IN THE GENERAL COURT OF JUSTICE
SUPERIOR COURT DIVISION
STATE OF NORTH CAROLINA, COUNTY OF FORSYTH
Civil Action No. 07-CVS-3770

VIDEOTAPE DEPOSITION OF: DAVID W. PYATT, Ph.D.
September 23, 2009

ANDREW WOLFE and DENISE WOLFE,

Plaintiffs,

v.

E.I. DUPONT DE NEMOURS AND COMPANY, et al.,

Defendants.

PURSUANT TO NOTICE the videotape deposition of DAVID W. PYATT, Ph.D., was taken on behalf of the Defendants at the St. Julien Hotel, 900 Walnut Street, Boulder, Colorado 80302, on September 23, 2009, at 9:37 a.m., before Teresa Coogle, Registered Professional Reporter, Certified Realtime Reporter, and Notary Public within Colorado.

A P P E A R A N C E S

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Also present:	Stacey Manela Jerry DeBoer, Videographer

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EXHIBIT DISPOSITION:
Original Exhibits: With transcript

THE VIDEOGRAPHER: We are on the record at 9:37 on September 23, 2009, at 900 Walnut Street, Boulder, Colorado. We are here for the videotape deposition of David W. Pyatt, Ph.D., in the matter of Andrew Wolfe and Denise Wolfe versus E.I. Du Pont de Nemours and Company, et al., in the General Court of Justice, Superior Court Division, State of North Carolina, County of Forsythe, Case No. 07-CVS-3770.

The videographer is Jerry DeBoer. The court reporter is the Teresa Coogle from Hunter + Geist, Inc. Will counsel please state their appearances beginning with the plaintiffs' counsel.

MR. GIANARIS: Ted Gianaris, Simmons firm, by way of pro hoc vice.

MR. SCHIRRMEISTER: Andrew Schirrmeister for DuPont.

MR. MILLER: James R. Miller for PPG Industries.

DAVID W. PYATT, Ph.D.,

having been first duly sworn to state the whole truth, testified as follows:

DIRECT EXAMINATION

BY MR. SCHIRRMEISTER:

Q. Would you into yourself -- introduce yourself to the court and to the ladies and gentlemen

of the jury?

A. Sure. My name is David Pyatt.

Q. Okay. And, David, you're appearing here by videotape rather than in person. Would you explain to the court and the jury why you're appearing by videotape.

A. I'm appearing by videotape because I will not be in the country when this goes to trial in October.

Q. Okay. What are you going to be doing?

A. I'm going to be -- I'm going to be on a climbing trip in Nepal.

Q. And how is it that you came to be on a climbing trip in Nepal during this period?

A. Well, my -- I've been trying to get to Nepal and climb this peak for three or four years. And finally, we got the permit. And I got the people together that I felt comfortable enough doing this climb with. And that all happened before this case got started. So that's why that took priority over -- over what we're doing here.

Q. Okay. Where are you originally from?

A. I was born in Pensacola, Florida, but I spent most of my childhood and early adulthood in North Carolina.

Q. Okay. Tell us where you grew up, where you went to high school, where you worked at -- up through your high school years?

A. I -- most of the time, we spent -- I lived in Raleigh, North Carolina, for a little while, and then we moved to the mountains. So I grew up and went to high school in a place called Marion, North Carolina. It's about an hour and a half further west from Winston Salem. That's where I went to school. And then when I -- after high school, which was McDowell High School, I moved back to Raleigh and went to North Carolina State University for my undergraduate. Both my parents still live in Western North Carolina. My mom is a retired nurse and she lives in Asheville, and my dad is a retired construction developer, and he lives in -- still lives in Marion.

Q. Okay. What year did you graduate from high school?

A. 1976.

Q. Okay. And tell the jury a little about yourself personally? Are you married? Do you have a family?

A. I am married. I've been married for 22 years. And I have two daughters. One is a junior in

college, and she's here at Boulder at CU. And the youngest one is a junior in high school, and she's, obviously, still living at home with us.

Q. Okay. I'm going to put up --

A. Go ahead. I can move.

Q. -- the first slide. So tell us about where you went to college and what you graduate --

A. I'm going to have to slide over. Is this okay with every one? This way. Just so that's not -- okay. Everybody all right with that?

Q. Yes, sir.

A. Okay.

Q. So just tell us where you went to college and what you studied there.

A. Thanks. My undergraduate at North Carolina State University was biological sciences. I think that was the name of the college. I also took a lot of chemistry classes, so I probably could have gotten a double major; but anyway, biological sciences, agricultural sciences, I think was the name of the school. And then I went back and got an additional B.S. degree in science education, because I wanted to -- I wanted to teach. And then my graduate degree was at the University of Colorado. And that was in toxicology.

Q. Okay. Explain to the jury what toxicology is, David.

A. Well, the -- the word means the study of poisons. So you are looking at why things are toxic, amounts that it takes to be toxic, potential remedies for that toxicity. So that's what toxicologists do. We study chemicals and other things that have the potential to be toxic and try to understand why that happens.

Q. Okay. And presently, do you have faculty appointments and teach students at the University of Colorado?

A. I do.

Q. Okay. And which -- which faculty appointments do you have?

A. I'm still with the same group that I got my Ph.D. with, and that's the University of Colorado, School Of Pharmacy, where I teach a toxicology course, I teach a risk assessment course. I have an appointment with the newly formed school of public health where I also help them teach their toxicology course. And I'm developing a risk assessment course for them.

I teach a toxicology course for the University of Colorado, the Denver campus, and also

teach an environmental epidemiology class for those. So I guess technically I have three different appointments, but it's all part of the University of Colorado.

Q. Okay. Now, you have a Ph.D., correct?

A. I do.

Q. And so it would be appropriate to address you as Dr. Pyatt?

A. I -- I think in a formal setting, yes.

Q. But I'm -- if it's all right with you, I'll just call you David.

A. That's perfectly fine.

Q. Okay. Are you currently or since your -- receiving your Ph.D., what type of work have you done and what research have you conducted?

A. When I first graduated with my Ph.D., I did what is typically called a post-doctoral fellowship. So this is when you -- you work with someone. They are the ones that got the grant, so they are paying you a salary out of their grants. And you're doing research that they have an interest in in their lab. And I did that with Dr. Richard Irons, and we were looking at benzene metabolites and the toxicity associated with the benzene metabolites in bone marrow cells in the immune system. And then

typically the way an academic profession goes is you start -- as you develop scientifically, you start writing your own grants and submitting your own grants. They were getting funded. My grants were getting funded.

So at that point, I kind of separated, and I was doing my own basic research in the lab. I had lab space, I had technicians that I was directing. And then my appointment at that -- was assistant research professor. And I did that until about 2003.

Q. Okay. Now, if you would go to the second page of the slides. The first bullet point says over 80 abstracts, peer-reviewed publications and book chapters primarily on bone marrow and immunological toxicity and/or benzene.

What are abstracts pier reviewed publications, David? In other words, what -- what are the nature of your publications?

A. The -- I've combined them together for this point. But sciences gets out into the scientific community several different ways. And you can write book chapters. I've written one book chapter specifically on benzene. Peer-reviewed publications, those are primarily literature where you're describing your research. You submit it to a journal. They --

they send it out for external reviewers. And then they publish it in the literature. Abstracts were typically smaller pieces of your science that you have submitted to various scientific organizations, and you usually present those at -- at national or international meetings.

Q. Okay.

A. Not all of those -- not -- not every abstract is peer-reviewed, but the -- the meetings that I attend, they do have a peer review process for their abstracts.

Q. Okay. And, David, if you could just scoot a little bit over. The light is --

A. Yeah, sorry.

Q. -- is on the side of your head there.

THE VIDEOGRAPHER: Counsel, may we briefly go off the record.

MR. SCHIRRMEISTER: Yes.

THE VIDEOGRAPHER: Going off the record. The time is 9:46.

(Recess taken, 9:46 a.m. to 9:47 a.m.)

THE VIDEOGRAPHER: We are back on the record. The time is 9:47.

Q. (BY MR. SCHIRRMEISTER) Okay. David, the second bullet point describes some of your clients and

the government agencies you work for. Briefly describe those for the jury.

A. Since about 2003 when I officially left the university in a -- a research capacity and I worked for a couple of consulting companies, and then in 2000 -- the end of 2004, I started my own, which is called Summit Toxicology. And within those organizations, I've done a variety of projects for lots of different kinds of clients. Those include private sector, various industries, various law firms, but also a wide variety of regulatory agencies that -- some of which are listed here. I've done work for the USEPA. I worked for USEPA as a full-time employee for a while. Department of Health and Services, the CDC. I've done work for -- or we've done work for Health Canada, various state organizations: California, Texas, Colorado. So there -- it's been a long -- pretty long list of clients that we've had through the years.

Q. Okay. What type of work have you done for the centers for disease control?

A. Mostly just reviewed scientific literature.

Q. Okay. How about for the Environmental Protection Agency?

A. The Environmental Protection Agency, what I did for them -- well, I used to work for them. And then we were evaluating a specific hazardous waste site here in the Denver region called the Rocky Mountain Arsenal. So I was kind of an amateur toxicologist, because I was working with -- this is before I got my Ph.D., and I was working with a toxicologist on that site. I really liked him, really respected him. And, in fact, that was part of the motivation for me taking the vow of poverty, if you will, and going back to graduate school and getting my own Ph.D. in toxicology.

Q. Okay. Now, the-second-to-the-last bullet point is, "Organizer presenter, Benzene 2009, Health Effects and Mechanism of Bone Marrow Toxicity. What is that, David?

A. About every four or five years, all of the researchers across the country, really, across the -- the globe are -- who have an active research program or an active interest in benzene, they get together and put these symposiums together where people can discuss and -- and present all of the new things that have come out in the world of benzene toxicology. And the first one I attended was in 2004, and it was in Munich, Germany.

This one, 2009, I was on the planning committee, so I designed a web site for the meeting and was part of the review process for some of the abstracts for the posters. But anyway, it was a big meeting that was held two weeks ago in Munich, Germany. It was about four days' worth of presentation; so, you know, anyone really that had anything to say about benzene research and toxicology, most everyone was there.

Q. Okay. David, I'm going to hand you what has been marked as Defendants' Deposition Exhibit No. 1. Would you identify that, please.

A. This is my CV or resume.

Q. Okay. Is that a true-and-correct copy of the most recent CV that you have?

A. Yes. This looks to be very recent.

Q. Okay.

MR. SCHIRRMEISTER: I'll offer Defendants' Exhibit No. 1.

THE DEPONENT: What -- what do I do with it?

MR. SCHIRRMEISTER: Just put it down there. That's fine. Thank you.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I'm going to also hand you what has been marked as

Defendants' No. 2, which is slide page No. 4. And would you identify that first for the jury, please.

A. What you've handed me is what's on the projector there, the slide. This is a list of -- kind of a summary of my opinions in this case.

Q. Okay. Did you prepare that?

A. Yes.

Q. Okay. And that has been marked as Defendants' Exhibit 2?

A. Okay.

Q. Would you just confirm that, that's --

A. Oh, yes.

Q. Okay.

A. Yes.

MR. SCHIRRMEISTER: Okay. I'll offer Exhibit 2.

MR. GIANARIS: Over my objection.

Q. (BY MR. SCHIRRMEISTER) Okay. Now, David, from this point forward, when I ask you to express your opinions, would you agree to do that for me that the standard is based upon reasonable and medical scientific probability?

A. Sure.

Q. And if there's any -- any opinion that you have on any other standard other than reasonable

medical and scientific probability, please tell us that.

A. Okay.

Q. Otherwise, we will assume that all your opinions are based upon reasonable and medical and scientific probability.

A. Sure.

Q. Are you agreeable to that?

A. Yes.

Q. Okay. Now, what is the first opinion?

A. That the paint solvents in Dupont or PPG, or really any paint product in these solvents are not established carcinogenic agents. They don't cause cancer generally or leukemia specifically.

MR. GIANARIS: Object to form.

Q. (BY MR. SCHIRRMEISTER) Okay. I'm going to offer -- offer you -- offer as well Defendants' No. 3. Would you identify that, please.

A. This is my report that I wrote and submitted in this case.

Q. Okay. And is that a true-and-correct copy of the report that you prepared and submitted in this case?

A. It doesn't appear to have been changed, so, yes.

Q. Okay.
A. I would say it is.
Q. Okay. And along with the supplemental report, which we will tender later, is a summary of your opinions that's been marked as Exhibit No. 2 a summary of the report that's been marked as Exhibit No. 3?
A. Yes. Yeah, it may not be exactly in this order; but, sure, all of these things are definitely covered in my report, this declaration.
Q. Okay. And when you say all of these things, are you referring to the summary of opinions --
A. Sorry.
Q. -- that's been marked as Exhibit 2?
A. Yeah, sorry. Yes.
Q. Okay. Okay. Now, let's -- okay. Would you read that to the jury, please.
A. The first opinion was "Dupont and PPG paint solvents do not cause cancer or leukemia."
Q. Okay. Now, let's look at the next slide, please. And I'm going to hand you what has been marked as Defendants' Exhibit 4. Okay. David, what solvents are in Dupont and PPG paints?
MR. GIANARIS: Object to the form.

A. The predominant ones which you're going to find are going to be toluene, which I think is in the highest concentrations. Xylene. It depends on the product so that the -- the composition can change somewhat. And then a variety of things that would fall under the category of petroleum distillates. So that would be mineral spirits, Stoddard solvent, various naphthas, things of that nature, which, from a toxicological point of view, are all very -- are all very similar. And typically in these regulatory groups, they -- they -- they combine all of those together.

Q. Okay. And what do you understand the plaintiffs' claims to be in this lawsuit?

A. My understanding is that the -- the trace levels of naturally occurring benzene that are likely found in these various solvents were of sufficient magnitude to cause Mr. Wolfe's acute promyelocytic leukemia.

Q. Okay. You understand Mr. Wolfe never worked with benzene, correct?

A. I've never seen any evidence --

MR. GIANARIS: Object to form.

A. -- that that's true, yes.

Q. (BY MR. SCHIRRMEISTER) Okay. Now, on

the bottom of the slide, there are a number of different agencies. And then there are those color circles above in the graph. What -- are you familiar with organizations -- or are there organizations that study and evaluate chemicals as to whether they are capable of causing cancer or leukemia?

A. Yes.

Q. Okay. The first one on there is the IARC, I-A-R-C. What is IARC?

A. IARC is -- the -- the acronym stands for the International Agency for Research on Cancer. And it is an international grouping of preeminent scientists, toxicologists, physicians, epidemiologists that look at -- collectively look at the sum total of the published literature.

So for them, they have a bright line that it has to be published before it will be evaluated under the IARC kind of in their panel. But then they evaluate various chemicals, drugs, viruses, anything that is a potential cause of human cancer.

Q. Okay. David, the next one is N-I-O-S-H, NIOSH. What is NIOSH?

A. NIOSH stands for the National Institute of Occupational Safety and Health. And that's the research arm of OSHA. So they conduct research on

behalf of OSHA in support of OSHA's regulations that -- that they have.

Q. Okay. ACGIH, what is that, David?

A. That's the American Conference of Governmental Industrial Hygienists. And this is a -- I think it's actually voluntary, but this is a large group of industrial hygienists that, from all different occupations across the country, some academic, some in -- actually out in the workplace, but they get together and discuss what they call a TLV, which is a threshold limit value. And it's essentially what they consider to be a safe working level of these various chemicals.

Q. Okay. Next one is NTP. What is that?

A. That's the National Cancer -- sorry. NTP stands for the National Toxicology Program, so that's a -- a branch of the federal government that also has a lot of collaborators within university and academic institutions. And they are the ones -- and it's usually dictated by the Environmental Protection Agency, but they are the ones that conduct or oversee or interpret the data that comes out of long-term animal studies.

So if anyone wants to know, does my chemical cause cancer in one or more experimental

animals, then they submit that to the National Toxicology Program, they rank them based on their priorities, and then they actually go out and conduct the study, and then report back whether there was evidence of carcinogenicity in that -- in those species.

Q. Okay. EPA?

A. That's the Environmental Protection Agency.

Q. Okay. And what do they -- what is their role with regard to determining whether toluene, xylene, or petroleum distillates are capable of causing cancer or APL?

A. Well, EPA is a -- their -- their role is to protect the environment and people that are exposed to chemicals from the environment, as well as animals and ecosystems. But they, for various chemicals -- excuse me -- publish -- put out what's called an IRIS database. And within that, they classify chemicals according to their toxigenicity.

Q. Okay. And, finally, the State of North Carolina. How does the State of North Carolina regulate or interpret the potential carcinogenicity of toluene, xylene, or petroleum distillates?

MR. GIANARIS: Object to the form.

A. My understanding is -- well, lots of states have environmental departments. My understanding of the State of North Carolina is they rely on these national organizations like OSHA, NIOSH. They -- they -- they base their decisions on what these other groups -- I don't think they do any independent evaluation.

Q. (BY MR. SCHIRRMEISTER) Okay. Now, let's --

A. And we skipped one, the AT -- ATSDR.

Q. Oh, I'm sorry. What is the ATSDR?

A. Just because there's a dot on there. That's the Agency for Toxic Substances and Disease Registry. So it is a specific branch of the CDC. The CDC is the Center for Disease Control. And they are tasked with and their -- their sole job is to go out and look at environmental exposures typically from hazardous waste sites, from Superfund sites, and conduct studies to see if the people living in proximity to these hazardous waste sites -- if there's any evidence of toxicity associated with those exposures.

Q. Okay. Now, I'm going to ask you questions about the classification by these agencies for these three chemicals. Okay?

Now, has the IARC ever classified toluene with naturally occurring trace benzene as a che -- as a chemical that causes cancer?

A. Say that again. I'm sorry.

Q. Yes. Has IARC ever classified toluene with naturally occurring trace benzene as a chemical that causes cancer?

A. No.

Q. Has IARC ever classified toluene with naturally occurring trace benzene as a chemical that causes APL?

A. No.

Q. Okay. Same question with regard to toluene and NIOSH. Has NIOSH ever classified toluene with naturally occurring trace benzene as a chemical that causes cancer or APL?

A. No. NIOSH puts out a report where they make a -- a comprehensive listing of all of the chemicals that they believe there is some evidence of carcinogenicity. And toluene is not included on that list.

Q. Okay. The ACGIH, David, has the ACGIH ever classified toluene with naturally occurring trace benzene as a chemical of causing cancer or APL?

A. No. The ACGIH publish what I consider to

be really helpful, relatively short summaries of the toxicity have these various chemicals. And it's their documentation or their support for what they think the TLV would be, the threshold limit value. And within that, for toluene, they specifically say that there is not enough evidence to support that this is a human carcinogen.

Q. Okay. The NTP, the National Toxicology Program. Has the NTP ever classified toluene with naturally occurring trace benzene as a chemical that causes cancer or APL?

A. Again, like NIOSH, the NTP puts out these reports periodically. And the most recent was the eleventh report on carcinogenicity is what they call it. And they list very comprehensively any chemical for which there is evidence in their animal studies that there was evidence of carcinogenicity. And toluene is not listed on that.

Q. Okay. The next one is the ATSDR of the Centers for Disease Control. Has that agency ever classified toluene with naturally occurring trace benzene as a chemical that can cause cancer or APL?

A. It has not. The ATSDR publishes these exhaustive -- basically they are books that are called toxicological profiles on these various chemicals.

And within those toxicological profiles, they have all of the animal data, all of the epidemiology, all of the metabolism, all of the toxicity data associated with that chemical that they could find in the literature. And, no, the ATSDR does not believe that toluene is a carcinogen.

Q. Okay. The EPA, David. Does the Environmental Protection Agency -- has it ever classified toluene with naturally occurring trace benzene as either a chemical that causes cancer or APL?

MR. GIANARIS: Object to the form, foundation.

A. The USEPA publishes what's called the IRIS, Integrate Risk Information System. And in IRIS, it is a more condensed form than the AT -- ATSDR tox profiles, but it still goes through a relatively comprehensive review of the literature. And they have a various gradation of whether they think it's a known human carcinogen or whether it might be a human carcinogen. And toluene is neither of those in their opinion.

Q. (BY MR. SCHIRRMEISTER) Okay. And then, finally, the State of North Carolina. How does the State of North Carolina classify these -- classify

toluene as to whether it's capable of causing either cancer or APL?

MR. GIANARIS: Object to the form, foundation.

A. My understanding is that they are going to base their classification system on these other organizations. So there is nothing any different that they would say that hasn't been already said in these other groups by these other groups.

Q. (BY MR. SCHIRRMEISTER) Okay. Now, let me ask you about xylene next, David. Has the IARC, NIOSH, ACGIH, the NTP, the ATSDR, or the EPA ever classified xylene with naturally occurring trace benzene as a chemical capable of causing cancer?

MR. GIANARIS: Hold for one second before he answers. Did you put -- was North Carolina in that question? I didn't catch it.

MR. SCHIRRMEISTER: No.

MR. GIANARIS: So I don't interrupt you, may I just have a continuing objection to foundation as to questions regarding North Carolina?

MR. SCHIRRMEISTER: Yes, sir.

MR. GIANARIS: And you all can proceed. Thank you very much.

MR. SCHIRRMEISTER: Repeat the question?

A. Are we on? Am I going? I heard the question.

Q. (BY MR. SCHIRRMEISTER) Okay.

A. The answer is, no. I mean, xylene is not a thing. It's a -- it's -- actually, there's three different kinds of xylene, but none of them have been classified by any regulatory agency that I am aware of, including the ones listed here, as a human -- as a human carcinogen.

Q. Has the IARC, the International Agency for the Research of Cancer, the NIOSH, National Institute Occupational Safety and Health, the ACGIH, the American Conference of Governmental and Industrial Hygienists, the NTP, the National Toxicology Program, the ATSDR, the Agency For Toxic Substances and Disease Research, or the EPA, the Environmental Protection Agency -- has any of those agencies ever classified xylene with naturally occurring trace benzene as a chemical capable of causing APL?

A. No.

Q. Okay. Finally, to the petroleum distillates, same two questions, David. Has IARC, NIOSH, ASGIH, NTP, ATSDR, or the EPA ever classified petroleum distillates with naturally occurring trace benzene as capable of causing cancer?

A. Not that I've ever seen. And they -- there are -- depending on which group you're looking at, IARC or ASGIH or the ATSDR, they -- some of them have data on naphthas, some of them have data on mineral spirits, some of them have data on Stoddard solvents; but all of those things are chemically similar enough to where, from a toxicological point of view, it is fair to put them all into this group called petroleum distillates, which is what we did. And the answer is, no, none of those groups consider any of those various things that we would call a petroleum distillate as a -- a human carcinogen.

Q. Okay. And then, finally, do any of those agencies: The IARC, NIOSH, ACGIH, NTP, ATSDR, or EPA ever classify this group of chemicals under the name petroleum distillates as ones capable with naturally trace benzene as ones capable of causing APL?

A. No.

Q. Okay. Now, with regard to the classification by toluene -- of toluene, xylene, and petroleum distillates by the State of North Carolina, how do you understand the nate of -- State of North Carolina classifies and regulates these three chemicals?

A. My understanding is that -- and I've

never done any work for the State of North Carolina, so I'm not exactly sure what their inner workings are, but that they rely on OSHA and IARC. And these are their international and national agencies, and they base their decisions on what these other national groups say.

Q. Okay.

MR. GIANARIS: Same objection.

Q. (BY MR. SCHIRRMEISTER) David, does Exhibit No. 4 -- does this accurately portray the regulatory classifications of toluene, xylene, and petroleum distillates with naturally occurring trace benzene with regard to their classification as capable of causing cancer or APL?

A. Certainly my interpretation of -- of their various regulatory documents, yes.

MR. SCHIRRMEISTER: Okay. I'll offer Defendants' Exhibit 4.

MR. GIANARIS: Over my objection.

MR. SCHIRRMEISTER: We'll leave that there. Thank you, sir.

Q. (BY MR. SCHIRRMEISTER) David, would you turn -- I'm going to now hand you what has been marked as Exhibit 5.

A. Okay.

Q. And it is three pages long. And would you identify Exhibit 5 for us, please.

A. Yeah, this is what I was talking about with the NTP. They periodically publish these reports on carcinogens. And they are pretty -- pretty conservative with the listings. And by that I mean, if there's any evidence in their animal studies that there is an increased risk of tumor genesis in any of these species or that there's evidence of carcinogenicity in these species, then it makes it onto this list.

Q. Okay. So -- and is this the most recent list issued by the -- the National Toxicology Program?

A. The eleventh report is the most recent one that I've seen. When this one comes out, then they are automatically working on the next one. So internally, there's probably a twelfth; but this is the only one that I think is publically available.

Q. Okay. And anywhere on this eleventh report, that lists all of these different chemicals that cause cancer, do toluene, xylene, or petroleum distillates appear?

A. No.

MR. SCHIRRMEISTER: I offer Defendants' Exhibit 5. Would you like to take a break? Let's

take a five-minute break.

THE VIDEOGRAPHER: Going off the record. The time is 10:09.

(Recess taken, 10:09 a.m. to 10:19 a.m.)

THE VIDEOGRAPHER: We are back on the record. The time is 10:19.

Q. (BY MR. SCHIRRMEISTER) David, would you read the slide, which is page 11, please.

A. Oh. The APL, which stands for Acute Promyelocytic Leukemia, is a distinct clinical disease and is different than other subtypes of AML, which is acute myeloid or myelogenous leukemia.

Q. Okay. David, I'm going to hand you what has been marked as Exhibit 6.

A. Okay.

Q. And this is called "Why APL is a unique clinical disease," correct?

A. Yes.

Q. And just take us -- and I'm going to go back in detail, but -- what is No. 1, morphology? What does that mean?

A. Morphology is the shape of the cell. So that would be a way to describe how the cells would look microscopically. So if you took them out, looked at them under a microscope, a hematologist might do

that, they -- they have a very distinctive morphologic presentation. You would be able to recognize them.

Q. And are you talking about an APL cell?

A. Correct, APL.

Q. Okay.

A. Well, they all do. All of the different subtypes of AML have unique -- well, not all of the them, but what -- morphologic presentations, but APL is quite different than the other ones.

Q. And are -- is morphological a fancy way of saying it looks different under the microscope than other cells?

A. That's exactly what that means.

Q. Cell of origin, what does that do?

A. Cell of origin is we're -- we believe that the malignant transformation or the thing that causes the cell to become leukemia where that happens within the hematopoietic progression, which I think we have a slide or two to discuss.

Q. Okay. Psychogenetics, what is that data?

A. Sorry. Cytogenetics, that is a branch of medicine, science where you're looking at the shape of the chromosomes. So basically how many chromosomes are there, whether some are missing, some or broken, whether they are combining with each inappropriately.

So that's just a way to describe what the chromosomes look like. And that is critically important in the diagnosis and the prognosis for -- for all forms of leukemia.

Q. Okay. Treatment?

A. The treatment for A -- APL is categorically different than the treatment for -- for any other -- any other type of leukemia that I'm aware of.

Q. Okay. And lastly, epidemiology, what is that?

A. Epidemiology is the science where you're looking at what causes diseases in the human population. And because APL is unique enough, there have been epidemiological studies looking at it as -- not as a subtype of AML, but as its own unique entity.

Q. Okay. And are these five factors ones that you've identified to explain why APL is a unique disease?

MR. GIANARIS: Object to the form. Leading.

Q. (BY MR. SCHIRRMEISTER) Okay. What are these five factors? Why have you chosen these five factors?

A. These -- these categories are ways in

which there is clear -- there are clear differences between APL and the other forms of acute myelogenous leukemia.

Q. Okay.

A. So it is different -- distinctly different in each one of these categories.

Q. Okay.

MR. SCHIRRMEISTER: I'll offer Defendants' Exhibit 6.

MR. GIANARIS: Over my objection.

Q. (BY MR. SCHIRRMEISTER) Okay. David, now I'm going to put up what I will hand you and mark -- it's previously been marked as Exhibit 6A. And why don't you stand up, if you would, so you can explain to the jury --

A. Okay. I'll try not to trip.

Q. -- what this is.

A. This is going to work? Yeah. Okay. This is a slide that I actually made for a talk that I gave at SOT. And at that -- the Society of Toxicology. And what I was doing in that presentation was describing the role that the bone marrow microenvironment plays in various hemopoietic diseases. Why I have it in this presentation or today is it just gives a very simplistic view of the process

of hematopoiesis.

Q. Okay. Now -- okay. Okay. I'm going to interrupt you, David.

A. Okay.

Q. Have you spent -- how much of your career have you spent on this issue?

A. On hematopoiesis?

Q. Yes, sir.

A. That's -- that's really all that I've done from a research point of view as well as a consultant. It's either been hematopoietic development or toxicity in the bone marrow that impacts on hematopoietic development. That's -- that's what I do.

Q. Okay. Now, let's start with the left-hand side where it says, "bone." What are we looking at there and what is happening? What happens in your bone marrow?

A. Yeah, this is just a depiction of a bone. And this we don't really need to talk about. It's just kind of a blow-up of this region right on the inside of the -- the -- the trabecular bone. But it's demonstrating -- these cells here: Killer cells, lymphocytes, basophils, platelets, red blood cells, all of these things, these are cells that you would

recognize in the peripheral circulation.

So if I took blood out of anyone here and looked at it under a microscope, I would see these various cell types. So everyone knows what they are probably and what they do. But what is beautiful about the hematopoietic system is that all of these various cell types all are thought to be derived from an ancestral cell that resides in the bone marrow.

Q. Okay. Now, as a beginning definition, hematopoietic, what is that -- what's that a big word for?

A. Hematopoietic is blood. It's -- so hematopoietic pieces is blood formation. So that's what's going on in the bone marrow that is producing the billions of red blood cells that we have and all of the other cell types that are -- that are found in the peripheral circulation.

Q. Okay. Now, you mentioned stem cell. What is the stem cell?

A. Well, this is a word that's given to -- you know, I mean, you'll get hematologists together, and they will argue passionately about the definition of a stem cell. It's pretty irrelevant. But what the stem cell is is a cell that retains the ability to turn into all of these different things.

So a cell at this level, it can turn into lymphocytes, it can turn into a red blood cell. But as it progresses through its maturation, eventually, it starts becoming what we consider to be lineage committed. And that point, it is only going to turn into one cell type.

Q. Okay.

A. And once it lineage commits and says, okay, I'm going to be a red blood cell, then it won't have the ability -- it will not have the ability to turn into these other cell types.

Q. Okay. Now, how -- generally, how many different blood cell types are there for our purposes?

A. I -- that's a hard question to answer.

Q. Okay. Well --

A. Six, seven.

Q. Well, I've heard of red blood cells. What is the function of a red blood cell?

A. The -- they -- have hemoglobin in them, so their primary function is to carry oxygen.

Q. Okay.

A. Or to carry carbon dioxide away from the tissues.

Q. Okay. White blood cells?

A. White blood cells, there's a lot of

different kinds of white blood cells, which is why I paused on your question. There probably are five or six different things that a hematologist would be interested in knowing if they were looking at your blood, all of which would be considered a white blood cell.

In general, the white blood cells, all of them collectively, their primary role is to fight infection.

Q. Okay. And then how about a platelet? What is the function of a platelet?

A. A platelet comes from a megakaryocyte, which is this thing, and its primary role is in blood clotting or wound healing.

Q. And how does a stem cell become all of these different types of blood cells? Just walk us through that process, David.

A. I don't -- that's a really hard question.

Q. Okay.

A. I mean, basically, you've got a very elaborate system of growth factors and feedback loops. And it's a very complicated process; but, essentially, this cell becomes more and more committed, more and more mature, more and more differentiated as it's moving through this pathway.

Q. Okay.

A. And it's also proliferating, so you're getting more and more of them. But that's not -- I can't answer that question in two minutes.

Q. Okay. How about -- well -- how about this, the different types of cell lines. How many different types of cell lines are there in the blood? And if you would show where those are on Exhibit 6.

A. You know, from a hematopoietic -- I mean, all of these are different cell lines. But typically, hematologists will consider three -- there are platelets, red blood cells, and white blood cells; so those are considered to be the three main cell lines.

Q. Okay. And I've heard of the term lymphoid lines and myelocytic or myeloid lines.

A. Right. Well, that's a whole different classification system. It doesn't fit in with what I just said. So you don't really consider those cell lineages based -- it depends on how you're defining those terms. But you do have lymphoid cells -- and this isn't a very good picture to depict that, but you do have lymphoid cells and you have myeloid cells, and that is a pretty major break in the differentiation of these cells.

Q. Okay. And where -- where is the cell

here that is the APL cell?

A. You --

Q. Or should we look at it in a different slide?

A. You can't see it on this slide.

Q. Okay.

MR. SCHIRRMEISTER: I want to -- I'd like to offer Exhibit 6A.

MR. GIANARIS: Over my objection.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I'm going to hand you what has been marked as Exhibit 7, please.

A. Okay. Um-hum.

Q. Now, would you explain -- first of all, explain what chromosomes are.

A. Chromosomes are the structures within the nucleus of a cell that -- that are within a cell that -- that contain all of the genetic information.

So the genetic information, hair color and weight and height, and all of the other things that make a person a person, all of that is contained in the DNA. And that DNA is really long, and so you can't just have these really long strands -- you know, hundreds of yards, this DNA is, and you can't have these strands of DNA just flopping around. So they

are condensed. They are wrapped up around certain structures into very nice, neat, little tight packages. And those little, neat, tight packages, you can see under a microscope, they look like this, and we call those chromosomes. But really all that is is a tight collection of DNA that can be unwound when you're expressing certain proteins. So those are your chromosomes.

Q. Okay. And how many chromosomes do -- do we all have in each cell?

A. 46.

Q. Okay. And do the pairs of chromosomes have different functions in the body?

A. Each chromosome has different roles, correct.

Q. Okay. Now, give us an example that the jury may be familiar with of a chromosomal problem that leads to a disease or leads to a condition.

A. Really, for as long as we've been able to identify chromosomes -- and that hasn't been that long ago. I mean, probably in the '50s was the first time that we were able to clearly identify -- reproducibly identify chromosomes. We've known that if you see alterations in the karyotype or you see alterations in the number of chromosomes that it can have important

pathologic manifestations or diseases that can arise from that.

Probably the most common, I think the one that most everyone has heard of is a disease called Down's Syndrome. And Down's Syndrome means you have an extra copy of chromosome 21. So you have three copies of chromosome 21.

Q. Okay.

A. Now, we don't know exactly how that extra copy manifests itself in Down's Syndrome, but it's clear that that is the basis for that -- that syndrome.

Q. Okay. Now, you have here chromosomes in the 15;17 translocation. How is the -- how does a chromosomal condition in APL, the disease that Mr. Wolfe had -- how do they relate to one another?

A. The -- the cytogenetics, however, are extremely important in defining any type of leukemia. And, in fact, of the New World Health Organization in 2008 for diagnosing and identifying leukemias, they are relying more and more and more heavily cytogenetics.

So different forms of leukemia have specific cytogenetic problems associated with those. And hematologists are keenly interested in that

because it allows them to accurately diagnose it, it allows them to give a good prognosis or an accurate prognosis to the family or the patient as well as more appropriately treating it.

Acute promyelocytic leukemia or APL has a very distinctive cytogenetic abnormality associated with it, and that is a translocation between chromosomes 15 and 17.

Q. Okay. So explain to us what is happening here that leads to APL.

A. This 15;17 translocation, it is a hallmark of APL. So in the United States, anyway, you -- they will not diagnosis a patient with APL unless they see this 15;17 translocation. And the translocation means that a little piece of chromosome 15 is going over here. And this is chromosome 17. And a little piece of chromosome 17 is moving over here. So some of 15 is now part of 17 and some of 17 is now part of 15. And you can't see it on these slides, but chromosomes are banded. So there is a unique set of bands that allow people, under a microscope, to identify which piece of which chromosome belongs where.

So you can see this under -- under an appropriate microscope.

Q. Okay. And does Mr. Wolfe have a 15;17 translocation?

A. Correct, he does. And when you get this 15;17 translocation -- and this is a little complicated, but the result of that translocation is what's called a fusion protein. And it is because you have these pieces that are combining with each other inappropriately. And that's what this is down here. And we don't know exactly how it works, but this fusion protein is thought to be the -- is thought to underlie why someone gets APL.

Q. Okay. Very simplistically, would it be correct that some of the information that should be on chromosome 15 ends up on chromosome 17 and some of the information that should be on chromosome 17 ends up on chromosome 15?

MR. GIANARIS: Object to the form.

A. That's exactly what happens.

Q. (BY MR. SCHIRRMEISTER) And does that lead to APL?

A. It certainly leads to something that you can always see in APL.

Q. Okay.

A. You know, we're not at a point where we can absolutely draw out a path and say here's exactly

where the transformation occurs, but this is always in APL. So clearly, it has something to do with the disease.

Q. Okay.

MR. SCHIRRMEISTER: Offer Defendants' Exhibit 7.

MR. GIANARIS: Over objection.

Q. (BY MR. SCHIRRMEISTER) Okay. I'm now going to offer what has been marked as 8B.

MR. GIANARIS: You're offering it into evidence or just showing it to him.

MR. SCHIRRMEISTER: I'm going to show it to him first.

MR. GIANARIS: So you haven't offered it yet?

MR. SCHIRRMEISTER: I will in a moment.

MR. GIANARIS: Okay.

MR. SCHIRRMEISTER: I will -- I will, 8B.

MR. GIANARIS: I'll make my objection now and we'll be all right. Mr. Schirrmeister, just so I don't interrupt you so you don't have to edit this as much if you play it, may I just have a standing objection to offering the exhibits that you guys have created into evidence so I don't have to make an objection every time?

MR. SCHIRRMEISTER: Sure.

MR. GIANARIS: Okay. Thank you.

Q. (BY MR. SCHIRRMEISTER) Okay, David. Now, this is called hematopoiesis, correct?

A. Yes.

Q. And tell us -- and I -- we -- we prepared this slide in connection with the treatment, understanding the disease and the treatment, correct?

A. Yes. I think that's why we put it in. It actually provides a clearer picture of what you were asking me earlier about the different lineages, because they are all listed here --

Q. Okay.

A. -- pretty clearly. And it also depicts how these -- this differentiation occurs. As the cells are marching through this pathway, they are picking up characteristics of these various cell types. So they start picking up the functionality. Like for the red blood cell, somewhere around in here, they start expressing hemoglobin. Okay. This cell doesn't express hemoglobin, so it -- it has said, I'm going to be a red blood cell, and it has started in that process to allow it to be an erythrocyte. So that's -- that is depicted very nicely on this slide.

Q. Okay. Now, show us where on this slide

you believe the APL disease develops.

A. This is called a granulocyte. And there's actually lots of different kinds of granulocytes, so this is a mature cell that you can find in the periphery. And some of the precursor cells or the cells that are more immature but are leading to the formation of a granulocyte have different names. This one is known as a myelocyte. This one is called a promyelocyte. Pro means it's a little earlier than that. And so promyelocytic leukemia is happening at this level.

Q. Okay. And the -- what -- what -- the myelocyte, what type of cell is that? Is it a red blood cell, a white blood cell, a platelet? What type of cell is it?

A. Yeah, all of these are going to be -- all of these are going to be white blood cells. This is the only red blood cell. This is the only platelet. Even lymphocytes would be considered white blood cells. So that determination doesn't really fit with this categorization, but these are -- are going to be granulocytes. They are white blood cells. They are important in fighting infections.

Q. Okay. So the -- a cell that is affected in APL, what is its function in the blood?

A. The cell that is affected in APL, its function is just to continue to mature and differentiate and ultimately produce a granulocyte, whose role is to fight infections.

Q. Okay. Now, tell us what the disease of APL is vis-a-vis what is happening on Exhibit 8B.

A. Yeah. What happens is -- which is why the treatment is -- is so beautiful, really, is that we know that in this process in patients that have acute promyelocytic leukemia, the process stops right here or, perhaps, right here; but it is not allowed to take the final differentiation step.

So it stops at a promyelocyte stage. So you get an increase in promyelocytes. Those do not take the next step and produce functioning granulocytes.

So the individuals don't -- they are not as capable in fighting infection, they don't have enough granulocytes, and they have too many of these cells that are -- that are present in the bone marrow or the periphery.

Q. Okay. And is that the APL disease?

A. That is the APL disease. You have -- the leukemia is that you have a huge collection of these promyelocytes that are not fully functional, so they

are not really doing anything; but they are blocked in their differentiation probably by that fusion protein that I showed you on the 15;17. That blocks it in its differentiation and -- and prevents it from turning into a granulocyte.

Q. Okay. And how -- what -- what is the standard treatment and how does it work?

A. The standard treatment, the first -- the first stage treatment is a drug called altransretnoic acid. And altransretonoic acid, it really is kind of magical, because what it does is it allows that block and differentiation to be overcome.

So it allows this promyelocyte to take these additional steps and differentiate and mature into a granulocyte. Now, why that's important in treating leukemia is that granulocytes only live about eight hours. So they have a relatively short half life. So when you -- this one can live for a long time. But when you allow a promyelocyte to convert itself to a granulocyte, it dies on its own. So the drug, altransretnoic acid, doesn't have to kill proliferating cells, like most other chemotherapeutic agents do. It is a differentiation agent, and it allows this cell, basically, to kill itself by converting itself to a granulocyte.

Q. Okay. How -- and what is the name of that treatment?

A. The drug is called altransretonoic acid, ATRA.

Q. Okay. And that is the treatment that Mr. Wolfe received?

A. Yes.

Q. Okay. And is -- and how is the altrans -- this treatment, altransretonoic acid treatment, different than other chemotherapy drugs such that it is unique?

A. There's no -- there's really nothing like it. Any -- in any treatment of any disease that I've seen, altransretonoic acid induces differentiation like I just described. So it's causing these cells to turn into granulocytes; they die on their own.

Every other form of treatment for various types of leukemia -- now, there is a new one for CML. But for AML, all of the different subtypes of AML, they are treated with anti-proliferating agents. So these are alkylating agents, or somehow these are drugs that kill cells that are dividing. Since the leukemia cells are dividing, the thought is, well, we can kill the leukemia cells, and that's going to help the patient. So all of these drugs -- and there's,

you know, too many to list, but they all act basically the same way, and that is they are killing proliferating cells.

Q. Okay.

A. Because proliferating dividing cells are not just leukemia cells, and you've got dividing cells all over the place, then that causes a lot of the side effects that people know and appreciate with these chemotherapeutic agents, like hair falling out, because the hair follicles have a lot of proliferating cells in them. And some of the nausea that's associated with these chemotherapeutic agents, because it's killing these proliferating cells in the GI tract. So -- I mean, altransretonoic acid has some side effects, too, but it's a different -- totally different process than what are treating the other AML types.

Q. Okay. And then the next --

MR. SCHIRRMEISTER: I'm going to offer Exhibit 8B. Okay.

Q. (BY MR. SCHIRRMEISTER) David, the next reason you mention was epidemiology. And what is this article?

A. This is a study by Dr. Douer. It's in California. And he's not the only one, but this is

where he went out and, recognizing these distinct clinical characteristics of APL, he published a paper on the epidemiology of this specific subtype.

Q. Okay. Now, some experts on behalf of the plaintiffs, a Dr. Milman, I believe, has testified that it is not possible to conduct epidemiology studies on APL. Do you agree with that?

A. I -- I don't understand the basis for that comment, because we're -- we're going to talk about lots, and there are many that we aren't going to put in this presentation. So APL -- either APL or the 15;17, which they are synonymous, they are the same thing, people look for those all of the time and have done studies on those all the time.

Q. Okay. Are any other forms or subtypes of AML studied uniquely in epidemiology?

A. To my knowledge, there are no more FAB subtypes, but people are starting to think -- epidemiologists today are starting -- they recognize that the cytogenetic changes, let's say, an 8;21 translocation, every one that has an 8;21 translocation, their leukemias are very similar to one another. So they are starting to conduct epidemiological studies based on these cytogenetic profiles.

So people are recognizing that that is a more appropriate way of grouping these diseases, so the 15;17 or 8;21 or inversion 16 or any of the other ones, that is where the field of epidemiology is going. Up to this point, the answer to your question is no.

Q. No. Okay. Now, tell us why you've selected this study and what the significance is.

A. Well, I selected this study because it specifically addressed the question at hand, and it illustrates pretty clearly that Dr. Douer, based on his research, concluded that APL is very different from the other subtypes of -- of AML. In fact, I'll just read it. "Descriptive epidem" --

Q. Why don't -- just -- if you would -- well go ahead read the top and the bottom, too. I'm going to ask you about the bottom.

A. Do you want me to read the top?

Q. Why don't you read the bottom.

A. Okay. "It is, therefore, likely that etiological factors for APL are different from those of other AML subtypes. So far, no environmental and/or occupational risk factors have been found for APL."

Q. Okay. And -- and that is based on an

epidemiology study?

A. Yes. And his review of the literature and his understanding of the disease and all of the things that we've been talking about.

Q. Okay.

MR. SCHIRRMEISTER: I'm going to offer Exhibits 9 and 10.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I'm now going to hand you what has been marked as Exhibits 11 and 12. Would you identify those, please.

THE REPORTER: Can we go off the record for a second, please?

MR. SCHIRRMEISTER: Yeah.

THE VIDEOGRAPHER: Going off the record. The time is 10:45.

(Discussion off the record.)

THE VIDEOGRAPHER: We are back on the record. The time is 10:45.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I've handed you what has been marked as Exhibits 11 and 12. Would you identify these for us, please.

A. Oh, right. This is another study that really did the same thing. The epidemiology of acute promyelocytic leukemia in Italy. Recognizing that it's a distinct disease, they went out and conducted

an epidemiological study and looked for occupational risk factors associated with this specific disease, APL. And as you can see -- and I think it might be on the second page, but they concluded, "The results obtained in our study confirm that epidemiologically, as well as clinically and hematologically, that APL behaves differently from other subtypes of" -- this is acute non-lymphocytic leukemia, which is just another way of saying AML basically.

Q. Okay. Now, David, I'm going to go back to your five factors that you identified. Would you, again, describe the significance of these five factors with regard to how APL is a different disease than AML?

A. Sure. We didn't show any pictures; but morphologically, you can identify APL, and it is different than the other subtypes. That's probably less important, because there can be variability in the way the cells look.

The cell of origin we discussed. And that the cell of origin for APL is thought to arise at a different place than the other subtypes of AML subject to some -- you know, some discussion in the literature about, you know, really where that happens. We don't know definitively where it occurs, but it

seems to be happening where I pointed out.

The cytogenetics -- this is totally distinct. There is nothing else like APL that has the 15;17. If it's a leukemia and it has 15;17, it's called APL. If it's APL, it has the 15;17. The treatment -- I only talked about altransretonoic acid, because that is the front-line therapy. If that fails, there are other things. And eventually, if everything fails, they will fall back on anti-proliferation agents and, you know, just try to kill dividing cells. But most people have pretty good response to altransretonoic acid. And because it is a differentiation agent and not just killing dividing cells, it's -- it's unique. There's really nothing else like it in the treatment of leukemia. And then as we just discussed, there are two nice examples of epidemiological studies where they concluded, they agreed that APL is different. It is not just another subtype of AML.

Q. Okay. And if the plaintiff -- if a plaintiff's expert were to testify that there is no difference between an APL and an AML, would you disagree with that?

A. I -- I would hope that everyone would disagree with that. But, yes, I would, for sure.

Q. Okay.

MR. SCHIRRMEISTER: I'm going to move and I would like to offer into evidence Exhibits 11 and 12.

THE DEPONENT: I'm not keeping these in a very nice order over here.

MR. SCHIRRMEISTER: That's all right.

Q. (BY MR. SCHIRRMEISTER) Okay. David, why don't you sit now. And I'm going to ask you about --

MR. GIANARIS: Sit.

Q. (BY MR. SCHIRRMEISTER) -- ask you about a few other things. The next opinion that you have, would you read this opinion, please.

MR. GIANARIS: Object to the form, leading.

A. I'm going to get to where we are, so --

Q. (BY MR. SCHIRRMEISTER) Yes, I'm sorry.

A. That's fine. I got it.

Q. It's page 18 on the slides.

A. That the best available scientific literature does not support a link between benzene and acute promyelocytic leukemia or APL.

Q. Okay. And, again, I asked you earlier what you understood the plaintiffs' allegations in this case to be. How is that statement relevant to

what you understand the plaintiffs' allegations in this case to be?

A. Well, I think this is completely germane to this question. I mean, I think what they want to say is that there was enough benzene in these solvents to cause leukemia, not segregate out and discuss APL as a distinct entity, and not back away and talk about toluene and xylene, but try to make this about, you know, a really, really minor component in those solvents. So I think that this question here has been asked, and it is directly relevant to what they are alleging.

Q. Okay. So is the --

MR. GIANARIS: Object -- object to the form. Nonresponsive. Move to strike.

Q. (BY MR. SCHIRRMEISTER) Is the al -- is the -- is -- the next slides that we'll be seeing, have you examined the literature in order to identify studies that question whether there is a link or an association between benzene and Mr. Wolfe's disease, APL?

MR. GIANARIS: Object to form.

A. As far as I know, I have looked at all of the scientific evidence that could be used to discuss a link between benzene or organic solvents or

occupational exposures and the development of acute promyelocytic leukemia.

Q. (BY MR. SCHIRRMEISTER) Okay. David, would you scoot a little over. You're in the --

A. Yeah.

Q. Sorry about that. Okay. Now, I'm going to hand you what's been marked as Exhibits 13 and 14.

A. Okay.

Q. Now, tell us what type of studies have you looked for? What type of question were you attempting to answer as you examined the literature here?

A. Well, I mean, I -- I cast the net as wide as I possibly could, and I looked for what anyone -- what anyone has said about the epidemiology associated with APL. And I've looked at what anyone said with regard to what benzenes can do and the various subtypes of AML. So I've -- I've looked at all of the literature that I can think of that is -- that has some angle of relevance to this question.

Q. Okay. Now, would you identify this article, please, and tell us the author and what it was about.

A. This was a study published by Dr. Mitelman. And chromosomal pattern -- the title

is, "Chromosome pattern, occupational, and clinical features in patients with acute non-lymphocytic leukemia," which is another word for AML. And they -- they really did exactly what we're talking about. What he wanted to do was to understand whether there was a specific chromosomal pattern or a specific pattern of various types of AML associated with occupation. And one of the diseases that he segregated out was acute promyelocytic leukemia. And as you can see in the exposed group, they were exposed to solvents, insecticides -- I mean, there were different categories -- or petroleum solvents. There was only one case of APL in that group. There were five cases in the nonexposed. So this -- I mean, he clearly did not see an increase in APL with regard to these exposures.

Q. Okay. So does this graph, then, show that people who were not exposed to solvents, insecticides, or petroleum products were -- there were five people who developed APL, whereas the people who were exposed only developed -- there was only one?

MR. GIANARIS: Form objection.

A. That's correct. That's correct. In that, you know, the overriding conclusion was that that red box that there's more APL cases in the

unexposed individuals.

Q. (BY MR. SCHIRRMEISTER) Okay. And what does that tell you with regard to Mr. Wolfe's claims in this case?

A. Well, this study would not support that anything that he was exposed to, as far as solvents go or petroleum products, or anything that he was doing, played a role in the development of his disease.

Q. Okay.

MR. SCHIRRMEISTER: Offer Exhibit -- Defendants' Exhibits 13 and 14.

Q. (BY MR. SCHIRRMEISTER) Okay. David, would you look at -- I'm going to hand you what is marked as Exhibit 15 and 16. And is this another article?

A. Yes. Yes. This was by Dr. Golomb at the University of Chicago. And James Vardiman -- I just noticed that. And James Vardiman was an author as well, basically doing the same thing. They were looking for a correlation with occupation. Here were the groups of solvents that they looked at and whether or not they saw a difference in karyotypes, whether they saw any change in -- in this case, they were looking for 15;17, but that really is the same epidemiological question, is trying to understand

whether people are getting APL. So those -- because those go hand in hand.

Q. Okay. And how -- how many people were exposed and developed APL versus how many people who were never exposed and developed APL?

A. There was one case of APL out of the exposed group, and there were four cases of APL in the control group. So --

Q. Okay.

A. -- clearly this cannot be used to support the position that exposure to insecticides, solvents, or petroleum products increased the risk of developing the 15;17 translocation, which is APL.

Q. Okay. And how -- what is the relevance of this to Mr. Wolfe's claim that exposure to toluene, xylene, and petroleum distillates which naturally occur in trace benzene caused him to develop APL?

A. Well, I think those would either fall into the solvent category or these petroleum products would have just as much benzene in them or perhaps more than what toluene and xylene is going to have in it. And there is just not -- based on this study, there is not a relationship between exposure to those solvents and this specific disease.

MR. SCHIRRMEISTER: Okay. Offer

Defendants' 15 and 16.

Q. (BY MR. SCHIRRMEISTER) David, I'd like -- I'm going to hand you what has been marked as Exhibit 17 and 18.

A. Okay.

Q. And is this another study?

A. Yes.

Q. Okay. And tell us what the -- who the author is and the name of the study.

A. Dr. Crane. I -- I don't know him. But this is a Journal of the American Medical Association. But it was really the same thing, environmental exposures, although they actually look at occupation, in cytogenetically defined subsets.

So they were using cytogenetics to define the types of AML that they were interested in. Here's the 15;17. So this is specifically relevant to APL. And in their study, they didn't find any cases in their exposed group.

Q. Okay.

A. Now, it's a small study -- all of these are very small studies, but it certainly does not support that there was an increase based on their occupational exposures. And that was very wide. As I recall, there were solvents, but there were lots of

other things that they were potentially exposed to as well.

Q. And what is this rel -- what is the relevance to this study to Mr. Wolfe's claim that toluene, xylene, and petroleum distillates with naturally occurring trace benzene caused him to develop APL?

A. That there were no cases of people that had any kind of occupational exposure to APL. So this does not support that APL was a disease that was associated with occupational exposures.

Q. Okay.

MR. SCHIRRMEISTER: Offer Exhibits 18 -- let's see, was that 18 and 19? What were the last two that I handed you?

THE DEPONENT: 18 and 9 -- 17.

MR. SCHIRRMEISTER: 17.

THE DEPONENT: 17 and 18.

MR. SCHIRRMEISTER: Offer 17 and 18.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I'm going to hand you what's been marked as Exhibits 19 and 20, Fagioli, 1991 articles. If you would take a look at those, sir.

A. Thanks.

Q. Okay. What is the title of this article

and what were the authors attempting to do?

A. The title is "Distinct cytogenetic and Clinicopathologic Features in Acute Myeloid Leukemia After Occupational Exposures to Pesticides and Organic Solvents."

So this is directly relevant to the question that we are asking, whether or not exposure to organic solvents causes AML or a specific subtype of AML, in this case, APL. And what they concluded and was in their abstract is, whereas the classic AML-specific translocations -- those are the ones that you classically see in de novo or new leukemias that have no occupation, i.e., the 15;17. So this is the APL. They were detected only in unexposed subjects. So they did not see any people with the 15;17 in the exposed -- people exposed to pesticides or organic solvents. There weren't any. So they couldn't do any statistical analysis of them.

Q. Okay. And what is the relevance of these authors' conclusions with regard to Mr. Wolfe's claims in this case?

A. The -- this -- these conclusions in this study does not support that occupational exposure to organic solvents increases the risk or increases the number of people that have acute promyelocytic

leukemia and express this 15;17 translocation.

MR. SCHIRRMEISTER: I'll offer Defendants' 19 and 20.

Q. (BY MR. SCHIRRMEISTER) David, I'm going to hand you what's been marked as Exhibits 21 and 22, sir.

A. Okay.

Q. Would you identify this particular study for the jury and explain its significance.

A. This study came out of China. And this is a certified translation from the National Investigative Group for the Survey of Leukemia and Aplastic Anemia. And what they did was a countrywide analysis of various types of AML, various sub -- various types of leukemia as well as some subtype analysis of AML and aplastic anemia. They looked at a variety of agents. And this was -- the data that they presented, the little asterisk here means that it achieved statistical significance. We'll discuss that in a minute; but that means that it, you know, is likely a real association.

They found an association between benzene and M2. They did not find an association between benzene and M3. So this study -- and it's a really large study. They looked at over -- it was over a

million people. They looked at a 1,200 cases of leukemia. So it was way bigger than all of these others combined. But they did not find a statistically significant excess of M3s, acute promyelocytic leukemia, in these Chinese populations associated with benzene exposure.

Q. Okay. And M3 is APL?

A. M3 is APL. That is the same -- that's the FAB classification system for it. So this stands really in direct contradiction to the allegations in this case, that benzene can cause APL.

Q. Okay. To your knowledge, is this the only study anywhere that has specifically examined benzene and increased risk of APL?

A. It's the only one that I know of that has -- has done it to this level of specificity. I mean, all of those other studies with organic solvents and this, that, and the other, they are going to have some benzene in there. But this one specified benzene as a chemical -- a specific chemical and looked at the relationship between benzene and APL.

Q. Okay.

A. There has -- there's not another one that I am aware of.

Q. Okay. And what is the significance of

the results of this study for Mr. Wolfe's claims in this case?

A. It -- it does not support the position that exposure to benzene is a risk factor for the development of this particular subtype of APL. And it did have the statistical power. It was a strong enough study to see statistical changes. So it did find a risk with M2s, but it did not find one with M3. So you can't really look at this and argue that this study isn't big enough and it didn't have the power to -- to measure that association.

Q. Okay.

MR. SCHIRRMEISTER: We're going to offer Exhibit -- Defendants' Exhibits 21 and 22. And I think this would be a good time to take a break.

MR. GIANARIS: Okay.

MR. SCHIRRMEISTER: Thank you, sir.

THE VIDEOGRAPHER: Going off the record. The time is 11:02.

(Recess taken, 11:02 a.m. to 11:18 a.m.)

THE VIDEOGRAPHER: We're back on the record. The time is 11:18.

Q. (BY MR. SCHIRRMEISTER) Okay. David, would you read your next opinion and briefly explain to the jury what you will be discussing --

MR. GIANARIS: Object to form.

Q. (BY MR. SCHIRRMEISTER) -- with this opinion?

A. This slide reads, "Mr. Wolfe was not exposed to enough benzene to develop any form of leukemia."

So, really, what we're going to talk about the -- well, the fact that everyone is exposed to some level of benzene, just in our normal environment. So that's one aspect. And the other aspect is that people have conducted quantitative epidemiology to see and in -- and investigate and evaluate how much benzene it takes before you start meaningfully increasing someone's risk of developing some form of leukemia. And we're going to put Mr. Wolfe's estimated exposures into context with that quantitative epidemiology.

Q. Okay. Now, would you explain -- first of all, I'm going to offer -- I'm going to hand you what has been marked as Defendants' Exhibit 23. Would you identify Exhibit 23, please.

A. This is a picture of North Carolina with all of the counties color-coded. And it really is just to illustrate to -- to the folks in this trial that there's benzene -- there's benzene everywhere.

We are all exposed to low levels of benzene just in the ambient environment.

Q. Okay.

A. So in this room, in that room, there's benzene in the air in the United States.

Q. Okay. So we're sitting in a conference room here today in Colorado. Are we breathing benzene?

A. We most certainly are.

Q. Okay. And the folks sitting in the Courthouse before Judge Craig in Forsythe County, are they breathing benzene?

A. They are.

Q. Okay. And what is the source for this data?

A. The source for this data is the USEPA.

Q. Okay. And specifically what program from the EPA there?

A. It looks like NATA, the National Scale Air Toxics Assessment.

Q. Okay.

A. I'm not familiar with what they do, but it came from USEPA.

Q. Okay. And describe for us the -- why are there some higher elevated benzene concentrations in

certain areas of North Carolina than others?

A. The -- the -- the background benzene in the atmosphere typically comes from vehicle exhaust, so you're going to find increases in the amount where there are major cities. So Charlotte, Asheville, Winston Salem, Greensboro, those are the ones that tend to have more benzene than more rural areas. And it doesn't really matter about these numbers, but none of these levels of benzene from high to low are thought to have any toxicological relevance. So it's just -- it's not to scare anyone, it's just to show people that we are all exposed to benzene every day.

MR. SCHIRRMEISTER: Offer Defendants' 23.

Q. (BY MR. SCHIRRMEISTER) David, I'm going to hand you what has been marked as Exhibit 24. And what is this slide and what's the relevance, please?

A. This is just a continuation of that concept and to show people that things that they are exposed to in their daily life, that their diet has benzene in it. Benzene is a ubiquitous chemical. It's a really simple aromatic ring, and you find it all over the place. You find it in drinking water, you find it in air, you find it in bananas, you find it in cake, pizza, fried chicken, et cetera. So no matter what it is, what kind of diet you're on, there

is some low level of benzene that are -- are in those products that you're eating.

Q. Okay. And what is the source for this data?

A. This is from FDA, the Food and Drug Administration.

Q. Okay.

MR. SCHIRRMEISTER: I'll tender Exhibit No. -- offer Exhibit 24.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I'm going to hand you what's been marked as Exhibit 25. Would you take a look at that, please. And explain to us dose response, and specifically with regard to the bullet points on Exhibit 25.

MR. GIANARIS: Objection, leading.

A. A dose response, it's not magical; but it is the most important concept in toxicology. And I will say, whether I'm teaching toxicology to medical students, to physicians, to graduate students, it is the very first thing that I teach them. And it is the first thing on the first day; and at the end of the course, it is the thing that I hope that they remember the most. And that is that there has to be a meaningful relationship between the dose and the response. And if you increase the dose, if a person's

exposure increases, then the response needs to change according to that change in dose.

So that's -- there has to be a connection between the dose and the response. It doesn't always have to go up. Sometimes it can go down. It can be quite complicated, but there has to be a connection. If you change the dose and the response does not change, then probably, from a scientific point of view, you are not looking at what you think you're looking at, and the response is related to something else other than the dose. That's why toxicologists are so keenly interested in this notion of a dose response relationship.

Q. (BY MR. SCHIRRMEISTER) Okay. I'm going to offer 25 and hand you what has been marked as Exhibit 26. It's a dose response chart. Would you explain that to the jury, please.

A. So this is the simplist dose response relationship that we could come up with. And down here, you see the dose is increasing. And then whatever you're defining as the response is on the Y axis here, and so that's going up. And it's just showing you that as the dose increases, you're starting to see more of a response.

So down here, you don't have an effect.

So the dose is too low for there to be an effect. At this dose, you start seeing a slight effect, a moderate effect, a more serious effect. And then if it's a really toxic chemical or exposure, you know, perhaps it goes all the way to death of the animal.

So this is the simplest kind of dose response; but it illustrates the point, you know, to the jury, or to whoever is looking at this, that as the dose is changing, the response is changing along with it.

MR. SCHIRRMEISTER: Okay. I'll offer 26 and hand you 27, David.

Q. (BY MR. SCHIRRMEISTER) Now, this is called Doses of Common Substances, a typical dose and a toxic dose. And if you would explain this to the jury; and then, secondly, explain the significance with regard to the dose response concept.

A. Really, this -- this is -- when you talk about a dose response, that means that it takes a certain amount of the chemical to be toxic. So even things like benzene, if you're exposed to low enough levels of benzene, there's no evidence that it's toxic. And even really common, what we consider to be safe substances, like water, sugar, salt, if you're exposed to too much of those things, then you can get

toxicity.

So as Paracelsus said -- and we kind of skipped that, but that's fine. He was an early toxicologist. He said probably the most famous quote in this field is that the dose makes the poison. So you can't say that a chemical is toxic unless you also understand what the exposures are, unless you also understand what the doses of that chemical that you're talking about.

So a quart of the water is not toxic. Seven-and-a-half gallons is typically lethal. It messes up the electrolytes and causes cardiovascular problems. So everyone has heard of people that drink too much Gatorade and potentially died from it or, you know, got really sick because of it.

So that's what this slide is showing you, that a little bit of salt is okay, too much salt is toxic. So it's -- it's a concept that most everyone is familiar with.

Q. Okay. And does this concept of dose response also apply to benzene?

A. Of course, it does. That's -- I mean, the reason why we had those slides earlier was to show people that we are all exposed to benzene every day of our life all of the time. And there is no adverse

effects associated with those exposures. So those are below a point where anyone is going to think that there is adverse toxicity associated with it.

Q. Okay. And if a plaintiff's expert were to testify that dose does -- dose of benzene -- dose not matter, would you disagree with that?

A. I -- I would as well as the scientific world would disagree with that statement.

MR. SCHIRRMEISTER: Offer 27.

Q. (BY MR. SCHIRRMEISTER) I'm going to hand you what has been marked as Exhibit 28. David, this is a slide called Dose Response Curve For Aspirin. Would you explain this to the jury, please.

A. Sure. This is just a little more complicated verse, but down here is the dose, over here is the response. This one is a little different because it makes a change or it delineates the difference between a beneficial effect and a toxic effect. And this dotted line here means that doses below this line actually have a beneficial effect; doses above this line, that's where you start seeing toxicity.

So this red line here is the -- the dose response curve. And we know from epidemiology that low doses of aspirin are beneficial in that you

actually have a decreased risk of certain cardiovascular problems. But if you start taking too much aspirin, then you start getting GI bleeding and other GI problems. So that's what this line is depicting. But this is a standard kind of dose response.

Now, this red line is actually supposed to be touching there. And the idea is that that would be called a threshold. And that is a dose below which you really don't see anything going on.

So at doses of aspirin below the threshold, you don't have a beneficial effect, you don't have a toxic effect, you can't tell that a person has been exposed to those levels of aspirin.

MR. SCHIRRMEISTER: Okay. I'll offer 28.

Q. (BY MR. SCHIRRMEISTER) David, the next exhibit I'm going to hand you is 29 titled Dose Response Curve For Alcohol. And would you, again, explain this to the jury.

A. This -- we put this in here -- I have put this in here just because this is something that most people are going to be either firsthand or secondhand familiar with. And that is that if you increase the number of -- the amount of alcohol, ethanol that a person is being exposed to, then you have a dose

response relationship. And a little bit of wine or a little bit of alcohol, you get a giddy feeling. Then you start becoming impaired from a driving point of view. And, you know, you could argue about where those are going to be. But as that increases, blurry vision, alcohol poisoning, coma, and death.

So it just, again, depicts this notion of a dose response. And that is what -- if you were going to ask someone or someone was going to ask you, Well, what will alcohol do to me? Will alcohol make me sick or will alcohol have some effect on me? Your question is -- your -- your answer is going to have to be, well, it depends on how much you're exposed to. If you're only exposed to a drop of alcohol, it's probably not going to do anything to you. If you drink two fifths of whiskey, you know, you're probably going to be up here in this category if you do that in a 24-hour period. So that's -- that's the purpose of this slide.

MR. SCHIRRMEISTER: Okay. Offer 29.

Q. (BY MR. SCHIRRMEISTER) Okay. David, the next I'm going to hand you is Exhibit 30. And what I'd like you to do is explain to the jury, please, the dose response concept that you just discussed and the quantitative measurement of benzene, number one. And

then, secondly, I'm going to ask you once you've done that to discuss the levels of permissible exposure limits set by OSHA --

A. Okay.

Q. -- for workplace exposure to benzene.

A. Yeah. Probably the easiest things is to just kind of go through the slide. But benzene exposures are described in the literature and probably in this case, when it's actually happening, in terms that not every one are familiar with. And the terms that epidemiologists and benzene toxicologists and people use to define or describe benzene exposure is this concept of a part per million year or a ppm year. And this really is not a -- a conceptually difficult concept. The part per million is a concentration.

What that means, we don't need to get into, but a ppm is a part per million and is a way of measuring how much benzene is in the air. Okay. It could also be in water and other things, but for our purpose, it's in the air. So a part per million tells you the concentration in the air. And then the time or the duration, the years, tells you how long you've been exposed to that concentration in the air. So that's what a -- a ppm is.

Q. Okay.

A. All right. And then the second part talks about what the Occupational Safety and Health Administration -- what they regulate as a permissible exposure to benzene. And they call that a PEL or a permissible exposure level. And --

Q. Okay. And -- and is that PEL set by OSHA?

A. That's correct.

Q. Okay.

A. Right. And that is a legally enforceable standard. So OSHA says this is -- the most that you can have a person occupationally be exposed to is the permissible exposure limit. And they do it over a time-weighted average, which is how much they are exposed to averaged out over an eight hour. And the permissible exposure limit is 1 PEL.

So it is one part per million benzene averaged out over -- that's what this TWA, time-weighted average -- averaged out over eight hours -- that's the permissible exposure level.

Q. Okay. And is this the permissible exposure limit that was in effect all during the time that Mr. Wolfe worked as a painter?

MR. GIANARIS: Object to foundation.

A. I don't recall exactly when the 1 ppm

became -- I think that was in '86. I don't -- he might have started working in 1985, is my recall. So I'm not positive about that.

Q. (BY MR. SCHIRRMEISTER) Okay. But at least from -- from 1986 forward, you understood it to be one part per million?

A. Correct.

Q. Okay.

A. Correct.

Q. So let's go to the next bullet point, average of 8 hour TWA over year is a ppm year, which would explain that concept.

A. Right. So that's just what we were talking about. Up here where this is kind of the metric, the way that people talk about benzene exposures. And the jury and the court, they are going to hear about this. You know, for as long as this goes on, people are going to be talking about part per million years. So a -- one part per million year is -- this is one way to get to it, is a 1 ppm exposure.

And keep in mind that that is an eight-hour time-weighted average, so it's not, I was exposed to 1 ppm for two seconds during the day. That's not going to be that much exposure. It's 1 ppm averaged out over an entire working day, eight-hour

working day for one year. That is a 1 ppm year.

Here is an example of a much higher exposure where the individual was exposed to 10 part per million. Again, that's not a two-second peak of 10 ppm. That averaged out over an eight-hour working day. And they maintained that exposure for 20 years. All right. So that's much higher exposures, and that would end up giving you a 200 ppm year cumulative exposure to benzene.

Q. Okay.

A. This bullet here is what, basically, OSHA will allow in United States, because they allow an eight-hour time-weighted average of 1 ppm. And that's the permissible exposure limit. And the -- the estimated working lifetime is 40 years. So they regulate benzene as if a 40 ppm year is an occupational safe level.

Q. Okay. Now, this 40 part per million years, is this benzene exposure measurement also used in epidemiology?

A. It is.

Q. Okay.

A. It is. It's probably the most common metric measurement of benzene exposure that you will find in that the epidemiology -- particular the

epidemiology that is quantitative. And by that -- and we'll see that in a second, but there's a lot of epidemiology that doesn't really describe people's exposures very well. And they are less useful when it comes to trying to understand whether exposures cause problems because of the dose response that we just talked about. The quantitative epidemiology that is in the literature with regard to benzene will almost all use this metric of ppm years.

Q. And that's the same metric that the U.S. government uses?

A. That's the same -- yes. Yeah.

Q. Okay. And what does the U.S. government establish as a safe likely work life for benzene exposure?

MR. GIANARIS: Object to the foundation.

A. Well, they base -- since the permissible exposure limit is 1 ppm over a 40-year life span or 40-year working life, then that ends up being 40 ppm years.

Q. (BY MR. SCHIRRMEISTER) Okay. All right.

MR. SCHIRRMEISTER: Offer Defendants' No. 30.

Q. (BY MR. SCHIRRMEISTER) Now, David, the next slide, I'll hand you Exhibit 31. Now, first of

all, tell us -- read the title and explain to us what the -- what the title means.

A. Benzene and acute myeloid leukemia: Dose Response, Quantitative Studies of Benzene and Leukemia. So in the world's literature, these are the studies that have actually gone and said how much were people being exposed to benzene and what was their risk of leukemia or AML associated with those exposures.

And that's what the values in yellow represent. Those are the exposures that were reported in this quantitative literature.

Q. Okay. Now, the first one -- I see the first two is called pliofilm. What is the pliofilm study and how was that used by the U.S. government in establishing a safe level of occupational exposure to benzene in the workplace?

MR. GIANARIS: Object to the foundation.

A. Pliofilm is a nickname given to rubber hydrochloride. It was like a -- a cellophane-like material that they made in three facilities, Goodyear facilities in Ohio. And the workers that made this pliofilm, they were using pure benzene as a solvent. So they were dissolving this material in pure benzene, they were spreading this material out on big drums,

they were collecting the benzene above the facility.

So these workers were exposed to pure benzene in -- in, by today's standards, really high concentrations. And the workers that were making this pliofilm at certain exposures did have an increased risk of developing leukemia.

Since there was a dose response that was evident, since these workers were not exposed to lots of other chemicals that could confuse the analysis, and since we really kind of had pretty good ideas about what these workers were doing, how long they were working there, that kind of thing, then the pliofilm cohort and the various analysis thereof -- and there's been probably 13 studies or 14 studies published on this -- this cohort, that forms the basis for all of our regulatory standards. In the United States, that forms the -- the regulatory basis for our understanding of benzene and leukemia.

Q. Okay.

A. EPA uses it, OSHA uses it, ACGIH uses it. Everyone uses the pliofilm today.

Q. Okay.

MR. GIANARIS: Object to the form. Just let me get it out before so I don't talk on you. Object to the form, lack of foundation, move to strike

for lack of foundation, nonresponsive.

Q. (BY MR. SCHIRRMEISTER) And the -- the first two studies, pliofilm, are those studies of that pliofilm group from the Goodyear plants in Ohio?

A. Correct. The first one that Bob Rinksy -- well, this actually wasn't the first one, but the one that Dr. Rinsky published in 1987, they used all leukemias combined.

There was a case of CML, there was a case of ALL. And then Dr. Otto Wong in 1995 did a subsequent analysis of this cohort, this group of individuals that worked at these plants. And he reevaluated only based on AML. So that's why those are two different studies. But, you know, the date is pretty similar. And they really were looking at the same workers.

Q. Okay. And just so we're clear, there were no APLs in any of these cases, correct?

A. You -- I can't say that. I don't know. They didn't evaluate APLs.

Q. None were identified on these studies?

A. That's right. They didn't look.

Q. Okay. So the -- then -- then -- so what is -- when the first two pliofilm -- significant excess at greater than 200 part per million years in

those first two, what does that mean? Explain that to the jury.

A. The significant excess -- and we'll talk about this a little later, is -- from an epidemiological point of view, it means that the excess was statistically significant, which means you can be reasonably confident that the excesses that you saw were, in fact, related to the exposures and not just random chance.

So statistical significance is a relatively important aspect of epidemiological studies and the evaluation of that data, because it allows you to rule out the role that chance could play. And in order to achieve a statistically significant excess, it took 200 part per million years of benzene exposure in these workers.

Q. The next one is NCI China. What is that?

A. That was a really large study that the National Cancer Institute did over about a 20-year period with the Chinese Academy of Preventative Medicine. They looked at some 78,000 workers in China employed in a variety of different occupations. They were shoe workers and -- and rubber workers and petroleum workers and painters, and all kinds of different things. And they then reported out a

collective cumulative sort of analysis of the benzene exposures. And in that analysis, they saw a statistically significant increase at something greater than 40 part per million years.

Q. Next one, Glass, Health Watch?

A. Deborah Glass published a paper in 2003, which was the latest iteration. Health Watch is a series of industry surveys that the Australian Petroleum Institute funded through Monash University and some others. And they basically followed these refinery workers and petrochemical workers that were employed in the petrochemical industry in Australia. And she reported that there was an increase -- significant increase in acute non-lymphocytic leukemia, which is AML, essentially -- at something greater than 8 ppm, but it wasn't any higher than 57 part per million years.

Q. Okay. Next one is the shoe worker study.

A. Costantini did a -- looked at Italian shoe workers and reported a consistent finding with the pliofilm that it took more than 200 p -- I think it was actually 199. It had to be greater than 199 ppm years.

Q. Okay. And then the three find no stat -- no significant access at different levels. Explain

those three to us.

A. Dr. Bloeman was doing a Dow Chemical worker study. And he had exposures -- he had workers in his study that were exposed to benzene concentrations at greater than 79.1 part per million years. And he did not report a statistically significant excess.

So we don't know how far it has to go before you would get one. All we know is that in his study, you know, basically 80 part per million years was not associated with an increased risk. And Rob Schnatter, Dr. Schnatter and Leslie Rushton, these two studies are very similar. Rob Schnatter did look at refinery and petroleum workers in Canada. Leslie Rushton looked at refinery and petroleum workers in the UK. And they both had an exposure category that was something greater than 45 ppm years. But in those highest-exposed individuals, they did not see a statistically significant increase, in -- in this case, leukemia.

Q. Okay. Now, David, on Exhibit 31, are these all the studies that exist that quantitatively analyze benzene exposure and potential for increased risk of leukemia?

A. The only one that I can think of that we

didn't put on here was a previous study to Bloeman and it was by James Collins. And he had an exposure -- it was pretty low. He had an exposure of 6 ppm years, but there was number of statistically significant increase.

So other than that one, if you slide that one in here -- and, basically, he didn't see anything -- that is it for the quantification of benzene exposure and AML risk that -- that I have seen.

Q. Okay. And how does OSHA use this data that is in the quantitative studies?

MR. GIANARIS: Object to the form, foundation.

A. My understanding is that -- I mean, at least based on what they say in their documentation is they are basing their dose response analysis on, not Wong or Bob Rinsky, but what we don't have on here. There -- there have been three separate exposure calculations done on these workers.

I actually -- Bob Rinsky did do one, I take that back. His '87 paper -- there were two others that have tried to get their arms around what these pliofilm workers were actually exposed to. EPA, OSHA, all of these other regulatory agencies used

those three quantifications or those three estimates of their exposures to set these standards.

Q. (BY MR. SCHIRRMEISTER) Okay. And is the standard they set the one that we looked at earlier of a likely work life of 40 part per million years?

A. Well, they set a standard of 1 part per million year, which would equate to 40 over a normal working lifetime.

MR. SCHIRRMEISTER: Okay. I'm going to offer into evidence Defendants' 31.

Q. (BY MR. SCHIRRMEISTER) And, David, I hand you Exhibit 32. Okay. What have we done in Exhibit 32?

A. This --

Q. There are four columns.

A. Right. This was just to depict these kind of graphically in a -- in a simple histogram, depict these various occupational levels. So here's what we're talking about with OSHA. So that's 40 ppm years. Here's the Rinsky and the Wong. So all of the other ones would be on here somewhere. And this little red box up here illustrates that it takes exposures greater than that before you start seeing a statistically significant increase in -- well, in this case, it's leukemia; and in this case, it's AML. And

then this little line here, which you can barely see, little tiny yellow line there, that's based on a quantification estimation of Mr. Wolfe's benzene exposure during his employment as a painter that was done by Brad Russell. And his cumulative exposure as still made by Brad Russell was .093 ppm years.

MR. GIANARIS: Just I want to make an objection, because I think the question went -- the answer went beyond the question, so I didn't have time to --

THE DEPONENT: Sorry.

MR. GIANARIS: -- inter -- interject the objection before the question. But this goes beyond the scope of his disclosure, beyond the scope of his report, and lacks foundation as to Mr. Wolfe's exposure.

Q. (BY MR. SCHIRRMEISTER) Okay. So let's -- let's go through OSHA -- this one here, 40 part per million. What does that represent?

A. What this -- this box represents the 40 part per million years. It's what OSHA allows in the occupational workplace if a person is exposed at the PEL, which is 1 ppm over a 40-year working life. These other two boxes are where Dr. Rinsky and Dr. Wong calculated a statistically significant

increase in either leukemia or AML.

Q. Okay. Now, is there any literature, David, anywhere in the world that you're aware of that finds an increased risk of leukemia when a person is exposed to .093 part per million years?

A. That's -- no. No. That's not any different, really, than a person's typical background exposures would be through their life. But, no, there is no evidence that that increases a person's risk of developing leukemia generally, AML or APL specifically.

MR. SCHIRRMEISTER: Okay. Offer Exhibit 40 -- 32.

Q. (BY MR. SCHIRRMEISTER) Okay. Now we're going to move on to your next opinion in this case, David. Would you read page 35 into the record, please.

MR. GIANARIS: Objection, leading.

A. Do you want me to just read this?

Q. (BY MR. SCHIRRMEISTER) Yes, sir.

A. "Painters as an occupational group are not at an increased risk of developing leukemia."

Q. Okay. I'm going to hand you what has been marked as Exhibit 33. And I think it might be easiest for you to stand David, if you want to.

A. Sure.
Q. Explain some of these concepts to the jury.
A. Well, what we needed to do in order for the jury to really have some, you know, basis for interpreting the literature, the literature being the epidemiology studies that have looked at the occupational risks associated with being a painter -- and as you're going to see in these subsequent slides, there is a great deal of literature on that topic. Lots and lots of people have reported on the occupational risks associated with being a painter.
Q. Okay. Now --
A. But in order to do that, we needed to provide at least a very superficial background on epidemiology.
Q. So do you understand that Dr. Milman has testified that a dose does not matter because Mr. Wolfe was a painter, and there are studies that say painters get leukemia?
A. That flies in the face of every toxicological principle that exists.
Q. Okay.
A. We're all exposed to benzene, painters are exposed to benzene, I'm exposed to benzene. You

have to quantitate the dose. You have to understand what the exposures were before you can say that there's an increased risk of -- of even developing the disease. And then it's another whole step to say that this person's disease was actually caused by those exposures. But, no, I don't -- I do not agree with that.

Q. Okay. Let's look at Exhibit 33, the first one, and explain -- explain epidemiology and what you're showing to the jury here, please.

A. So really epidemiology, in the simplest terms, is the study of diseases in the human population. So it's the study of the distribution of disease and those causes, whether they are exposures, or whatever else, in the human population.

So the purpose of all epidemiology is to understand the cause of disease in humans. And this is just to show here's a population, and those two individuals have whatever the disease is that -- that you're interested in looking for.

MR. SCHIRRMEISTER: Okay. Offer 33.

A. So these are very superficial in terms of their explanation.

Q. (BY MR. SCHIRRMEISTER) Okay. Now, David, I'm going to hand you Exhibit 34, which is one,

two, three, four, five, six slides. So let's go through these. The first slide is called Cohort Studies. Explain what you're showing here to the jury. And before you do this, David, set the basis for why these concepts are important for the jury to understand when you will show them, in a few minutes, paint slides or graphs from painter epidemiology studies.

A. Okay.

Q. That -- I'm sorry, that's a poor question.

A. No, I got it.

Q. But I think you understand what I'm asking.

A. Yeah. I mean, as I said earlier, these are just to give the court, the jury, whoever is watching this -- just to give them a really basic understanding of the science of epidemiology so that when we start looking at data in these epidemiological studies that they have the tools that they need to be able to interpret and to understand what those studies are really telling them. And it's not that hard. It's not magic, and it's not -- you don't have to have a Ph.D. in epidemiology to be able to look at the studies and understand what that data tells you.

Having a Ph.D. epidemiology is important if you're going to be doing the studies, but in terms of interpreting them, we can all do that.

Q. Okay. And how do you use epidemiology in your work?

A. I use epidemiology every day. I mean, I am evaluating human studies in any of the toxicological evaluations that I do for -- for any of my clients with regard to -- because it's human data.

So you never have to worry about the relevance of what this data means to people. When you're looking at toxicological studies that have used an inbred strain of mouse, while it's interesting and it may give you some information on mechanism, but you always have to ask yourself, Are we like that mouse? Are we likely to get the same diseases that that mouse is going to get? So you're always questioning the relevance of that mouse data to humans. From an epidemiological point of view, you've overcome that hurdle. And that's a very big hurdle toxicologically. This data is relevant because we are humans.

Q. Okay. And do you teach epidemiology as well?

A. I do. I teach environmental epidemiology class. I teach it with another person.

Q. Okay. Now, the first -- the first type of epidemiology study is a cohort. What is a cohort study?

A. Yeah, there are two -- there are two main kinds of epidemiological analysis, and they are pretty different in their design. And each -- all of these studies have pros and cons, they have strengths, they have weaknesses. The devil is in the details with an epidemiology study, so they are not easy to conduct. But the two basic kinds are cohort studies and case controls. And cohort really just means group. So a cohort is a group of people.

So when you're doing a cohort study, you're looking at a group of people that had some exposure, and you're comparing them to a group of people that did not have that exposure. And so by comparing these exposed people to what happened to these unexposed people, they will allow you to start making some statements about whether or not their -- that exposure had an affect on them.

Q. Okay. Now, what are we seeing here in the folks that are highlighted?

A. Okay. So this is exactly what I talked about. The ones that are highlighted, they would be the individuals that had the disease. So you're

looking at this cohort. This is my exposed cohort. And we found two individuals that had the disease, kidney cancer or -- or bladder cancer or lung cancer -- whatever it is that you're looking at. Cardiovascular disease. And then these were our nonexposed -- our nonexposed group. And the comparison here is only fair if this nonexposed group is chosen very carefully; but, you know, that's more than we need to talk about here. But, basically, you also see two individuals that have the disease in the nonexposed. So you have two in the exposed, two in the nonexposed.

Q. Okay. And when you --

THE VIDEOGRAPHER: Counsel, five minutes until tape change.

Q. (BY MR. SCHIRRMEISTER) Okay. When we look at this, what -- and this has cohort studies, three possibilities. Explain what the three possibilities are in a cohort study.

A. Sure. When you're making those comparisons -- so you're comparing the exposed group with the unexposed group, right? That's really what you do on a cohort. No matter how sophisticated they seem, that basic thing is happening.

You can either have one possibility that

it's the same. So you have two cases in the exposed. You have two cases in the unexposed. So the incidence of disease, the rate of disease is the same.

Q. Okay.

A. You can also -- in the exposed group, you have four cases here, compared to only two in the unexposed. And this is sometimes called the control group.

Q. Okay.

A. You only see two in the control group. So in that case, you see more cases of disease in the exposed than you see in the unexposed.

Q. Okay.

A. So something like this, you would start to think, well, maybe the exposures are having something to do with this increased incidence of disease.

Q. And what's a good example that we all know -- I think we all know about or we do know about with exposed folks getting more of a disease than unexposed people?

MR. GIANARIS: Objection, leading.

A. Yeah, there's lots of examples, but probably the easiest one that I think everyone would have heard about would be lung cancer in cigarette

smoking. And that one is pretty -- pretty unequivocal that people that are exposed to cigarette smoking, because they are smoking all the time, have a higher incidence of lung cancer than a controlled population does.

Q. Okay. And what is group No. 3?

A. And the other -- the third possibility is that when you look at your exposed cohort and you compare it to your control or your unexposed cohort, you actually see fewer cases in the exposed group than you see in the unexposed group. So here you see two cases of the disease and here you see four cases of the disease.

Q. Okay.

A. So you certainly wouldn't say that the exposure had an -- you know, influenced or had an increase on the risk. You may even say perhaps this exposure is somehow protecting these individuals from getting whatever this disease is.

Q. And what would be an example of that?

A. Probably an example would be what I showed with the aspirin where people that take an aspirin a day actually have a lower incidence of developing cardiovascular disease. So --

Q. Excuse me.

A. That's fine. If you're looking at cardiovascular disease, then the control people, these people are not taking aspirin. They have a higher rate than the people who are taking aspirin. And that is the basis for us being able to say that taking an aspirin is beneficial to your health, because they aren't developing that disease as much as the controls.

MR. SCHIRRMEISTER: Okay. I think we have to change the tape, David.

THE VIDEOGRAPHER: This is the end of tape No. 1. Going off the record. The time is 11:58.

(Recess taken, 11:58 a.m. to 12:11 p.m.)

THE VIDEOGRAPHER: We are back on the record. The time is 12:11. This is the beginning of tape No. 2.

Q. (BY MR. SCHIRRMEISTER) All right, David. We had just finished discussing this slide, page 39 of Exhibit 34. So go on to the next page. And explain here no association in this example of a cohort study.

A. Yeah, really the only point out of this slide, because this is the first example where the number of cases in the exposed was similar to the same as the number of cases in the unexposed. And then basically in epidemiological studies, you set up a

ratio. So you put this number over this number. And the math is not that important; but if that ratio is 1, that means that the top number and the bottom number are the same, that means that the number of cases in the exposed was the same as the number of case in the unexposed.

So by definition, if you see a relative risk, however that's defined, but that's a ratio that's describing the risk of getting this disease being exposed to whatever it is of 1.0, then there's no association.

Q. Okay. So if when we -- when the jury looks later at different epidemiology studies, if they see 1, there's no association?

A. If they see 1.0, that means that in the exposed group, they saw the same number of cases -- exactly the same number of cases as they saw in the unexposed group.

Q. Okay. Next slide is cohort studies with a positive association. Explain that, please, David.

A. Right. So it's the same -- it's a ratio of how many you saw in the exposed, in this case 8, versus how many you saw in the unexposed, in this case 2; but you set it up as a ratio. Doesn't matter how we got that. But in this case, the ratio is 4.0. So

that means that it was four times more likely in the exposed to find people with the disease than you would find in the unexposed.

So that would suggest that there might be a relationship between whatever this chemical was and this disease. So this is a positive association 4.0.

Q. And is that, for example, smokers more likely to get lung cancer than nonsmokers?

A. I think in that case when it's lung cancers and cigarette smoking, this relative risk is, like, 20 or 30. It's very high.

Q. But that speaks to a positive association?

A. That's correct.

Q. Okay. And then the next is cohort studies, no association in this example.

A. Right. This is the third case where you actually see fewer number of cases in the exposed compared to the unexposed. And so the ratio is going to be less than 1.

In this case, the ratio was .25, which means that you have a reduced chance of finding people with the disease associated with the exposure than you have in the control group.

Q. Okay.

A. So this would not indicate an association between exposure and disease, and could be used to support the position that these exposures actually protected the individuals from developing that disease, like we talked about with cardiovascular and aspirin.

MR. SCHIRRMEISTER: Okay. Offer 34.

Q. (BY MR. SCHIRRMEISTER) David, I'm going to hand you what's been marked as Exhibit 35, which is a two-page slide called Case Control Studies.

Now, how is a case control study different from a cohort study?

A. In a case control study, you basically -- and you use these if the disease is relatively rare. And there are other characteristics of these types of studies. But basically, you get a group of individuals that have the disease that you're interested in.

So you look at a bunch of people that have kidney cancer and you call those the cases. And then you get a group of individuals that you call the controls, and these individuals should be as close to these people as possible, with the exception that they don't have kidney cancer.

So they are the same age, same gender,

ethnicity, occupation, all of those things are going to be the same, except that they do not have -- they do not have kidney cancer. And then through one mechanism or another, you ask these people on interview, questionnaires, other methodologies -- you ask these people, Were you exposed to this chemical? And you ask these people, Were you exposed to this chemical? And then you look at the ratio of how many of these people that said, Yes, I use that, or, Yes, I worked with that, compared to this. So that is a case control study.

Q. Okay. And then this is a -- a slide that demonstrates a 2.5-fold association in a case control study. Would you explain this, David.

A. Right. Really, it's the same -- the ratio is the same. So this ratio is how many people responded yes, Yes, we were exposed in the -- in the cases versus how many people responded yes in the controls. And you set it up as a ratio just like you do with the cohort studies.

So in this case, the odds ratio is 2.5. An odds ratios are what you're calculating for case control studies. So this would suggest that there is an association. There is a 2.5-fold association in this example. This could be 1. And if it's 1.0, then

in this case control study, the same number of cases as the number of controls said, Yes, we were exposed to this or, Yes, we did this. And it can be less than 1.

MR. SCHIRRMEISTER: Okay. I'm going to offer 35.

Q. (BY MR. SCHIRRMEISTER) And hand what you has been mark as 36. This is Relative Risk and explaining the 1.0.

A. Okay. So on both cohort and case control, which we've just gone through, associations between exposure and diseases are measured by relative risk. And this relative risk can be called an odds ratio, it can be called a standardized mortality ratio. I mean, there's all kinds of ways, depending on specific that you're looking at. But essentially it's what we've been talking about. If it's 1.0, then that means that the controls and the exposed had the same probability or risk of developing the disease.

If it's greater than 1, then there were more people in the exposed that had the disease than than the unexposed, so that would suggest there might be an association. If it's less than 1, then there are fewer people in the exposed than you saw in the unexposed. And that suggests that there might be a

protective effect from that. So this is really just kind an overview of what we've been talking about.

MR. SCHIRRMEISTER: Okay. Offer 36.

Q. (BY MR. SCHIRRMEISTER) David, I'm going to hand you 37, which goes on to a slightly different topic, a two-page slide called Evaluating The Role Of Chance.

Now, the first there -- would you just read that to the jury and explain the P-value and the 95 percent confidence interval and explain how that is used in epidemiology studies, both cohort studies and case control studies.

A. Right. There's really no difference between those. All you really need to know about this slide is that when you're conducting an epidemiology study, whether it's a cohort or a case control, it's not a very precise science, and there is uncertainty about your answer. There is variability around that answer. And what you need to do, because of that variability, is you want to -- to the best of your ability scientifically, you want to be able to say this is a real certification and not due to just random chance. Not due to the fact that, you know, we just flipped a coin, and it ended up heads six times in a row. Okay. That there really is something going

on.

These two measures are ways that epidemiologists mathematically -- it allows them to account for the role that chance can play in their studies, the P-value and the 95 percent confidence interval. But really all you need to remember -- that's fine. All you need to remember is that it's ways that epidemiologists account for the role that chance could play in their findings.

MR. SCHIRRMEISTER: Okay. Offer 37.

Q. (BY MR. SCHIRRMEISTER) David, I'm going it hand you 38 and 39, which are two studies -- or two slides that speak to confidence intervals.

A. Might we go back. We didn't do the P value.

Q. I'm sorry.

A. So really, we'll just go through it. The P-value is the probability that you could have obtained the data that you got and the association that you calculated from that data by chance alone.

Q. Um-hum. All right.

A. Okay. So that's what a P-value does. So it tells you what's the probability that we really don't have an association at all but that random chance gave us the data that we're looking at.

So if the P-value is .01, then that means that we are 99 percent sure that chance did not give us the answer that the study reports. So that would be a very good study.

So we are only 1 percent chance -- there's only a 1 percent probability that chance is the reason why we got the results that we got. And by convention, scientifically, we say that of a P-value of .05 or less means that it is statistically significant, which means we are willing to accept -- the scientific community is willing to accept, all of us that, the EPA, the FDA with clinical trials, all of us, the scientific community is willing to accept a 5 percent chance that -- that just dumb luck is what we're looking at, but we are 95 percent sure that -- that -- that, really, we are looking at the real data.

Q. Okay. So now on to Exhibit 38 and 39 called a confidence interval. The first slide speaks to the president's approval rating or a president's approval rating. And then the second describes a relative risk. So would you explain Exhibit 38, please, David.

A. Yeah, a confidence interval is just a descriptor. And a lot of epidemiologists think that this is actually the most important part of the

epidemiology data, that the number, the point estimate doesn't tell you merely as much as the 95 percent confidence interval. But what that is is that when you calculate a relative risk, there's variability around that, and you don't know what the exact answer is.

So as an example, if they did a president's approval rating and some poll and they would say, yeah, their approval rating is 60 percent. Okay. That's pretty good. But they would also say, We don't know if it's exactly 60 percent. We say it's 60 percent, plus or minus 3 percent. So that plus or minus 3 percent is the confidence interval.

They say, We don't know that the confidence -- I mean, that the approval rating is actually 60, but we are pretty sure that it's somewhere between 57 and 63. Maybe it's 62, maybe it's 59. We don't know, but we are 95 percent sure -- if it's a 95 percent confidence interval, we're 95 percent sure that the real answer is from 63 to 57. That's very important in evaluating epidemiology studies.

Q. Okay. Now we have the next slide, which is Exhibit 39. And this is the 95 percent confidence interval.

A. So this is exactly what we were just describing in the previous study, but it's looking at real epidemiological data.

So you pick up a study and you show that the relative risk is 2.0, which means that there were more people in the exposed that had the disease than were in the unexposed. All right. So this ratio was greater than 1. But we don't know that the real answer is 2.0.

Okay. All we know is based on the size of the study and the -- and the conduct of the study that we are 95 percent sure that the real answer is within this range. And this range is the 95 percent confidence interval. In this case, it's 0.9 to 4.4.

So we don't know if the answer is 2.0, we don't know if the answer is 1.0, we don't know if the answer is 4.0. Okay. And that's really important, because if the answer is 4.0, that's a really different interpretation than if the answer is 1.0. 1.0, no association; 4.0, a positive association. So this 95 percent confidence interval really is critically important in evaluating this study.

Q. Okay. Now, we did this originally, David, to speak about painter epidemiology studies.

MR. SCHIRRMEISTER: Before we do that,

offer 38 and 39.

Q. (BY MR. SCHIRRMEISTER) I'm going to hand you what has been marked as Exhibit 40. Would you read the title of this study.

A. Studies show painters do not have an elevated risk of AML or leukemia.

Q. Okay. Now, what have you done here, David?

A. This slide just depicts -- and we separated it out, I separated in cohort, because now we know what those are, as opposed to case control studies. These registry based -- this is a different type of epidemiological study design, but it's looking at the same thing. These are all the studies that -- that I know of that specifically evaluated the relationship between leukemia and/or AML and occupation of being a painter.

Q. Okay.

A. And as you can see, there is a -- a lot of studies. This has been looked at from an epidemiological point of view for years.

Q. Okay. Now, Dr. Milman, one of Mr. Wolfe's experts, has testified that painters are at an increased risk for developing AML or leukemia. Do you believe the epidemiology literature as set

forth on Exhibit 40 supports that?

A. Well, anytime you ask this question over and over and over again, just by random chance, you're likely to find some positives. And there are a few positive studies scattered throughout the literature. So if all you were going to do is look through the literature and say, Well, here's a positive study or two, and that's what's going to be the basis of your opinion, you know, then -- then that's basically what you would say. But, you know, a fair interpretation is you've got to look at this overall literature. You can't just pick out the sporadic positives. You have got to look at the collective body of studies. And there's a huge amount of data out there. And collectively, this does not support that painters have an increased risk of developing AML.

Q. Okay. How many cohort studies were you able to identify?

A. I could find 19. 15 have some kind of positives, so 19 -- I'm sorry, 15 have some -- 4 have some positive finding, 15 are negative. So 15 out of 19 were negative. 14 out of 17 for the case controls. Almost all of the registry studies were negative.

It's important to point out that simply listing them and then counting up the number of

positives and negatives isn't really an appropriate way of evaluating this literature, because these studies are -- some of them are more important than others. Some of them are more powerful, some of them are bigger. And so, really, you -- you can't just, you know, go like this and say, Well, there's more papers on the positive side or the negative side. You've got to look at what these individual studies say.

MR. SCHIRRMEISTER: Okay. Offer 40.

Q. (BY MR. SCHIRRMEISTER) David, did you go into all of these different studies and select those that you thought were most important?

A. The ones that I picked out were the most recent and they were also the largest.

Q. Okay.

A. So from a size point of view --

Q. So --

A. -- the bigger the study is, the better able those investigators are going to be able to -- to find a -- a potential association.

Q. Okay. I'm going to hand you what has been marked as Exhibits 41 and 42. 41 is this slide. 42 is the underlying study. So describe this study. First of all, it's -- what type of study is it?

A. Okay. This was an epidemiological study that was conducted by Kyle Steenland and a co-investigator from NIOSH, government-sponsored study. And they were specifically looking at mortality associated with being a painter.

Q. Okay.

A. And they evaluated the mortality experience of 57,000 painters.

Q. Okay. And who -- and who sponsored this study?

A. This was NIOSH, the National Institute of Occupational Safety and Health. So this was done on behalf of OSHA.

Q. Okay. And you have a -- and how many painters did they look at?

A. They looked at 57,000. So this was a really, really big study. And the larger the study is, it allows them to be able to see potential changes that are small. The bigger the study, the better able the investigators to see if there are small increases in risk. And 57,000, that's the biggest one that I -- that I think is in existence right now on painters. And they did look specifically at leukemia. And here is their results from that leukemia.

Q. Okay. And what is the relative risk that

was found?

A. Yeah. So in this case, it was standardized mortality ratio, which is their form of a relative risk with the 95 percent confidence interval.

So, I mean, that's exactly what we were just talking about. Think found an overall relative risk of leukemia of .92. So that is actually less than 1. So it does not support that there was an association between being a painter and getting AML. The 95 percent confidence interval -- so this is really what we think the real answer is going to be. Somewhere between .78 and 1.1. We don't know what it is, but the overall risk does not suggest there's an increase. The overall study does not suggest there's an increase.

Q. Okay. And risk is actually below 1?

A. That's right.

Q. And the significance of that is?

A. Well, the significance is it clearly doesn't support an association. If you have a number that's below 1, then you could say, well, that suggests there's a protective effect. But I don't believe, unless it's statistically significant, you can really rely on that.

So here we have one that's 1.3. Well, I

don't believe that is meaningfully elevated any more than I believe this is more meaningfully depressed, because you don't have statistical significance associated with those. So while this is less than 1, my interpretation is that just says there's no association.

Q. Okay. And when you say no association, no association between what and what?

A. No association between being a painter and dying from leukemia, because this is a mortality study.

MR. SCHIRRMEISTER: Okay. Offer 41 and 42.

Q. (BY MR. SCHIRRMEISTER) Okay. The next one, David, is another study. And you selected the second study, correct?

A. Yes.

Q. Okay. What is this study? Who performed it? Who were the authors? What -- what did it do?

A. This was published in 2002. Dr. Brown, I don't -- I don't actually know -- the National Cancer Institute. Yep. So they were from NCI. So that's a federal agency, NCI.

Q. And, again, National Cancer Institute being the sponsoring organization?

A. Correct. Or where these people worked. And they looked at exposures into painting trades and paint manufacturing industry and risk of cancer among men and women in Sweden.

Q. Okay. How many painters?

A. And they looked at 49,000 painters. And in this case, they reported an SIR. The previous study was SMR for mortality. So they were looking at death certificates and how many people died. These folks were looking at medical records and how many people got the disease. So this is I for incidents, SIR. But it's a relative risk. Same concept.

Q. Okay. And what was the relative risk reported for leukemia and painters?

A. In this study, they look at leukemia collectively. They had 115. That's what the N means. That's how many cases they looked although. The SIR or the relative risk was exactly the same as the other one, .9, a little bit below 1. The 95 percent confidence interval was .8 to 1.1.

Q. Okay.

A. So this very large study from the NCI does not support that being a painter increases your risk of developing leukemia.

MR. SCHIRRMEISTER: Okay. Offer 43 and

44.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I'm going to now point you to Exhibit 45 and 46. Would you identify that for us, please.

A. Another recent study, 2002. This was a registry-based study, Association Of -- Swiss Cancer Registries.

Q. Okay.

A. Another really large one.

Q. How many painters?

A. 28 -- excuse me, 58,000 painters. And they were looking at -- so this is kind of a case control, which is why you have odds ratios. And this 043 at the beginning of the study, they listed all of the various occupations and assigned a numerical code to those occupations. And on 043 was the painter occupation.

Q. Okay.

A. That's not on the slide. You just have to take my word for it; but for the painters, for leukemia, the odds ratio was .7. So that's below 1. Here's the 95 percent confidence interval. And then for myeloid leukemia -- which is a little more relevant, because it includes chronic myelogenous, as well as acute myelogenous, but the relative risks

really don't change -- it's 0.6, and the 95 percent confidence interval is .6 to 1.2.

Q. Okay. And in each case, is the odds ratio below 1?

A. In these cases, they are below 1; but since it's not statistically significant to me, that really just says, in this study, they did not see an association or any hint of an association between being a painter and developing either leukemia or myeloid leukemia.

Q. Okay. So if we were to add the painters together in all three studies, we would have approximately how many painters?

A. I don't know, 150,000.

Q. Okay.

A. 160,000.

Q. Okay. And in those three studies, did any of the authors find that increased risk between being a painter and developing leukemia?

A. There were no increased risks of any of those three which were relatively recent large, large epidemiological studies, no.

Q. Okay. And do you believe that being a painter puts you occupationally at an increased risk for developing leukemia?

A. The only way you can answer that kind of question is by looking collectively at the epidemiology that investigates that question. And that's what we've been doing.

These three largest, but as well as the overall list that we showed earlier and collectively, no, this literature does not support that painters, as an occupational group, have an increased risk of developing leukemia.

MR. SCHIRRMEISTER: Okay. Offer 45 and 46.

Q. (BY MR. SCHIRRMEISTER) Okay. Now, David, we're going to go on to our final area here. Would you read that, please.

A. "The best available scientific literature does not support a link between formaldehyde and acute myeloid leukemia or acute promyelocytic leukemia."

Q. Okay. Now, I'm going to hand you what has been marked as Exhibit 47. Did you prepare a supplemental report in this case regarding formaldehyde and --

A. I did.

Q. -- AML or APL?

A. I did.

Q. Okay. And is Exhibit 47 a

true-and-correct copy of your supplemental report dated July 9, 2009?

A. It looks to be. I don't have any reason to think you would have changed it.

Q. No.

A. Looks right, yes.

Q. Okay.

MR. SCHIRRMEISTER: Offer 49. Excuse me, was that 47?

THE DEPONENT: It is 47.

MR. SCHIRRMEISTER: Okay. Offer 47.

MR. GIANARIS: Same objection.

Q. (BY MR. SCHIRRMEISTER) Okay. And just so we're clear, your -- Exhibit 47 is a true-and-correct copy of your report of your report -- of your supplemental report dated July 9, 2009?

A. Correct.

MR. SCHIRRMEISTER: Okay. Offer 47.

Q. (BY MR. SCHIRRMEISTER) Okay. Now, what did you do, David, to address the question of whether formaldehyde exposure places folks at an increased risk for developing leukemia or AML?

A. Well, really, in the report, I just summarized the literature. I -- I have done some work over the last two or three years interactive with EPA,

worked with some formaldehyde industries that use formaldehyde on the biological basis and whether or not the epidemiological literature supports that formaldehyde is a -- a cause of human leukemia.

So I knew what the literature was addressing this issue. So, really, all I did in my report was describe it in a way that I hoped was understandable.

Q. Okay. And did you select studies that you believed best addressed -- best addressed this question?

A. Yes. There's a lot of old literature that -- that people talk about. And I've read them all. But these are looking at workers that are embalmers, funeral directors, pathologists. And while they were exposed to formaldehyde, they were also exposed to lots of other things. And so there are some really small epidemiological studies that evaluated the leukemia risk in those groups. But then more recently, there have been really large cohort studies of workers who were exposed to formaldehyde in which they actually measured formaldehyde.

So there was a quantification of the exposures and not just, yes, he was an embalmer, we think he was exposed to formaldehyde. They have

actually done -- done the air monitoring and the exposures. And so that's what I made some slides of, those three big modern studies.

Q. Okay. And tell us what the first study is, David. I'm going to -- I'm going to hand you -- this is -- Exhibit 48 is the slide and Exhibit 49 is the underlying study.

A. Right. So Dr. Pinkleton -- Pinkerton worked for NIOSH, so did this out of NIOSH, and looked at garment workers exposed to formaldehyde in an update. So this was the most recent. I think there were three -- two -- two prior to that. But in this case, they specifically addressed the question in the overall cohort, was there a risk associated with myeloid leukemia. In this case, acute myeloid leukemia, so -- I mean, they didn't do acute promyelocytic leukemia, but this is as close as we get.

And in that case, there was a slight increase in the SMR of 1.34. But it was not statistically significant, so my interpretation of that is that it's -- there's -- there's -- this study does not provide any evidence that being exposed to formaldehyde as a garment worker increases a person's risk of developing AML.

MR. SCHIRRMEISTER: Okay. Offer 48 and 49.

Q. (BY MR. SCHIRRMEISTER) David, I'm going to hand you what has been marked as Exhibit 50 and 51. The slide and the underlying Coggon study.

A. Right. I think Dr. Coggon was from the National --

MR. GIANARIS: I got to object. No question pending.

THE DEPONENT: Oh, sorry.

Q. (BY MR. SCHIRRMEISTER) Who was Dr. Coggon?

A. I don't know him, but he apparently was working with the Medical Research Council in England. And he published this paper, "Extended follow-up of a cohort of British chemical workers that were exposed to formaldehyde."

Q. Okay. And -- and did -- are there tables contained within that study that measure --

THE REPORTER: I'm sorry. Can we go off the record.

THE VIDEOGRAPHER: Going off the record. The time is 12:40.

(Recess taken, 12:40 p.m. to 12:42 p.m.)

THE VIDEOGRAPHER: We are back on the

record. The time is 12:42.

Q. (BY MR. SCHIRRMEISTER) Okay. David, I've handed you what has been marked as Exhibits 50, which is the slide pertaining to the Coggon 2003 study, and then 51, the underlying study. Tell us who Dr. Coggon is and what his study was about.

A. I -- I don't know Dr. Coggon, but he apparently, based on the affiliations, works for the Medical Research Council, which is a federal agency in the UK. That's my understanding. But he did a follow-up of a large cohort of British chemical workers that were exposed to formaldehyde.

Q. And was -- are there tables there examining the relative risk of developing leukemia following exposure to formaldehyde?

A. Yes.

Q. Okay. And there are -- just explain the tables there, the three relative risks there.

A. What he did was looked at leukemia based on these ICD codes, the International Classification of Diseases, and did it based on the total cohort. And then when -- segregated out certain segments of the cohort. But in all cases -- and we put them up here so that you could see the numbers a little clearer. So this is the relative risk and this is the

95 percent confidence interval. And no matter how you cut out the workers based on where and when, the overall cohort was 0.91. So is less than 1. There is no association with being a formaldehyde worker and leukemia. And there's the 95 percent confidence interval. Same -- basically the same all the way across.

Q. Okay. And what is the significance of the study with regard to Mr. Wolfe's claim that exposure to formaldehyde caused him to develop leukemia?

A. This study does not support the -- the notion that even people that are working with a lot of formaldehyde in the chemical industry have an increased risk of developing leukemia.

MR. SCHIRRMEISTER: Okay. Offer 50 and 51.

Q. (BY MR. SCHIRRMEISTER) And, David, I'm going to hand you what has been marked as 52 and 53. 52 is the slide. 53 is Dr. Beane Freeman's underlying study. Who was Dr. Beane Freeman and what's the name of -- well, what is her study about and who performed it?

A. Dr. Beane Freeman is a researcher with the National Cancer Institute. I don't know her. I

know some of the other authors. This was -- this was published just this year. And it was their update of the NCI cohort that they have been following now for -- well, since '86, I think, was the first one. And it was looking at the mortality from a lymphohematopoietic malignancy. That's all of them. That's lymphomas, multiple myelomas, Hodgkin's lymphoma, all of the different kinds of leukemias. So malignancies in workers in the formaldehyde industry. So this is NCI's cohort.

Q. Okay. And what was the result of folks who were exposed to formaldehyde? Did they have an increased risk of developing leukemia?

A. This table was the overall workers in this cohort. And the ones that were exposed to formaldehyde, based on myeloid leukemia, so it's not AML, but it at least segregates out the lymphoid leukemias and the myelomas and the others. And as you can see, the SMR, the Standardized Mortality Ratio, relative risk, and the 95 percent confidence interval is right here in the exposed group. And it does not indicate that there was an association between myeloid leukemia and working as a formaldehyde worker.

Q. Okay. And why did you select these three studies to --

A. These three, the last three, these are the most important in the formaldehyde world, because these have large numbers of workers and they have quantification of the formaldehyde exposures.

So the older studies looking at embalmers and pathologists, no one really knows what those people were exposed to. So if you see an increased risk or a decreased risk, it's specla -- it's speculative to say, well, we think that's because of formaldehyde or we think it's because it's some other people, or whatever. You don't know. At least here in these studies, they have gone in and quantitated the formaldehyde exposures.

Q. Okay. So I'd like to go back to our summary slide, David. So, again, opinion No. 1, would you read that into the -- read that to the jury and summarize your testimony.

A. That DuPont and PPG paint solvents do not cause cancer or leukemia. So we discussed toluene and xylene and the petroleum distillates, all the various regulatory agencies that have evaluated those chemicals. Those -- those toluene and xylenes all have small amounts of benzene naturally occurring. They always have. And so when these regulatory agencies evaluate these chemicals and say they do not

cause cancer, they are basing that on the fact that it has a little bit of benzene in there.

Q. Okay. And No. 2?

A. That APL, acute promyelocytic leukemia is not just another subtype of AML, it is a -- a clearly distinct clinical entity. It's treated differently. It's diagnosed differently. The prognosis is different. It's treated different. The cell of origin is probably different. And as we discussed, epidemiologically it appears that APL is a different thing than AML. It behaves differently in these epidemiological studies.

Q. Okay. Point No. 3?

A. That the best available scientific literature does not support a link between benzene and APL. So ignoring the fact that Mr. Wolfe really wasn't ever using benzene, but was using solvents and other things, but what -- what does the literature tell us about people who were using benzene or that had solvents -- used solvents with benzene in them and APL. And that literature does not support that benzene really causes this -- this disease.

Q. Okay. No. 4?

A. This was the quantification that Mr. Wolfe was not exposed to enough benzene to develop

any form of leukemia.

Q. And does that include your discussion of dose response?

A. That's right. So this was the dose response and the quantitative epidemiology, who has published papers and said benzene causes leukemia, AML. We can all agree on that, but it takes a certain amount. And that it takes a certain amount is what that quantitative epi was important in understanding. What is that line? How much does it take before we would start saying, yes, that -- that's a reasonable assumption that that exposure could have caused that disease?

Q. Okay. And -- and you're familiar with Mr. Russell's exposure estimate for Mr. Wolfe?

A. Mr. Russell's and Mr. Keller's for PPG. And I've read all of them, yes.

Q. Okay. And do they approach the levels that have been found to cause an increased risk in disease?

A. They do not. As depicted on that slide. And even if you don't agree and think that it's going to be ten times more than what Mr. Russell potentially estimated, it's still nowhere near enough to get to where there's an meaningful increased risk of

leukemia.

Q. And you disagree -- or do you disagree with Dr. Milman's opinion that dose is irrelevant?

A. I cannot understand the basis for that statement. So, yes, I completely disagree with that.

Q. What is No. -- opinion No. 5?

A. That was the "Painters as an occupational group collectively are not at an increased risk of developing leukemia."

So the 40 or some studies that we talked about, we chose the three largest and most recent; but overall, that literature does not support that being say painter, in and itself, increases your risk of developing leukemia.

Q. Okay. And finally No. 6?

A. That even pretty high exposures to formaldehyde -- you can't be exposed to really high levels of formaldehyde, because it's so irritating. So the exposures are relatively low even in these industries where they are using formaldehyde. But when they have quantitated and actually gone in and said, Okay, these people were exposed to formaldehyde, you do not see an increase in leukemia or AML.

There's no data to say whether you see an increase in APL. There's certainly no information

that would suggest that you would.

Q. Okay. And you've done this work in this case so far and appeared today on behalf of my client, DuPont, and on behalf of Mr. Miller's client, PPG, correct?

A. Correct. That's -- that's who I'm here today on behalf of, yes.

Q. Okay. And what's your rate that you charge us for the work that you've done in the case?

A. My hourly rate is $300 an hour --

Q. Okay.

A. -- for this kind of work.

Q. And you've been paid for your work that you've done so far today?

A. I -- I've been paid some, yes.

Q. Okay.

A. Yes.

Q. All right. And, again, you're not going --

A. We're not even, if that's what you're implying.

Q. No, I'm not -- I'm not, sir. I'm not, sir. And again you won't -- the jury will see you by videotape, correct?

A. I guess.

Q. Right. Okay. And that's because you have a long planned trip that requires you to be out of the country?

A. Yes. I would like to be in North Carolina, but I'll be in Nepal instead.

Q. Right.

MR. SCHIRRMEISTER: Okay. Finally, as a matter of housekeeping, I want to offer 52 and 53. I believe I've offered all Exhibits 1 through 53, but if I have missed one in the course of my questioning, David, I will just reoffer all Exhibits 1 through 53 and pass the witness. Thank you for your time, sir.

THE DEPONENT: You're welcome.

MR. GIANARIS: Renew all of my objections and the standing objection to the admission of these exhibits that were created for this trial.

THE DEPONENT: Could we take a five-minute. Thanks.

THE VIDEOGRAPHER: Going off the record. The time is 12:53.

(Recess taken, 12:53 p.m to 1:05 p.m.)

(The following testimony was on the record, but not on the videotape record.)

MR. GIANARIS: For the record, this is a voir dire of the witness outside of the presence of

the jury on the issue of some testimony regarding the State of North Carolina.

VOIR DIRE EXAMINATION

BY MR. GIANARIS:

Q. Sir, for this part of the deposition and for the remainder of my portion of the deposition, can we agree, like you agreed with Mr. Schirrmeister, that any opinions you give will be to the same legal and scientific scrutiny that you agreed with Mr. Schirrmeister on?

A. The standard? Sure.

Q. The standard.

A. Yes.

Q. Yes. Okay. Are you familiar with the benzene regulations or regulations as to benzene within the state of North Carolina?

A. No.

Q. Okay. Have you studied them in any way?

A. State of North Carolina?

Q. Yes.

A. I've never done any work for the State of North Carolina.

Q. And preparing for this case and preparing for the testimony you just gave on direct examination, you didn't study the regulation as they related to

benzene within the state of North Carolina?

A. No.

Q. You didn't study what North Carolina said or hasn't said about what different products can cause cancer or not, have you?

A. No.

Q. And you don't know -- you're assuming, but you don't know, what national organizations or federal governmental bodies that do regulate these topics or have spoken on these topics -- you don't know which ones North Carolina has adopted or not adopted, do you?

A. I can't specifically address each one. My understanding -- and they had it on -- I think it was the North Carolina Department of Labor -- on their web site. My read of that was that they are deferring to OSHA and ACGIH and EPA and all of the ones that are listed. And they are -- they are adopting -- they are not doing their same -- they are not conducting their own epidemiological evaluation. That's -- that's my understanding.

Q. Right. You don't know -- you have no information that North Carolina has ever conducted their own epidemiological investigations on these topics?

A. I do not know that, no.
Q. And you've been to the web site for the Department of Labor in North Carolina, and you see that they have on their web site some other organizations' epidemiological studies; is that right?
A. Right. That they -- my understanding was that they are deferring to these national groups for their own position, but I -- only that one page.
Q. You just read one page?
A. Yeah.
Q. Okay. All right. That's enough, thanks.

VOIR DIRE EXAMINATION

BY MR. SCHIRRMEISTER:

Q. Just take a question. So, David, your testimony is that with regard to the standards -- workplace standards followed by the state of North Carolina, you examined the North Carolina Department of Labor web site, which stated that it incorporated the standards set by several federal or private agencies; is that correct?
A. That's my understanding of what they did, correct.
Q. Okay. As opposed to the North Carolina Department of Labor going out and independently doing its own studies?

A. That's -- that's exactly right, yes.
Q. Their web -- the North Carolina Department of Labor web site indicates that they incorporated the standards of the federal government, which, in turn, did those standards or evaluations?
A. And did those studies, that's my understanding, yes.
Q. Okay. And that forms the basis for your testimony in which you describe the position of North Carolina with regard to whether toluene, xylene, and petroleum distillates with naturally occurring trace benzene are capable of causing cancer or leukemia?
A. Correct.

VOIR DIRE EXAMINATION

BY MR. GIANARIS:

Q. Just so we're clear, can you tell me how you got that web site, where that web site is, or if you printed that page off?
A. You know, I did have it, and I didn't bring it with me. It actually came from Mr. Schirrmeister's office.
Q. Okay. So -- that's good to know. This page of the web site was printed off and sent to you by the lawyers?
A. It wasn't sent to me. It was present in

a meeting that I attended.

Q. Okay. So you attended a meeting with the lawyers in this web -- and this page from the web site had been printed off?

A. That's right.

Q. You've never even been to that web site?

A. We did look at it in that meeting. So I did go to it then, yes.

Q. That particular page?

A. Yeah.

Q. Okay. And you're your original answer to my question -- I think your answer to Mr. Schirrmeister -- or Mr. Schirrmeister's question was a little different than to my question. You said that it's your understanding that that's what North Carolina has done and deferred. I think he asked, Is that what you read? He asked you, Did you read that they deferred? It doesn't say on that page that they deferred to other organizations, did it?

A. I don't remember the exact language. It was just my understanding that they had deferred to other federal agencies. And that's what they are basing their position on.

MR. GIANARIS: Okay. All right. That's all I have. I just ask that that page be produced to

me so we can have it for the judge when we go over that issue.

MR. SCHIRRMEISTER: I'll be happy to do that. Please send me a letter.

MR. GIANARIS: I might have to write on my hand.

MR. SCHIRRMEISTER: We'll have it in the transcript. It won't be a problem. The North Carolina Department of Labor web site. I can give you a piece of paper.

MR. GIANARIS: This is better. I won't lose it.

MS. MANELA: Hopefully not.

MR. GIANARIS: Ready when you all are.

THE VIDEOGRAPHER: Okay. One moment, please. I've finished with the voir dire.

(The following testimony was on the record and on the videotape record.)

THE VIDEOGRAPHER: We are back on the record. The time is 1:12.

CROSS-EXAMINATION

BY MR. GIANARIS:

Q. Good morning, Dr. Pyatt -- or afternoon now.

A. Afternoon.

Q. How are you?
A. I'm good, thank you.
Q. Just so the jury can reorient now and understand what we're doing. This is Ted Gianaris, and I'm one of the lawyers along with Bo Drew for the Wolfe family. You understand that?
A. Yes.
Q. Okay. And we're here in your home town in Boulder, Colorado?
A. Yes.
Q. And we're at the St. Julien Hotel and Spa; is that right?
A. I think they do have a spa here, yes.
Q. That's what the pens I think and the note pads in front of us say?
A. Yeah.
Q. This is -- this is where you give depositions; is that right?
A. I've given depositions here. It's not the only place, but, yes, I have used these facilities before.
Q. Okay. And you've given plenty of depositions in your time as an expert witness; is that right?
A. I -- I don't know what plenty means. I

-- I think at this point I've given 17.

Q. 17 depositions total?

A. Yes.

Q. Okay. And in all of those depositions, you get paid just like you're getting paid here today, right?

A. I -- in all of the work I do as a toxicologist, I get paid for my time.

Q. Right. And you told the ladies and gentlemen of the jury earlier that you took a vow of poverty when you went to graduate school, right?

A. Yes.

Q. Okay.

A. It's kind of a joke among graduate students, but, yes.

Q. I understand. And -- but you've sworn off of that vow of poverty now, and now you have a business of your own, a toxicology business?

A. I have a full-time job, yes.

Q. Okay. And you're getting paid $300 an hour to testify here and do your work in this case?

A. That's correct.

Q. Okay. And that's being paid at this point by DuPont and PPG?

A. At this point, I think that's right, yes.

Q. Okay. You've -- when you've worked on cases in the past, it's been more than 17 cases, but you've actually testified in deposition in 17 cases, right?

A. That's true.

Q. Okay. Every case you've ever looked at was a benzene case where a person, a worker mostly claimed that they had been exposed to benzene, breathed benzene on their job, and developed a -- some form of blood disease; is that right?

A. That -- yes, that -- those are all the cases that I've worked on, yes.

Q. And in every one of those cases, there's been a medical doctor who has said, on behalf of the worker that, yes, this benzene exposure caused this man's blood disease, right?

MR. SCHIRRMEISTER: Object to the residential?

A. That is -- that's incorrect.

Q. (BY MR. GIANARIS) That's incorrect?

A. Yes.

Q. In most of them, has it?

A. No.

MR. SCHIRRMEISTER: Object to the form.

A. Very rarely. As a matter of fact, I can

only think of, maybe, one or two that a treating physician associated with the patient said, I think this person's exposure had anything to do with their work.

Q. (BY MR. GIANARIS) Right. That wasn't my question. I said there was a medical doctor on the other side of the case, just like you --

A. Oh, oh, oh.

Q. -- on this case?

A. I'm sorry.

Q. You've been hired in this case -- first of all, you're not a medical doctor, right?

A. I am not. I have a Ph.D.

Q. Ph.D. In science -- in type of science, in toxicology?

A. Correct.

Q. Okay. And what I'm saying is the claims that have been brought by these workers, more than 17 of them -- you've given 17 depositions, but more than 17 claims that you've worked on, there's been a medical doctor on the other side, a trained medical doctor who said, yes, that benzene exposure caused that man's or that worker's blood disease?

MR. SCHIRRMEISTER: Object to the form.

A. Yeah. I'm sorry. I don't -- I mean,

there have been some cases where that's true for sure. I don't know whether it's the majority. There have been some cases where there wasn't an M.D. associated, there was other toxicologists and epidemiologists. But there have been some for sure.

Q. (BY MR. GIANARIS) Okay. Or a scientist like a toxicologist or an epidemiologist?

A. Well, now, there's always that.

Q. Yeah.

A. That's always true. It was the M.D. part that threw me off.

Q. Okay.

A. That's not always the case.

Q. Okay. And in every one of those 17 cases, you've worked for the defense; is that right? Or more than 17 cases you worked for the defense?

A. At this point, that's true.

Q. Okay. And in every one of those cases, you found no cause, right?

A. That's not true. In every one of the 17 cases that I've given depositions in, it was my opinion that either the disease was not linked to benzene exposure, that the disease characteristics weren't consistent with our understanding of what a benzene-exposed person would look like or,

alternatively, that the person was not exposed to enough benzene to meaningfully increase their risks. But that's not true for -- for all cases. I mean, I have seen some cases where I was unable to realistically say that I can -- let's put this way. There were some cases that I was not able to say I don't know whether the benzene had any effect or not.

Q. And in those cases, the defendants or it is defendants' lawyers didn't use you as an expert, right?

A. They didn't use me past that point, that's true.

Q. Okay.

A. I don't think those cases went much further.

Q. Okay. And you're -- you're a -- you're a scientist who specializes his -- his toxicology or focuses his toxicology on diseases of the blood, correct?

A. That's true, yes.

Q. Okay. What's that called again?

A. Well, hematotoxicity, hematopoietic toxicology, immunotoxicology. That's probably the biggest category.

If you look at SOT, the Society of

Toxicology, and you look at all of the various subspecialties, hematotoxicity there's maybe one me. But immunotoxicity, there's going to be several. So those are the categories.

Q. But there's a lot of doctors who study these diseases of blood. In fact, you said you just went over to Germany, and there was a big group of you all got together and talked about the sciences; is that right?

A. That's correct. And by doctor, of all -- all denominations.

Q. Right. So kind of an ecumenical group.

A. Well, there was M.D.s, there were Ph.D.s, you know, so -- so there were -- there were both.

Q. Okay. The stem cell, that is the basic building block of the blood; is that right?

A. I think that's a fair assessment. It is the most primitive cell that, to the best of our ability, we believe gives rise to all the other blood cells.

Q. And you told us earlier -- when Mr. Schirrmeister asked you a question about the definition of stem cells, you were kind of taken aback and you were thinking about it, and you told us that blood -- the scientists who study blood, when they get

together, they have passionate arguments over the definition of a stem cell; isn't that right?

A. They do indeed.

Q. They do. But then you told us -- you said but it's really irrelevant, didn't you?

A. Well, what I meant by that -- I mean, they certainly would not think it's irrelevant and then they would be passionately arguing with me over that comment. What I meant is it wasn't that important to our slide.

Q. Oh, okay.

A. To what we were discussing today in this -- in this case.

Q. Okay. Let's look for a minute at slide -- page 7, if we could.

MR. GIANARIS: We can go off for a second.

THE VIDEOGRAPHER: Going off the record. The time is 1:19.

(Recess taken, 1:19 p.m. to 1:20 p.m.)

THE VIDEOGRAPHER: We are back on the record. Time is 1:20.

Q. (BY MR. GIANARIS) Okay. We put up what is page No. 7 -- I think it was Defendants' Exhibit 4 -- for a moment.

Now, I want to talk to you about these categories for a minute: Toluene, xylene, and petroleum distillates. Let's start with toluene. The -- the toluene in DuPont and PPG's paints and solvents is contaminated with benzene, isn't it?

MR. SCHIRRMEISTER: Object to the form.

A. Well, I think even -- even today, if you hydro-treat toluene, which is a really sophisticated process, you cannot get all of the benzene out. So there is going to be some benzene that is part of that toluene. So there's -- there's always going to be a little benzene in toluene.

Q. (BY MR. GIANARIS) So DuPont and PPG's toluene is contaminated with benzene?

MR. SCHIRRMEISTER: Object to the form.

A. It has some naturally occurring benzene in it.

Q. (BY MR. GIANARIS) You don't like the word contaminated? You're using naturally occurring rather than contaminated?

A. Well, I think it's the same.

Q. Okay. It's the same. I thought so. Okay. Xylene in DuPont and PPG's paints and paint solvents is contaminated with benzene, isn't it?

MR. SCHIRRMEISTER: Object --

Q. (BY MR. GIANARIS) -- that second box?
MR. SCHIRRMEISTER: Object to the form.
A. Because of the way they produced xylene -- not they, but the way xylene is produced, I don't think it's possible to get every molecule of benzene out of xylene.
Q. (BY MR. GIANARIS) Okay. So it's contaminated with -- DuPont and PPG's xylene is contaminated with some benzene?
MR. SCHIRRMEISTER: Object to the form.
A. It has some benzene in it. Sorry.
Q. (BY MR. GIANARIS) And the petroleum distillates -- distillates --
A. Distillates.
Q. -- distillates. Easy for you to say.
A. Yeah.
Q. The petroleum distillates in the DuPont and PPG paints and solvents are contaminated with benzene, aren't they?
MR. SCHIRRMEISTER: Object to the form.
A. I would put those into the same category. They all contain small amounts of benzene.
Q. (BY MR. GIANARIS) Okay. And then down on the bottom of this slide, we have IARC and NIOSH and ACGIH, which I think is the American Conference of

Government and Industrial Hygienists, NTP, ATSDR, EPA.

I just want to talk about those groups for a minute and the heading there. This isn't completely accurate, is it, this slide?

A. Well, you'll have to be a little more specific. We -- this is one of the things that I specifically went through and looked at these various documents and piled up these documents from these agencies and made sure that they had all weighed in on these various classes of chemicals.

Q. Right. So when you went through the -- you went through the IARC documents, piled them up and you waded through them?

A. Yes.

Q. Same with the NIOSH documents, same with the ICGIH documents, et cetera, right?

A. Well, I actually did it on a chemical -- per-chemical basis.

Q. Okay.

A. I said, Okay, here's all of the things on toluene. Do I have something that IARC said about toluene? Do I have something that NIOSH said about toluene? So I -- I did it that way.

Q. Okay. And none of these organizations talked about what you -- you have up here in the

heading of this particular slide, right, they didn't look at DuPont and PPG paints and solvents. They didn't extensively evaluate and study DuPont and PPG's paints and solvents, does they?

A. No, they didn't evaluate these specific products.

Q. Okay.

A. But since those are the main ingredients in those paint solvents, that's -- that -- it has some bearing to what we were looking at.

Q. Right. It has some bearing; but by no stretch of the imagination did any of these groups down here at the bottom look at DuPont and PPG paints and solvents and extensively evaluate and study them, did they?

MR. SCHIRRMEISTER: Object to the form.

A. Oh, that's the part that you said is not accurate.

Q. (BY MR. GIANARIS) Yeah.

A. Oh, I got it. Well, I guess it depends on your definition. I mean, if the paint solvents that you're referring to are toluene and xylene and the petroleum distillates, then it is totally accurate.

If you're saying did they go get a PPG

paint solvent that is something different than these, then, you know, that would not be accurate. But these are the PPG and DuPont paint solvents, the most common. And they did look for those. So it depends on your definition, but I think it's accurate.

Q. Okay. But you don't -- and maybe it's tomato, tomato, but these groups down at the bottom, so we're clear, did not in any way, shape, or form evaluate DuPont and PPG paints and solvents? They didn't actually go look at them and evaluate them and write about them?

MR. SCHIRRMEISTER: Object to the form.

A. What they evaluated -- I think I see where you're going. What they evaluated was toluene.

Q. (BY MR. GIANARIS) Okay.

A. So when you look at a DuPont paint product and it has toluene in it, okay, well, that evaluation, in my view, is relevant to the toluene that's in a DuPont product.

Q. Okay.

A. Did they go and pull a DuPont product off the shelf and say, Is this toluene any different than all of this other toluene? I don't know the answer to that. My guess is no.

Q. Right. Okay. Let's go to page 29,

please, Exhibit 27. Okay. You used this to describe the dose response -- to describe dose response, right?

A. Well, kind of. What -- what we tried to depict here was this notion that even very safe things that everyone considers and are exposed to all of the time, like water and salt, can be toxic if you are exposed to enough of them.

Q. Right. And what we're talking about over here in the red -- just so the jury -- we make sure it's clear to the jury, in the red is an acute overdose. That's -- that's getting 7 1/2 gallons of water pretty fast or 5 pounds of sugar, I guess, shoved down your throat, right?

A. Yes.

Q. Be kind of crazy scenario?

A. Yes.

Q. Okay.

A. That's true.

Q. That's not over time? I mean, you could have 5 pounds of sugar maybe a week or maybe even less. You might gain some pounds, but you're not going to die?

A. You're absolutely right. And we even -- I even thought about putting that this was acute. But you're exactly right. It wasn't -- I was not trying

to make any claims about sugar's toxicity, only illustrate to the jury that you need to understand how much a person is exposed to before you can really get an idea of the toxicity of that compound.

Q. Sure. But there's different types of exposure? I mean, the length of time you're exposed is important, right?

A. You're ab -- absolutely right.

Q. Chronic exposures over time are different than acute exposures, like 5 pounds of sugar all at once?

A. That is absolutely true.

Q. Okay. Let's go to page 30, please, which is Exhibit 28. Okay. Now, this is also on that same part of your testimony that you said is critical to understanding toxicology. And here you're talking about something that when it's on the left side of the threshold for toxicity line is beneficial. Isn't that right? In this -- in this case, this example?

A. Um-hum, yes.

Q. All right. We're talking about aspirin and how it can help keep you from having a heart attack?

A. Yes.

Q. And again -- I don't want to go back to

the last slide, but if you take a bunch of bottles of aspirin all at once, you're doing to die?

A. Well, not even -- I mean, some of the gastrointestinal bleeding, you would get that if you took three or four aspirin on a chronic basis, so that actually is not an acute -- aspirin -- well, I mean, I understand what you're saying, and that's true. Aspirin doesn't have that much of an acute toxicity, but you certainly can OD on it.

Q. Okay. And but -- so the jury understands, benzene does not -- this -- this isn't really applicable to benzene. Benzene doesn't have a beneficial effect below the -- the -- on the left side of the broken line there, does it?

A. It was not my intent to imply that.

Q. Okay.

A. I don't think anyone has uncovered a -- a potential beneficial effect of low exposures to benzene.

There is a guy in the University of Texas, Stephen Safe, who's -- he has put forth this notion of -- it's called hormesis. And in hormesis almost every dose response curve is biphasic, and that really low exposures do have beneficial effects. We just always don't know what those are. I'm not saying

that's true -- for benzene, I'm certainly not saying that.

Q. Okay. Let's go to page 31, please, Exhibit 29. Okay. Now, this is the same basic testimony you gave. This illustrates the same basic testimony about dose response. And here we're talking about alcohol; is that right?

A. Correct. Ethanol, yes.

Q. Ethanol, which is in beer --

A. It's what people drink, yes.

Q. Okay. And what you're illustrating here is if you don't drink, you don't get drunk; but if you drink way too much, you can have a coma or die, right?

A. Correct.

Q. Okay. This is -- again, this is an acute exposure, right, something that happens fast and all at once in a continuous exposure, not chronic over time?

A. That's true. The chronic effects would be cirrhosis of the liver and other kinds of effects for chronic alcoholism. But, sure, this is more on -- like a night out.

Q. Night out. Okay. Good night out, you get giddy; bad night out, you go in a coma?

A. I would hope that doesn't ever happen to

anyone, but it does.

Q. Okay. And you just mentioned -- you kind of anticipated my next question. About types of injuries you can have exposures -- low, low exposures to alcohol, drink a few drinks a day, never get anything more than, maybe, giddy, but still have injury, right, like cirrhosis of the liver?

MR. SCHIRRMEISTER: Object to the form.

A. Yeah, that might not be the best example. For one thing, a few drinks a day, I wouldn't consider that to be low exposure. Now, the giddy part is true. People develop tolerances for that, and they may take a lot more to have this, you know, euphoric, giddy feeling. But cirrhosis is a complete reorganization of a person's liver with scar tissue, and it takes a lot of alcohol over a long period of time.

Q. (BY MR. GIANARIS) If you drink over 20 years, you never get drunk enough to have an acute reaction injury, like coma or death, but you drink over a long period of time, you can have chronic injury, right?

A. That -- that's true. You may not have to go -- in your individual episodes, you may not have to go to alcohol poisoning to get cirrhosis. But I -- you know, the lower end, I don't think evidence

supports that -- that these would give you cirrhosis.

Q. They could give you some injuries in a susceptible person, couldn't they?

A. I'm not sure. I don't really know that much about the dose response relationship with cirrhosis; but looking at cirrhotic livers and what I do know about that literature is that those people that have it, they drink a lot.

Q. Over a long period of time --

A. Correct.

Q. -- they drink?

A. Correct.

Q. Okay. Let's look at slide 33, please, which is Exhibit 31. Now, this slide, you -- when you were talking about it on direct examination with Mr. Schirrmeister, he asked you if they saw any -- and I'm paraphrasing here, but if they saw any APLs here. And you kind of leaned back and said, Well, they weren't looking for APLs in these cases -- in these studies, right?

A. Well, he was asking about pliofilm.

Q. Okay.

A. So that -- that question you just asked will vary depending on which of these studies you're talking about.

Q. Let's talk about the study he was talking about, the pliofilm studies, the two up at the top. They weren't -- you wouldn't find any APLs because they weren't looking for APLs, right?

A. They did not specifically look for APLs. And it was a retroactive mortality study, so you're basing the analysis on death certificates.

However, in these cases with the leukemia cases, they did write up case descriptions of the various types of leukemia. And they classified them, like, erythroleukemia and other forms of AML.

So there are some examples where you can say definitively this was not APL. There are two cases in the pliofilm that I think you can't say definitively that this was or was not APL.

So part of your question is right. They didn't specifically look. But I don't think we're in a complete vacuum in terms of the information, because most of those were not APL. We do know that.

Q. Okay. Let's go -- let's go back --

MR. GIANARIS: Let's go off the record.

THE VIDEOGRAPHER: Going off the record. The time is 1:33.

(Recess taken, 1:33 p.m. to 1:34 p.m.)

THE VIDEOGRAPHER: We are back on the

record. The time is 1:34.

Q. (BY MR. GIANARIS) A moment ago, a few moments ago, on direct examination, you all talked about some studies where your testimony was they were looking at APL. And you -- you gave a caveat that they were very small studies. Do you remember that?

A. Some of them were small studies, yes.

Q. It's important to know the size of a study, because the size of the study is -- correlates to the study's power; is that right?

A. That much is true, yes.

Q. Okay. And the larger the study, the higher the power, everything else being equal?

A. All things being equal, you're right. The larger the study the -- the better the statistical power.

Q. And statistical power has to do with the likelihood that the results in the study aren't based on chance, right?

A. It has to do -- that partially, but it also has to do with the ability of the study to detect subtle changes, small changes in risk.

For example, if you have smoking and -- smoking and lung cancer or some other -- where the risks are really high, you can see those in a

relatively small study. But if you have a 50 percent increase in risk or a 75 percent increase in risk, you may not see that in a little study. You have to -- you need a bigger cohort to evaluate those.

Q. So small studies don't -- one thing they -- they -- small studies are more likely to have their results based on chance and they don't detect subtle differences in risk; is that right?

A. I think the last part is --

MR. SCHIRRMEISTER: Object to the form.

A. -- is accurate.

Q. (BY MR. GIANARIS) Okay. You talked about chemotherapy and Mr. Wolfe's chemotherapy; is that right?

A. Well, I think I -- I answered a question about -- but I talked in specifics about altransretonoic acid, the form of chemotherapy that -- that you treat APL with.

Q. And you said that's what Mr. Wolfe had?

A. Right.

Q. Right. And you know Mr. Wolfe had rounds of chemotherapy, correct?

A. Um-hum.

Q. And you know that he had a recurrence of his disease, don't you?

A. I -- I do. I read all of his medical records, but it's been some time; but I do recall that, yes.

Q. And he had a stem cell transplant?

A. Right.

Q. Okay. And Mr. Wolfe's not cured, is he?

A. That, I don't know.

Q. Okay.

A. I really wouldn't be qualified to make that determination.

Q. From what you've seen in the medical records, you don't have an indication that he -- he's been cured of his disease? It's in remission for right now; is that right?

MR. SCHIRRMEISTER: Object to the form.

A. I don't remember how long it's been in remission, and I don't remember the data associated with that. If it's in cytogenetic remission and it has been in remission for some time, then his long-time -- long-term prognosis is pretty good, but that's -- that's really not my area.

Q. (BY MR. GIANARIS) Okay. You know that Andy Wolfe's blood cancer, his leukemia or APL, was caused by -- or he claims it was caused by repeated exposure to solvents in paint products over his

career?

A. That's my understanding of the allegations, yes.

Q. Okay. And you've studied Andy Wolfe's case, you've read his transcripts, you've read his medical records, you've read exposure reports; is that right?

A. All of that has been some time ago; but, yes, at one point or another, I did.

Q. You needed to do that to give your opinions that you talked about today. I mean, at one to another, you had to do that to educate yourself about the case, right?

A. Right. I mean, most of what I did to educate myself on this case was the -- I mean, I'm a toxicologist, so I was mostly interested in the scientific data associated with his disease.

The various exposure experts, it was their job to figure out what he did and where he did it and what his exposures might have been while he was doing that. So I read his report -- their reports and I was interested in it, but I didn't do that.

Q. But you've read them?

A. Yes.

Q. Okay. You read -- I saw in your report

you read his deposition?

A. Yes.

Q. Okay. And you know he worked for over 20 years as a car painter?

A. '85 to 2004. I think --

Q. Or almost 20 years?

A. I think that's right, yes.

Q. Started in high school. When he was in high school, he started painting cars?

A. Right. Right. On the weekends and things, yes.

Q. Okay. And you know, from what you've read and what you've been told, that he regularly worked with PPG paints and DuPont paints and the products that go along with those paints, don't you?

MR. SCHIRRMEISTER: Object to the form.

A. That, I'm not so sure about. I mean, I -- from reading the experts, the exposure reports, I don't think he used PPG products as long as he did some of the other products; but I'm -- that's not my area.

Q. (BY MR. GIANARIS) So he used DuPont products the most?

A. That -- that's my understanding, but that could be wrong.

Q. Okay. But that's kind of important, isn't it, to know what the guy was exposed to when you're going to give opinions about what caused his disease, isn't it?

A. Well, it depends. It depends on what disease it is and what the exposures are. As I said, there were five individuals, all of them trained in industrial hygiene, and all of them more qualified than me, that -- that quantitated or wrote detailed reports about his exposures.

The other side of that is if a person says, I'm exposed to toluene and I have a disease that's associated with toluene, well, I mean, it's useful and it's -- and it's informative to know what his exposures to toluene were. But the first thing I'm going to do is say, Well, can toluene exposure at any level cause this person's disease?

Q. Okay. So, really, you didn't have to read anything in this case, you just had to know what his disease was, and you could have said right away toluene, xylene, or any amount of benzene didn't cause this disease? You could have done that without reviewing anything?

MR. SCHIRRMEISTER: Object to the form.

Q. You already had that scientific

knowledge? In fact, the lawyers knew that opinion, because you've given that opinion for them before.

MR. SCHIRRMEISTER: Object to the form.

A. Not -- no, not exactly like that. It is -- I mean, I do like knowing what the individual was doing.

Q. (BY MR. GIANARIS) Right.

A. But that was not part and parcel of my opinion. I did base my opinion somewhat on the quantitative exposure information that they said that Mr. Russell provided in his report and Mr. Keller provided in his report where they were looking -- they quantitated the benzene exposure. But I don't believe that -- I do not believe that toluene exposure at any -- at any level is associated with an increased risk of leukemia.

Q. Right. So -- and Mr. Schirrmeister and the lawyers for PPG, they knew that opinion before they ever brought you the Wolfe case, because you've given them that opinion in other cases in the past?

MR. SCHIRRMEISTER: Object to the form.

A. That, I don't know about. I've never given -- I've never worked in a specific toluene, xylene, distillate case and acute promyelocytic leukemia before.

Q. (BY MR. GIANARIS) So did you or did you not have to review Mr. Wolfe's exposures to come up with your opinions?

A. I wouldn't say that I had to, no.

Q. Okay. But you did read his transcripts. And you know that he worked with paints and paint products for over -- or almost 20 years or from high school until he got sick?

A. That's my recollection of what he said, yes. And it's also what the various exposure experts summarized in their reports.

Q. And you know he used PPG products and mostly used DuPont products?

A. That's my understanding, yes.

Q. You know he mixed paints?

A. I don't remember that specifically, but that wouldn't surprise me based on what he did.

Q. Do you know he sprayed paints?

A. I think that goes -- yes, I do know that.

Q. You know he cleaned with solvents?

A. That's my understanding, yes.

Q. Okay. You know he was around these paints and these solvents on a daily basis, at least, you know, unless he was on vacation, five days a week for his career?

MR. SCHIRRMEISTER: Object to the form.

A. I can't speak to that. I think that's right, yes.

Q. (BY MR. GIANARIS) And the benzene exposure that Mr. Wolfe would have had would have been doing that occupation, it would have been to the -- to the contaminants, the benzene contaminants in those paints and solvents, right?

MR. SCHIRRMEISTER: Object to the form.

A. The occupational exposures that Mr. Wolfe would have had would have been because it is a naturally occurring component of those various solvents. I don't know what else Mr. Wolfe did. And we're all exposed to benzene. So he has some background exposure to benzene, just like we all do.

Q. (BY MR. GIANARIS) Okay. And you've -- you've written about those exposures in your -- your report or your letter to the defense lawyers, haven't you?

A. About what exposures?

Q. About the exposures we -- we talked about.

A. I -- you're going to have to be more specific.

Q. Well, we just talked about all the things

that Andy has worked with -- well, let me show you, just so we're on the same page. Paragraph 18, why don't you read that highlighted portion into the record.

A. Okay. "Based on deposition testimony and discovery responses, the only possible occupational exposures to benzene that Mr. Wolfe potentially received would have been as a contaminant of paints, paint and lacquer thinners, adhesives, adhesive cleaners, rubber compounds, other paint-related products and various solvents, such as toluene or mineral spirits."

Q. Okay. So that's -- that's -- you wrote that?

A. Yes.

Q. Okay. Thank you. So it's your opinion, it's in your report, that Mr. Wolfe was exposed to benzene contamination in paints, paint lacquer thinners, adhesives, adhesive cleaners, rubbing compounds, other paint-related products and various solvents?

A. Yes.

Q. Okay. And then you say that "In order to achieve exposure of benzene required to meaningfully increase one's risk of developing AML, there could

necessarily be concurrent exposures to other chemicals present in these products."

So there's other -- there's other product -- chemicals in the products that he used that we just discussed and it would be concurrent exposures, right?

MR. SCHIRRMEISTER: Object to the form.

A. What I meant by that -- I mean, I think what you said is right, that if you're going to say how much benzene is a person exposed to if they are using a solvent that is mostly toluene or xylene and you're going to calculate a dose of benzene, when they are exposed to that benzene, they are also being exposed to other aromatic things that are in that mixture. They are being exposed to toluene, they are being exposed to xylene, they are being exposed to other things. That was -- that was what I intended to convey in that paragraph.

Q. (BY MR. GIANARIS) What does hematological toxicity mean?

A. Hematological just means the blood. So it would be toxicity to the blood or the blood-forming organs.

Q. And you write also in your report, "As hematological toxicity is universally observed

following exposure to benzene, this indicates that either there is some type of interaction that reduced benzene toxicity or that there is simply not enough benzene present to be a toxicological issue."

I want to ask you what you mean by hematological toxicity is universally observed following exposure to benzene.

A. Right.

Q. What does that highlighted sentence mean?

A. Right. So -- well, what I mean is that occupational workers who were exposed to high levels of benzene, they get hematological toxicity associated with them, cytopenias and hypocellular marrows and aplastic anemia, depending on the dose and the longevity and other things. But when you're looking at people that are exposed to toluene, methylbenzene toluene, you don't -- you don't see that. You don't see it in the animals, you don't see it in the human studies.

So while those animals are being exposed to benzene through the toluene, you don't see the same profile of toxicity that you see when those animals are exposed to pure benzene. So there's something else involved, whether it's the toluene decreasing the toxicity or whether there's just not enough benzene in

the mixture to cause those effects. That's what that paragraph was intended to imply.

Q. Okay. When a solvent gets on a person's skin, can the -- can the chemicals in that solvent pass through the barrier of the skin and get in the bloodstream?

MR. SCHIRRMEISTER: Object to the form.

A. I think as a -- just a general principle, some can. But it varies considerably, depending on the solvent. It varies considerably, depending on characteristics of the skin and where that, you know, exposure occurs, how long it's -- I mean, there's so many variables. But as a general principle, yes, dermal exposure can occur.

Q. (BY MR. GIANARIS) That's a pathway of exposure just like inhalation?

A. It is a pathway of exposure for benzene. It is certainly not considered to be nearly as important as inhalation, but you can't totally rule it out.

Q. How about ingestion? Is that a pathway of exposure? I mean, if -- if chemicals or solvents get on some -- in someone's coffee or their hands and they rub their mouth, can you ingest benzene that way, too?

A. You know, that's so -- well, okay, yeah. I mean, benzene in experimental animals that is given via injection as a bolus, it goes right in. So the absorption through the GI tract is thought to be very high.

Now, how realistic is it, from an occupational setting standpoint, that someone is getting solvent in their mouth and ingesting them, that's really not my area. But if it got in, if benzene got into the GI tract, it would be absorbed. That's our understanding.

Q. And once -- once benzene is inside the body, whether it comes in through inhalation or through the skin or through ingestion, what organ does it go to from the blood? Where does it get metabolized?

A. Once it gets into the blood.

Q. Um-hum.

A. You know, because inhalation is going to the lungs first.

Q. Right.

A. So that's a little different. And even the GI tract is a little different because of something that we call first-pass metabolism. But ultimately the benzene that's in the blood is -- we

believe that it's -- it starts its metabolic pathway in the liver.

Q. And then the metabolites from that benzene can go to the bone marrow?

A. That is our general -- sort of our working hypothesis at this point is that the benzene goes to the liver, depending on where and how and other things; but that it produces metabolites that are long-lived enough to get back into the circulation and travel to the bone marrow.

Not -- not all metabolites will -- will ever get out of the liver, but some probably do. And then they are subsequently metabolized in the bone marrow. That -- that's our understanding.

Q. And when benzene causes cancer, that's where the cancer begins, isn't it, in the bone marrow?

A. Well --

MR. SCHIRRMEISTER: Object to the form.

A. Yeah, cancer is too general of a term.

Q. (BY MR. GIANARIS) Leukemia?

A. And I don't even believe that benzene causes all forms of leukemia. AML --

Q. You have it.

A. Okay.

Q. Let's say when benzene causes AML, that's

where the cancer begins, in the --

A. That is our --

Q. -- bone marrow?

A. That is our understanding, yes.

Q. Okay. Tell us -- tell the jury why a healthy and properly functioning bone marrow is so important.

A. The bone marrow is the source of our blood. So in order for us to have adequate oxygen distribution, get rid of carbon dioxide, fight infections, wound healing, all of the things -- clot blood, all of the things that we associate with blood cells in the periphery, we believe most of that development occurs -- not all of it, some of it occurs in the lymphoid system. But a lot of it occurs in the bone marrow.

So the stem cell that we were talking about earlier, how -- whatever you want to say about it, it likely lives in the bone marrow. And it starts its developmental process to these various cell types inside the bone marrow.

Q. And let's talk about exposures that can damage the bone marrow and exposures that can't. You talked about, sort of, a background exposure. And it's your opinion that -- tell me if I'm wrong -- that

a guy who, let's say, works in this hotel, okay? St. Julien Hotel. And he walks around on the streets and he drives his car and he pumps his gas and he mows his lawn. He is breathing some benzene, right?

A. He is.

Q. Okay. But he is below a threshold. That benzene won't hurt that guy?

A. There is no scientific evidence to suggest that those levels have benzene are having any effect on him. It -- adverse effect on him.

Q. And it -- but it's your opinion that there are some groups of workers that are exposed to enough benzene that they are above the threshold and that exposure can put them in increased risk or cause damage to the bone marrow and maybe acute myelog -- AML?

A. Or aplastic anemia. Absolutely.

Q. Okay. And it's your opinion that Mr. Wolfe is -- despite his 20 years of work as a painter is below that threshold. He's in the same group as the guy working at this hotel, mowing his lawn, pumping his gas, right?

A. Well, he's in the same group in that I don't believe that he had a sufficient exposure to benzene to cause bone marrow toxicity.

I would -- I wouldn't say that he had the same exposures to -- as a guy that works in this hotel; but as that slide indicates, we're not completely in the dark about how much benzene it -- that we believe that it takes to meaningfully increase a person's risk of AML. And it's far higher than what the estimates that I've seen of Mr. Wolfe.

Q. Okay. But if you draw that little line you drew, that broken line threshold, Mr. Wolfe would be below the threshold, just like the guy working in the hotel? He wouldn't be above the threshold like the group -- some groups of workers who are at an increased risk?

A. That's true.

Q. Okay. Chronic exposures to high concentrations of benzene are toxic to the blood and the bone marrow; is that right?

MR. SCHIRRMEISTER: Object to the form.

A. Chronic exposures to high concentrations of benzene? Yes, we've known that for a hundred years.

Q. (BY MR. GIANARIS) And the flip side of that is that the risk of developing AML associated with benzene exposure below the threshold that we talked about is indistinguishable from background. In

that a guy like Mr. Wolfe who worked for 20 years as a painter, he doesn't have any more risk than a guy walking down the street?

A. He doesn't have an increased risk that can be identified epidemiologically. I mean, it depends on how you -- how you regulate benzene and what you think about the dose response for benzene, but he has not even approached the levels that we believe are required in order to meaningfully increase his risk of bone marrow damage.

Q. Okay. Let me -- let me just show you your report, because that's what you -- you wrote exactly what I said.

A. Okay.

Q. Right here. That is the risk of developing AML associated with benzene exposure below this threshold would be indistinguishable from background.

A. Right.

Q. Right?

A. Yes. And I said -- I mean, it may not be exactly background, but with current epidemiological methods, you won't be able to tell those apart.

Q. It's indistinguishable from the background?

A. Exactly.
Q. Okay. So just so the jury understands, it's your opinion that Mr. Wolfe, with his 20 years of painting, with potential inhalation, ingestion, and dermal contact to solvents, is below that threshold, and his risk of developing AML is indistinguishable from background? That's your opinion in this case?
A. Yes.
Q. Okay.
MR. GIANARIS: Let's go off for a moment.
THE VIDEOGRAPHER: Going off the record. The time is 1:55.
(Recess taken, 1:55 p.m. to 2:02 p.m.)
THE VIDEOGRAPHER: We are back on the record. The time is 2:02.
Q. (BY MR. GIANARIS) Dr. Pyatt, just refresh the jury's memory what IARC is?
A. IARC. The International Agency for the Research on Cancer.
Q. And you're a cancer researcher, aren't you?
A. Sure.
Q. Okay. IARC is well-respected by cancer researchers, isn't it?
A. Yes. Yes, IARC -- yes, it is a

well-respected group of scientists.

Q. And I want to ask you about some other groups. Are you familiar with the National Safety Council?

A. The National Safety Council? I've seen them, but I'm not as familiar with them -- I've never read anything that they have written.

Q. Okay. I'm going to ask you about that in a moment. I want to tell you the National Safety Council has its headquarters in Chicago, and it's a trade organization, has members from industry and product manufacturers, worker representatives, unions, safety men, scientists. And their -- their mission is to -- is to disseminate information to create safer workplaces.

A. Okay.

Q. You're familiar with them and you understand that concept that type of group?

A. I do now, yes.

Q. Okay. Let's talk historically for a minute so the jury can put things in perspective. Way back in 1950, the National Safety Council wrote benzene is one of the most poison -- one of the most insidious poisons ever to find in industrial use. And then they wrote in the same -- in the same article a

victim may become incurably poisoned before he feels ill.

So I mean, is -- is that still the understanding today, that benzene is one of the most insidious poisons ever to find in industrial use?

MR. SCHIRRMEISTER: Object to the form.

A. I -- that's hard to answer. And I don't quite know what insidious means. So are you asking me if -- is that what people still think now?

Q. (BY MR. GIANARIS) Yeah.

A. There -- there might be some people that still think that.

Q. Okay. And does that surprise you that that was -- that was known and published back in 1950?

MR. SCHIRRMEISTER: Object to the form.

A. I -- I'm not surprised nor un -- un -- not surprised. I mean, we've -- we've known about benzene's toxicity for -- for some time.

Q. (BY MR. GIANARIS) Okay. Are you familiar with the American Petroleum Institute?

A. Yes.

Q. Okay. And you've -- you've testified in the past for defendants -- you've worked on cases for defendants who were members of the American Petroleum Institute; is that right?

MR. SCHIRRMEISTER: Object to the form.

A. I would assume that they -- they are members, yes.

Q. (BY MR. GIANARIS) You've worked for oil companies?

A. Correct. Yes.

Q. Okay. And oil companies make up the American Petroleum Institute, among other -- among other members, right?

A. That's correct.

Q. Are you familiar -- you've been shown that way back in the 1940s, the American Petroleum Institute reviewed the toxicology of benzene?

A. I've seen that 1948 Drinker paper, if that's the one you're referring to.

Q. Yeah, that's the one I'm talking about. So you're familiar with that?

A. Yes.

Q. We'll mark that as Exhibit 1. And I'll show you here -- read the highlighted portion there from the Drinker paper you just mentioned.

A. "Insomuch as the body develops no tolerance to benzene, and as there -- and as there is wide variation in individual susceptibility, it is generally considered that the only absolute safe

concentration for benzene is zero."

Q. Okay. And that's what the API was saying back in 1948, correct?

A. That's what this --

MR. SCHIRRMEISTER: Object to the form.

A. -- Professor Drinker wrote in this paper.

Q. (BY MR. GIANARIS) Published by the API?

A. Yeah. But I don't know what the API was -- I mean, I -- that's a big group, but that's what this one individual said, yes.

Q. Okay. And you've -- you've -- you've read and heard working in these cases about similar sentiments the N -- the NS -- what the National Safety Council said what Professor Drinker wrote for the API -- you've heard and read those over the years working on these cases, haven't you, that there's no safe level to exposure of benzene?

MR. SCHIRRMEISTER: Object to the form.

A. Well, I don't think that's the first thing. The -- the thing that you wrote from 1950 from the National Safety Council, that wasn't my interpretation that there's no safe limit. And even in the Drinker study, they go on to recommend a air concentration of -- of 50 part per million that they consider to be regulatory and -- and safe. And you

also have to put it in the context of what our ability -- what the science allowed these people to actually measure back in the '40s in terms of benzene.

You know, we can measure benzene at way lower concentrations than they were able to then. So what they considered to be zero, now we know that's not zero. There's benzene there.

Q. (BY MR. GIANARIS) Okay. So -- but way back then in 1948, Dr. Drinker was saying the only absolutely safe concentration of benzene is zero?

A. That's what he said, yes.

Q. Okay. And DuPont agreed with that, didn't they, back in the -- in the 1950s?

MR. SCHIRRMEISTER: Object to the form.

A. That, I can't speak to.

Q. (BY MR. GIANARIS) Have you ever looked at any DuPont documents? Has DuPont given you any internal correspondence or memos about what they knew about the -- about the dangers of benzene exposure?

A. No.

Q. Have you ever asked to see those?

A. No.

Q. Okay. Tell me what -- what you know about Andy Wolfe's disease, how it came on, how it progressed, how he was cared and treated for, et

cetera.

A. Can I refer to my report?

Q. Sure.

A. Because I just -- I don't -- it's been awhile since I've looked. I meaning, I can at least tell what you I had in my report. I think we did talk about it.

MR. GIANARIS: We can go off the video while he finds it.

THE VIDEOGRAPHER: Going off the record. The time is 2:09.

(Discussion off the record.)

THE VIDEOGRAPHER: We are back on the record. The time is 2:09.

A. My understanding that he presented with blood abnormalities in 2004, was diagnosed with APL. This is from memory. And I may be wrong, but that he was treated with altransretonoic acid, entered remission, relapsed, maybe put into remission again, had a stem cell transplant that seemed to take, and now is in long-term remission. That's my understanding.

Q. (BY MR. GIANARIS) Okay. Dr. Pyatt, I just asked you about his treatment and you asked to see your report. You didn't put anything about his --

his care and treatment or the progression of his medical condition in your report, did you?

A. No.

Q. You went through and couldn't find anything?

A. That's right. Sometimes, I do. Sometimes, I write little medical histories; but this time, I didn't.

Q. Okay. And do you remember much about -- you gave us a little summary -- a couple of sentences about his medical. How about his exposure? Do you -- do you remember much about what Andy did on a daily basis, how he worked, how he was exposed?

A. No.

Q. Okay.

A. Other than he was, you know, painting cars.

Q. I'd like you to tell me, please -- or give me a definition or an example of what you feel is appropriate for an ethically responsible company to tell the users of its products when its products contain a dangerous substance.

MR. SCHIRRMEISTER: Object to the form.

A. I don't understand that question.

Q. (BY MR. GIANARIS) Okay. Some products,

the people use in their work places contain dangerous substances, right?

A. Right.

Q. Toxic substances?

A. Correct.

Q. Substances that can cause severe injuries?

A. Sure.

Q. Okay. What is the appropriate -- the ethically responsible thing for a company that makes products like that that contain chemicals that can cause those kinds of injuries to tell the end user?

MR. SCHIRRMEISTER: Object to the form.

A. It's really not my area. I mean, this whole warnings and how you word things and how you print things out and how big it is and color and signs, that's a whole area of science that I'm just not familiar with.

So I really -- that's just outside of my area to discuss. I mean, there are people that specifically understand that and what people pay attention to and what they read. And that's just not anything that I know anything about.

Q. (BY MR. GIANARIS) Well, you know what -- what an ethical company ought to do, don't you, I

mean, if they -- if they ought to tell people about the dangers of their products or not?

A. Well, at that level, that's a relatively simplistic level. And I think absolutely they should.

Q. Okay. Do you agree --

A. But what I was saying is I don't know how, what's the best mechanism for disseminating that information. That's -- that is not my area.

Q. Do you agree that it's a good rule that a company should tell workers using its products about all of the dangers that come from using the products?

MR. SCHIRRMEISTER: Object to the form.

A. That's a harder question to answer. I mean, I think that gets into this notion of warnings and what you say and what you don't say and just -- I'm just not comfortable answering that.

I mean, some of the -- at the superficial level, yes; but if it's an exceedingly rare side effect or it occurs at exposures that aren't going to happen, you know, I don't -- I don't know. I mean, I think some companies do that. They put all that on MSDS sheets and some -- I -- I don't know. I just don't know -- I don't know about that. Sorry.

Q. (BY MR. GIANARIS) Have you ever -- have you ever thought about that? Have you -- when you

testify in cases like this, have you listened to other experts testify and sat and thought I wonder -- I mean, what -- what -- what I feel in my gut they ought to do, a company ought to do?

MR. SCHIRRMEISTER: Object to the form.

A. Well, most -- the times that I've looked at it -- and I haven't looked at it as a scientist, just like you said, just kind of on the periphery, it seems like they are.

The -- the companies that I work for, they have made a good faith effort to disseminate this information where it was required. But how and when and what information you specifically include on these, that's just not my area.

Q. (BY MR. GIANARIS) So it's a good thing for a company to make a good faith effort to disseminate the information they have to the workers?

MR. SCHIRRMEISTER: Object to the form.

A. Say that again.

Q. (BY MR. GIANARIS) It's a good thing for a company to disseminate the information they have regarding these possible harmful effects to the workers?

MR. SCHIRRMEISTER: Object to the form.

A. Yes.

Q. (BY MR. GIANARIS) Okay.
A. At that level, yes.
Q. All right.
MR. GIANARIS: That's all I have. Thanks.
MR. SCHIRRMEISTER: Jim?
MR. MILLER: Nothing.
MR. SCHIRRMEISTER: No questions. Thank you, Dr. Pyatt.
THE DEPONENT: You're welcome.
THE VIDEOGRAPHER: This is the end of tape 2 of 2. Going off the record. The time is 2:14.
WHEREUPON, the within proceedings were concluded at the approximate hour of 2:14 p.m. on the 23rd day of September, 2009.

* * * * *

I, DAVID W. PYATT, Ph.D., do hereby certify that I have read the above and foregoing deposition and that the same is a true and accurate transcription of my testimony, except for attached amendments, if any.

Amendments attached ( ) Yes ( ) No

_____

DAVID W. PYATT, Ph.D.

The signature above of DAVID W. PYATT, Ph.D., was subscribed and sworn to before me in the county of _____, state of _____ , this _____ day of _____, 2009.

_____

Notary Public

My commission expires

Andrew Wolfe 9/23/09 (tc)

REPORTER'S CERTIFICATE

STATE OF COLORADO)
) ss.
CITY AND COUNTY OF DENVER)

I, TERESA COOGLE, Shorthand Reporter and Notary Public, State of Colorado, do hereby certify that previous to the commencement of the examination, the said DAVID W. PYATT, Ph.D., was duly sworn by me to testify to the truth in relation to the matters in controversy between the parties hereto; that the said deposition was taken in machine shorthand by me at the time and place aforesaid and was thereafter reduced to typewritten form; that the foregoing is a true transcript of the questions asked, testimony given, and proceedings had.

I further certify that I am not employed by, related to, nor counsel for any of the parties herein, nor otherwise interested in the outcome of this litigation.

IN WITNESS WHEREOF, I have affixed my signature this 1st day of October, 2009.

My commission expires May 8, 2010.

__X__ Reading and Signing was requested.
_____ Reading and Signing was waived.
_____ Reading and Signing was not required.

October 1, 2009

DAVID W. PYATT, Ph.D.
1944 Cedaridge Circle
Superior, Colorado 80027

Re: Andrew Wolfe and Denise Wolfe v.
E.I. DUPONT DE NEMOURS AND COMPANY, et al.
Deposition of: David W. Pyatt, Ph.D.

Dear Dr. Pyatt:

Enclosed you will find a complimentary copy of your deposition taken in the above matter. Also enclosed are amendment sheets for changes if necessary. Please return the signed and notarized signature page and amendment sheet(s), if any, to our office on or before October 19, 2009, in order that the deposition may be filed in time for trial.
Thank you for your attention to this matter.
Sincerely,

Teresa Coogle, RPR
HUNTER + GEIST, INC.
Registered Professional Reporters

c: Ted N. Gianaris, Esq.
James R. Miller, Esq.
Andrew C. Schirrmeister, Esq.