do before this 8 company -- I mean, why should these people -- why 9 should the members of the class have to come out 10 of their own pocket for being exposed to something 11 that they had nothing to do with? 12 This company put those chemicals in their 13 community, not them. Why should they have to go 14 out and pay for that? 15 A. I think that if you're going to claim 16 that somebody's harmed you and you have the 17 opportunity to show whether they've harmed you, 18 it's a reasonable thing to do. A blood lead test 19 is not a very expensive test. 20 Q. So you think they had that obligation, 21 it's on them. That's your testimony. 22 A. I think it's a reasonable foundation, if 23 you want to make a case that somebody's hurt you. 24 Q. Okay. Well, let's go back to the dioxin	Page 4346 1 that settled back on the community. 2 And we found very high levels of dioxins 3 in the soil in that part of the city. 4 There was a second pattern in the river 5 -- there's a river that runs through the plant. 6 It's the Tittabawassee River, and the 7 Tittabawassee River had a different pattern of 8 contamination. 9 It was heavily loaded with 10 polychlorinated dibenzofurans, not heavily loaded 11 with the dioxins, and it's not clear where those 12 furans came from, but they probably came from 13 Dow's operations in the early part of the 20th 14 century, maybe 80, 70, 60 years ago, and the soils 15 in the flood plain of the river were highly 16 variable, but many of them were heavily 17 contaminated with those furans. 18 The household dust -- so we actually 19 vacuum cleaned -- or vacuumed people's homes. The 20 household dust showed elevated levels of dioxins, 21 and they correlated with what was in the soil 22 outside the home. 23 We tested people's blood. The blood 24 levels showed virtually no relationship to what
Page 4345 1 thing. Let's finish your study with Dow. You 2 found in your study high levels of dioxin in 3 homes, soil and people, didn't you? Wasn't that 4 one of the results of your study? 5 A. You'd have to bring up the results. You 6 could go right to the web site and find it. 7 Everything we had found is on our web site. 8 Q. You don't remember that? Weren't you the 9 principal investigator? 10 A. I remember in exquisite detail what we 11 found. Do you want me to go through it? 12 Q. I just asked you a very simple question. 13 You found high levels of dioxins in the homes, the 14 soil and people's blood, didn't you? 15 A. Okay. 16 Q. I mean, that's "yes" or "no." You either 17 did or you didn't. 18 A. Well, a "yes" or "no" answer wouldn't 19 characterize it accurately. Let me answer, 20 please. Okay. We found that there was extensive 21 soil contamination in Midland and Saginaw, and it 22 occurred in two patterns. One pattern was to the 23 north and northeast of the plant, and likely 24 represented aerosol deposition from air emissions	Page 4347 1 was in the soil or what was in the household dust. 2 So even though the soil was contaminated and the 3 dust was contaminated, there did not seem to be 4 any direct pathway from the contaminated soil and 5 contaminated dust into people's blood. 6 I can go on -- 7 Q. I mean -- 8 A. -- for as many hours as you'd like to 9 hear. 10 Q. If you want to waste the jury's time, 11 that's up to you. 12 A. I don't, sir. 13 Q. I asked you a very simple "yes" or "no" 14 question, and the answer is "yes." You found high 15 levels in the soil, you found high levels in the 16 dust. 17 A. But not in the blood. 18 Q. Fine. Now, in your study, did you do any 19 research on the potential health effects of dioxin 20 and the high levels in the soil? Was that part of 21 your study? 22 A. No. And this is the critical issue. 23 Because before you're going to study health 24 effects, you have to establish that exposure has

Page 4348 1 led to an increased dose. In other words, it 2 doesn't make sense to go out and do a health 3 effects study until you've established that the 4 chemical in the environment is actually getting 5 into the people's bodies. It doesn't make sense 6 to do it. It's nonsense. 7 And so our study was designed to see 8 whether the chemicals in the environment got into 9 people's bodies, and by what pathways they got in. 10 Because that's the critical issue. 11 If you want to do something about the 12 exposure, you have to figure out how it gets from 13 the environment into people's bodies. And then 14 you can take effective action to stop the 15 exposure. 16 What you don't do is say, "Oh, we'll go 17 out and study health effects" before you can 18 establish that something in the environment even 19 gets into people's bodies. Because it's nonsense. 20 You've got to go step by step, and you 21 have to establish the first thing first, and then 22 if you find that the body burdens are increased 23 and you've figured out which pathways, then it 24 makes sense to think about health studies.	Page 4350 1 saying, there has been no testing in this class on 2 blood. He asked him. 3 THE COURT: But the witness was 4 instructed by the Court, through counsel, not to 5 answer that, so I'm going to instruct the jury 6 that they're to disregard it, that the witness 7 answered inappropriately, contrary to the Court's 8 ruling, and they're not to consider. 9 MR. GALLAGHER: I think it was invited, 10 but if that's the Court's ruling -- 11 (Counsel returned to counsel table and 12 the proceedings continued in the presence and 13 hearing of the jury as follows:) 14 THE COURT: Ladies and gentlemen of the 15 jury, there's been an appropriate objection as to 16 the last answer of the witness which violated a 17 prior Court order, so the Court is striking that 18 answer from the record and instructing the jury 19 not to consider it in any respect in arriving at 20 their decision. 21 So you may continue, sir. 22 BY MR. BARR: 23 Q. Going back to the dioxin study, would it 24 be fair to say that you went so far as
Page 4349 1 And of course that's what we did. 2 Q. In this case that we're presently here 3 for today, did DuPont do any of these tests that 4 you just talked about, any? One? Just one? 5 A. I'm not aware that DuPont or anyone else 6 has done tests except for the ATSDR who came in 7 and tested children and found nothing. 8 Q. You're not aware that plaintiffs -- that 9 the scientists that worked for the members of this 10 class went out, tested the soil, house dust and 11 air in the class area. You're not aware of that. 12 A. I am aware of the data that underlies 13 Doctor Brown's risk assessment model, and I'm 14 aware that there's been one blood test of a 15 plaintiff in the class. 16 MR. BARR: Your Honor, can we approach 17 real quick? 18 THE COURT: Yes, please. 19 (Counsel approached the bench and the 20 following proceedings were had out of the hearing 21 of the jury:) 22 MR. BARR: He was specifically instructed 23 not to get into that blood test, and he's -- 24 MR. GALLAGHER: He challenged him by	Page 4351 1 discouraging people that lived around the Dow 2 plant from going to other doctors and getting 3 their blood tested because you thought the Dow 4 study would be conclusive? 5 A. No. 6 Q. You did not do that. 7 A. I did not. 8 Q. Now Doctor, it's fairly common for you to 9 do corporate sponsored research, isn't it? 10 A. Well, the sponsors of my research include 11 the National Cancer Institute, the National 12 Institutes of Health, the National Institute for 13 Environmental Health Sciences, NIOSH, the State of 14 California, the American Cancer Society, the 15 United Auto Workers, Ford Motor Company National 16 Joint Committee, the Dow Chemical Company, the 17 Rohm and Haas Chemical Company, private 18 foundations. That's a few. 19 Q. I want to show you a few of the studies 20 you've done, just a couple. Let's start with -- 21 MR. BARR: Matt, can you pull up -- I 22 don't know that you have this. I'll just use the 23 Elmo. 24 Can I approach, your Honor?

Page 4352 1 THE COURT: Yes, sir. 2 Q. Do you recognize this study, Doctor 3 Garabrant? 4 A. I do. 5 Q. This is a study that you conducted, is it 6 not? See your name there? 7 A. I was a co-investigator on this, yes. 8 Q. And this was about scleroderma and 9 solvent exposure among women. You remember that? 10 A. I do. 11 Q. Do you know who this was -- who paid for 12 this study? 13 A. Yes, the Dow Corning Corporation. 14 Q. So that's just one study you've done that 15 was on behalf of Dow Corning, right? 16 A. That was a study done on behalf of the 17 Dow Corning -- actually, you know what? That's 18 not true. It says right in the study, "Supported 19 by grants from the Halogenated Solvents Industry 20 Alliance, the Dow Corning Corporation and the 21 National Institutes of Health." 22 Q. So this is one example of studies you've 23 done on behalf of the Dow Chemical Corporation, 24 similar to your -- not in the same realm, but	Page 4354 1 A. I sure do. 2 Q. This is the study we talked about 3 earlier? 4 A. Yes, we did a study of chlorpyrifos, 5 which is Dursban, and we did this with funding 6 from the Dow Chemical Company which is stated on 7 page 366. 8 Q. There's your name, right? You see that 9 right on the front. 10 A. Yes. 11 Q. And this is "The Effects of Occupational 12 Exposure to" -- how do you say that? 13 A. Chlorpyrifos. 14 Q. And that's the same as Dursban, isn't it? 15 A. Chlorpyrifos is the chemical name; 16 Dursban is the market name. 17 Q. And what was your conclusion in this 18 study? 19 A. It's written right on the first page. 20 Q. Where's it at? I see it. It was your 21 conclusion that -- here, I'll read it. Was your 22 conclusion that "Chronic chlorpyrifos exposure 23 produced no clinical evidence of cortical, 24 pyramidal tract, extrapyramidal, or other CNS
Page 4353 1 similar to the dioxin study, where Dow went out 2 and paid you or -- you know, your University, to 3 go out and conduct a study, didn't it? 4 A. No, you've mixed up two companies. This 5 is the Dow Corning Corporation; that is not the 6 Dow Chemical Company. It's a different company. 7 Q. So they're not related at all? 8 A. I'm not sure of the relationship. To the 9 extent I know, I believe Dow Corning was formed 10 during or just after World War II as a joint 11 interest between Dow Chemical and the Corning 12 Glass Company to explore new findings in the 13 silica (Phonetic) chemistry which had evolved 14 during World War II. 15 And I'm not an expert in corporate 16 finance, but I'm under the impression that this 17 was created as a separate corporate entity. 18 Q. Okay. The next study I want to show that 19 you've done, this was the study on Dursban that we 20 previously talked about. 21 MR. BARR: May I approach, your Honor? 22 THE COURT: Yes. 23 Q. Do you recognize this study, Doctor 24 Garabrant?	Page 4355 1 dysfunction among chlorpyrifos subjects compared 2 with referents, either at baseline or after one 3 year of additional exposure." 4 A. That's exactly what we wrote, yes. 5 Q. And this is a study you did for the Dow 6 Chemical Company, correct? 7 A. That's correct. 8 Q. And in this study, you even listed -- you 9 see down there at the acknowledgements? It says, 10 "Some of the authors have at times been retained 11 as consultants or served as expert witnesses in 12 litigation for firms or companies including Dow 13 Chemical, Agrosiences, concerned the manufacture 14 or use of insecticides." Do you see that? 15 A. Yes. That's absolutely appropriate to 16 put in, a full disclosure. 17 Q. So you published a study having to do 18 with Dursban, saying that there are no central 19 nervous system effects, and that study was paid 20 for by Dow Chemical, right? 21 A. We studied Dow employees who made Dursban 22 and who were heavily exposed, and the study was 23 funded by Dow, and they gave us access to their 24 employees. The study was done by the University

Page 4356 1 of Michigan with all of the appropriate 2 protections for human subjects in research and 3 protections of confidentiality as well, and I'm 4 very proud of the work. Yes, it's good science. 5 MR. BARR: May I approach, your Honor? 6 THE COURT: Yes, sir. 7 Q. Do you recognize this, Doctor Garabrant? 8 A. I don't know that I've seen this exact 9 document in the past, but I'm certainly aware of 10 the EPA's actions to restrict use of Dursban in 11 homes and consumer use in gardens. 12 Q. And they did that despite the fact that 13 you published a study that said there are no 14 central nervous system effects, right? 15 A. Well, I think you've got your dates out 16 of order. 17 Q. Your study actually came after this. 18 A. Right. Our study came out in 2004, so 19 it's hard to say that the EPA knew the results of 20 our study before we'd done it. 21 Q. Well, did the EPA -- have they since 22 lifted the ban? 23 A. I believe they have not. 24 Q. Now, Doctor Garabrant -- give me one	Page 4358 1 Mickey Leland Center, which is correctly called 2 the National Urban Air Toxics Research Center, 3 gets its money from the EPA, and they also get 4 donations from industries that are interested in 5 and concerned about urban air pollution. 6 MR. BARR: May I approach, your Honor? 7 THE COURT: Yes, sir. 8 Q. Not -- let me put this here so the jury 9 can see it. You see that this is about the 10 center, the Mickey Leland National Urban Air 11 Toxics Research Center. Do you see that? 12 A. Yes. Could you move it up a little bit? 13 Q. Is that better? Let me try and zoom out, 14 see if that helps. 15 A. That's too far. Let's go back in. 16 Q. You tell me where you want me to stop. 17 A. About right there -- whoa, that's good. 18 Q. Now, if you look -- obviously you're 19 going to want to point me to the part where this 20 is authorized by the U.S. Congress. 21 A. And funded by the Congress, too. 22 Q. Well, let's go to the third page -- well, 23 let's start with the second page. You see there 24 Under Current Scientific Advisory Panel Members?
Page 4357 1 second here to get organized. You also work with 2 research centers that are heavily funded by 3 corporations, don't you? 4 A. I don't know what you're referring to. 5 I'd have to say not to my knowledge, no. 6 Q. You don't -- you don't serve as an 7 advisor to the Mickey Leland National Air Toxic 8 Research Center? 9 A. The Mickey Leland Center is a 10 Congressionally-mandated center to study air 11 pollution and it is funded by the EPA. It has a 12 line item in the EPA budget. It's named after 13 representative Mickey Leland who was a strong 14 advocate for control of urban air pollution and 15 who was killed in that airline flight -- oh, I 16 want to say -- back in Serbia during the Clinton 17 administration. 18 The Center was named after him because of 19 his strong advocacy on behalf of cleaning up urban 20 air. 21 Q. And are you telling this jury that there 22 aren't corporate sponsors to the Mickey Leland 23 National Urban Air Toxic Research Center? 24 A. No, I'm not telling them that. The	Page 4359 1 A. I do, yes. 2 Q. It lists your name, doesn't it, David H. 3 Garabrant. That's you, isn't it? 4 A. Yes. 5 Q. And then if we go to the third page, it 6 says as the -- that's a short form for the center? 7 A. The National Urban Air Toxics Research 8 Center, that's correct. 9 Q. "Moves towards implementing its mission 10 and achieving its important goals, substantial 11 additional public and private support will be 12 needed." Do you see that? 13 A. Yes, I do. Let's look up at the first 14 paragraph at its mission. 15 Q. We'll get there -- I'll go where you want 16 to go once I get through with this. Okay? Is 17 that fair? 18 A. Yeah, sure. 19 Q. And it says, "More industrial firms, as 20 well as foundations and other sources, will be 21 asked to take part in the funding of the Center's 22 complex research." Do you see that? 23 A. Yeah, I think this is great, get 24 industries to pay for air pollution research.

Page 4360 1 Q. It says, "Past and present corporate and 2 other sponsors are: Ashland Chemical." Do you 3 see that? 4 A. I see it, yes. 5 Q. You see DuPont down there? 6 A. I do. 7 Q. Okay. You see companies like Goodyear, 8 Georgia-Pacific, Exxon Chemical, Sunoco, Union 9 Carbide -- 10 A. Yeah. 11 Q. So this study -- this research center is 12 heavily funded by industry, isn't it? 13 A. Well, it's heavily funded by the EPA, and 14 they're asking companies to assist in paying for 15 air pollution research. I think that's -- that's 16 a worthy mission. 17 Q. I'm going to move on a little bit, 18 Doctor. Would you agree with me that -- I'm not 19 the first person to criticize you for your ties to 20 the industries, am I -- to ties to corporate 21 industry, am I? 22 A. I've been criticized for doing work for 23 the UAW too. I mean, I work in a realm that is 24 highly contentious. When you start to research	Page 4362 1 years ago. I didn't memorize it. But if we could 2 look at it, I would be pleased. 3 Q. We'll -- I'll show it to you in a minute 4 here. 5 A. Okay. 6 Q. Would you agree with me that the Dow 7 Chemical Company is the number one producer of 8 ethylene oxide? 9 A. I have no idea. 10 Q. You have no idea. 11 A. -- what chemical company is the number 12 one producer of ethylene oxide. 13 Q. Well, let's show this to you. 14 MR. BARR: May I approach, your Honor? 15 THE COURT: Yes, sir. 16 Q. You recognize this, Doctor Garabrant? 17 A. Yes. 18 Q. Let's read what this says. This is June 19 16, 2006, right? You see that on the front? 20 A. I do. 21 Q. And it says, "Re," regarding, "Invitation 22 for comments on the short list candidates for the 23 Ethylene Oxide Review Panel, EPA Science Advisory 24 Board." Do you see that?
Page 4361 1 the health effects of chemicals, it's highly 2 contentious, and people criticize whatever you do. 3 Yes, I've been criticized. 4 I've also been the recipient of research 5 awards for outstanding research too. 6 Q. Okay. You remember being nominated to 7 the EPA Science Advisory Board on ethylene oxide? 8 A. Yes, I do. 9 Q. And you remember a group of public health 10 advocates objecting to you being on the panel? 11 A. I'm not sure that would characterize 12 correctly who they were, but -- 13 Q. You don't think that would -- okay, well, 14 I'll show you that in a second. But they were 15 concerned that you were a corporate insider, 16 weren't they? 17 A. Why don't we read exactly what they said? 18 Q. So you don't remember if they were 19 concerned that you had financial conflicts that 20 would prevent you from being an unbiased member of 21 the science advisory panel on ethylene oxide? You 22 don't remember that? I mean, seems like to me 23 that that's something that people would remember. 24 A. I saw their letter approximately two	Page 4363 1 A. Yes. 2 Q. And it says, "This letter is supported by 3 the following worker and public health 4 advocates." Do you see that? 5 A. I see that's what it says, yes. 6 Q. And it's supported by Jennifer Sass with 7 the Natural Resources Defense Council. You see 8 that? 9 A. Yes. 10 Q. By an Amanda Hawes from the Toxics Chair 11 WORKSAFE. You see that? 12 A. Yes, and my experience is Ms. Hawes -- 13 she's an attorney in the Bay area. 14 Q. Okay. You see that it's supported by 15 Kathleen Burns, the director of Science Core. You 16 see that? 17 A. I can read it very well. I have no idea 18 who some of them people are, to be honest. 19 Q. Well, they knew who you were, didn't 20 they? 21 A. I'm sorry? 22 Q. They knew who you were. 23 A. I have no idea -- 24 Q. Well, we'll get --

Page 4364 1 A. -- what they knew about me. 2 Q. We'll get to that in a minute. It's 3 sponsored by a Bill Borwegen, Occupational Health 4 and Safety Director, Service Employees 5 International Union. You see that? 6 A. Yes. 7 Q. By Michael Jacobson, Center for Science 8 in the Public Interest? 9 A. Yes. 10 Q. By Mikal Harbut, Chief Center for 11 Occupational and Environmental Medicine at Wayne 12 State University. You see that? 13 A. I see that. Although what does "For ID 14 only" mean? I don't know what that applies to. 15 Q. Well, he certainly signed his name, 16 didn't he? He allowed them to use his name on 17 there, didn't he? 18 A. I don't know. 19 Q. You see it says, "Jane Houlihan," there's 20 a Joe DiGangi, Environmental Health Fund, David 21 Michaels, Department of Environmental Health at 22 George Washington University, David LeGrande? You 23 see that -- see all the people listed here? 24 A. I see those names, yes.	Page 4366 1 done research under contract between the 2 University of Michigan and Dow. I need to make it 3 clear to the jury, I don't get any money from 4 doing that. I'm a tenured professor. My salary 5 is paid no matter what I do. 6 If I bring in a research grant, that 7 doesn't change my salary, I don't get anything. 8 So it doesn't matter to me if it comes from the 9 National Cancer Institute, United Auto Workers, 10 Ford Motor Company National Committee or the Dow 11 Chemical Company, if it's a good research project, 12 I will pursue it, but I get no financial reward 13 for doing that. 14 Q. Well, the only financial reward you get, 15 as the jury's already seen, is it comprises 50 16 percent of your total income. 17 A. No, no -- 18 Q. Testifying for people like Dow Chemical. 19 A. Unh-unh, unh-unh, you just mixed up two 20 separate things. The research money that comes to 21 the University of Michigan does not come to me, 22 not a penny of it. 23 Q. Okay. 24 A. Okay?
Page 4365 1 Q. Okay. Now, let's go to the second page. 2 It says, "Overview of ethylene oxide toxicity: 3 Ethylene oxide is a known carcinogen." Do you see 4 that? 5 A. I do. 6 Q. Now, let's go here when they start 7 talking about their comments. "Five of the 8 nominees" -- I'm on the third page, if you can 9 follow. "Five of the nominees have financial 10 conflicts: Gargas, Garabrant." You see that? 11 A. I see it. 12 Q. Then it lists four more nominees, and it 13 says, "These nine individuals should not be 14 selected for expert committee since several 15 represent a coordinated perspective, several have 16 overlapping expertise, and since at least five, to 17 our knowledge have a direct financial relationship 18 with the ethylene oxide and petrochemical 19 industry." 20 Do you see that? 21 A. I see it. 22 Q. You had such a tie with Dow Chemical 23 Company, didn't you? 24 A. Well, we've already discussed that I have	Page 4367 1 Q. Well, let's go to page 5 of this, and 2 let's see what they wrote about your financial 3 conflicts. You see that where it says "Garabrant 4 and Schnatter" -- is that how you say that, do you 5 know? 6 A. I do not know Doctor Schnatter. I have 7 seen his name in the peer-review literature. I've 8 never met him. It says, "Garabrant and Schnatter 9 have financial conflicts. David Garabrant has a 10 history of industry consulting and has served as 11 an expert witness to corporate defendants. 12 Between 2001-2005, Garabrant was 13 personally paid over \$264,800 for his consulting 14 for Ford and General Motors in asbestos 15 litigation. He is now conducting a 15 million 16 dollars stud on dioxin hazards funded by Dow 17 Chemical. Since Dow Chemical is the world's 18 largest producer of ethylene oxide, this 19 constitutes a direct financial conflict." See 20 that? 21 A. I see it. 22 Q. And then it goes on to say, "Garabrant's 23 financial relationship is not disclosed on the EPA 24 web site, an apparent violation of the law." Do

Page 4368 1 you see that? 2 A. That is -- first off, I have no knowledge 3 of how much or whether the Dow Chemical Company 4 produces ethylene oxide. I was surprised to read 5 that. And the fact that I didn't disclose it, I 6 don't know how I could have disclosed something I 7 didn't know. 8 Q. You didn't know that you had a financial 9 relationship? 10 A. Well, I don't receive money from the Dow 11 Chemical Company. I didn't know I had a financial 12 relationship with them. 13 Q. And this group thought that your failure 14 to disclose that was a violation of the law. 15 A. Since I don't get money from them, I 16 didn't know I had a financial relationship. 17 Q. Now, ultimately, Doctor, you weren't 18 appointed to that advisory board, were you? 19 A. I was not. 20 Q. Now, I want to move now into your 21 opinions regarding this case and the class area 22 and what your opinions are, okay? Now, you hold 23 the opinion, if I'm correct, that there's been no 24 significant exposure to the members of the class	Page 4370 1 want to show me? 2 Q. I'm just asking you if that's something 3 you knew before you came in here and told this 4 jury that the members of this class have not been 5 significantly exposed to cadmium and arsenic and 6 lead. Did you know that? 7 A. I -- I've already answered the question. 8 I'm not aware of that number, but -- what -- what 9 document are we referring to? Could I see it? 10 Q. I'm not going to waste this jury's time 11 with it. They've seen this stuff a lot. The 12 important point is, there's a lot of stuff you 13 don't know, isn't there? 14 A. Well, sir, if you're going to ask me 15 questions about documents I haven't seen, I think 16 it would be fair to let me see them. 17 Q. Well, I'm going to show you some 18 documents here in a little bit. When did you 19 actually arrive in town to testify in this trial? 20 A. Early in the morning of Tuesday morning, 21 in the wee hours. 22 Q. Okay. And it's now Wednesday, right? 23 A. Yes. 24 Q. Okay. So you've been here today. Is
Page 4369 1 area from the DuPont plant. Is that your opinion? 2 A. I hold the opinion that Doctor Brown's 3 models show relatively low level mild exposures -- 4 Q. Now -- 5 A. -- the opinion that there is no evidence 6 of internal dose to the plaintiffs. 7 Q. Other than Doctor Brown's data, which the 8 members of the class paid for, not DuPont -- other 9 than his data, what do you know about the 10 exposures of these members of the class to 11 cadmium, arsenic and lead? You don't know 12 anything, do you? 13 A. I know they live in a community in which 14 there is a former smelter, and I know that studies 15 of similar communities around the world have not 16 demonstrated excess risks of cancer. 17 Q. Okay. Do you -- and the jury's heard 18 this, so I don't want to go through all the 19 documents on this, but you apparently don't know 20 this -- are you aware that this smelter released 21 5600 pounds of dust a day? Is that something you 22 knew? 23 A. I do not recall those numbers. I don't 24 know the source of that. Is there a document you	Page 4371 1 this the first time you've ever come to 2 Clarksburg? 3 A. To the best of my recollection, yes. 4 Q. So you've never gone out and looked at 5 the facility and the class area, have you? 6 A. I'd actually very much like to do that. 7 Q. Well, you're saying you haven't had the 8 chance or the opportunity to go do that? 9 A. I have not had the opportunity to do 10 that. 11 Q. How long have you -- 12 A. I'll do that, actually, when we finish 13 today. I'd very much like to see it. 14 Q. Oh, after you testify and tell this jury 15 that there's no significant exposure, then you'll 16 go look? 17 A. I'd still like to see the site. 18 Q. Well, how long have you been retained as 19 an expert in this case? 20 A. I'm not sure I recall. I think perhaps 21 nine months, could be a year. I'm not sure. 22 Q. And in that nine months to a year, you've 23 never had the opportunity to come to Clarksburg 24 and talk to the members of this class about their

Page 4372 1 exposures to cadmium and arsenic and lead from the 2 smelter. 3 A. I don't know how I could have spoken with 4 them. I don't know that I'm allowed to talk with 5 plaintiffs in litigation when I've been retained 6 by the other side. Am I allowed to do that? 7 MR. BARR: Your Honor, I don't have any 8 further questions for Doctor Garabrant. 9 THE COURT: Any follow-up, counsel? 10 MR. GALLAGHER: Yes, your Honor. 11 THE COURT: And if you'll share the 12 microphone, please. 13 MR. GALLAGHER: Your Honor, there is one 14 thing that I need to approach about. 15 THE COURT: Please. 16 (Counsel approached the bench and the 17 following proceedings were had out of the hearing 18 of the jury:) 19 MR. GALLAGHER: Your Honor, we're back to 20 the topic of Lenora Perrine's blood. He asked 21 him, "What do you know about exposures in the 22 class area to arsenic, cadmium and lead? You 23 don't know anything, do you?" 24 He -- he impeached him with his lack of	Page 4374 1 MR. THOMAS: Thank you, your Honor. 2 DuPont calls Kelly Nelson. 3 THE COURT: If you'll come forward, sir, 4 please. Could counsel approach, please? 5 (Counsel approached the bench and the 6 following proceedings were had out of the hearing 7 of the jury:) 8 THE COURT: I'm just asking -- it's my 9 general understanding -- you correct me -- that 10 Doctor Nelson's being called as a fact witness. 11 Is that correct? 12 MR. THOMAS: No, that's not correct. 13 He's going to be called as an expert in primary 14 care, treatment of patients in the area. 15 THE COURT: Okay. I just kind of 16 remember our prior conversations, so -- okay, 17 that's fine. Thank you. 18 (Counsel returned to counsel table and 19 the proceedings continued in the presence and 20 hearing of the jury as follows:) 21 THE COURT: Sir, if you'll raise your 22 right hand. 23 (The witness was sworn.) 24 THE COURT: You may have a seat up in the
Page 4373 1 knowledge, when he has knowledge. You can't do 2 that. He's -- you can't move in limine to keep 3 something out and then ask a question that is 4 designed to impeach somebody when they can't 5 answer it. He's opened the door to that, and I 6 should be allowed to -- 7 THE COURT: But didn't you ask the exact 8 same question? "There isn't any evidence that 9 you've seen other than the ATSDR study concerning 10 this," and I think it's the same question. So the 11 ruling of the Court remains the same. 12 So you may continue. 13 (Counsel returned to counsel table and 14 the proceedings continued in the presence and 15 hearing of the jury as follows:) 16 MR. GALLAGHER: Your Honor, there's 17 nothing I need to ask. 18 THE COURT: May this witness be excused? 19 MR. BARR: Yes, your Honor. 20 THE COURT: Sir, you are excused. Thank 21 you for your time. 22 THE WITNESS: Thank you, your Honor. 23 THE COURT: Mr. Thomas, you may call your 24 next witness.	Page 4375 1 witness stand, please, sir. 2 K E L L Y N E L S O N 3 was called as a witness by the Defendant, and 4 having been first duly sworn, testified as 5 follows: 6 DIRECT EXAMINATION 7 BY MR. THOMAS: 8 Q. State your name, please. 9 A. Kelly R. Nelson. 10 Q. And it's Doctor Nelson, isn't it? 11 A. That's correct, yes, sir. 12 Q. Doctor, where do you live? 13 A. 46 Juniper Lane, Bridgeport, West 14 Virginia, right here in Harrison County. 15 Q. And where do you work? 16 A. A place called Medbrook. 17 Q. Would you tell the jury what Medbrook is? 18 A. Medbrook is a large group practice 19 located in Bridgeport, 1370 Johnson Avenue. We're 20 a group of family physicians. We do primary care, 21 continuity care, do some occupational medicine. 22 We also have a large urgent care practice. 23 Q. And what's your role currently at 24 Medbrook?