IN THE SUPERIOR COURT OF THE STATE OF ARIZONA
IN AND FOR THE COUNTY OF MARICOPA

Case No. CV-07-005399

VIDEO DEPOSITION OF DAVID PYATT October 8, 2009

RAUL ZENDEJAS and ARACELI ZENDEJAS,

Plaintiffs,

vs.

SHELL OIL COMPANY; SHELL CHEMICAL LP, Individually and as successor-in-interest to SHELL CHEMICAL CORPORATION; CONOCOPHILLIPS COMPANY, VON VERDE CITRUS PACKING HOUSE, INC., an Arizona corporation; VON VERDE HARVESTING, INC., a dissolved Arizona corporation; and VON VERDE CITRUS GROWERS COOPERATIVE, INC., a dissolved Arizona Corporation,

Defendants.

APPEARANCES:

SHRADER & ASSOCIATES
By Keith E. Patton, Esq.
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Appearing on behalf of Plaintiffs.

HAYNES AND BOONE, LLP
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Appearing on behalf of Defendant Shell Oil Company and shell Chemical LP.

Also Present: Nick Borgia, Videographer

Pursuant to Notice and the Arizona Rules of Civil Procedure, the video deposition of DAVID PYATT, called by Plaintiffs, was taken on Thursday, October 8, 2009, commencing at 9:34 a.m., at 7001 Yampa Street, Board Room 1, Denver, Colorado, before Janet Lee Priestley, Registered Professional Reporter and Notary Public within and for the State of Colorado.

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P R O C E E D I N G S

THE VIDEOGRAPHER: We're on the record at 9:34 a.m. Today is October 8, 2009. This begins the videotape deposition of David Pyatt Ph.D. taken by the plaintiff in the matter of Raul Zendejas and Araceli Zendejas vs. Shell Oil Company, et al. We're located at Embassy Suites, 7001 Yampa Street, Board Room 1, in Denver, Colorado. The court reporter is Janet Lee Priestley, and the videographer is Nick Borgia.

Counsel will now introduce themselves and the parties they represent beginning on my left.

MR. PATTON: Keith Patton for plaintiff.

MR. PERRY: Stan Perry for shell.

THE VIDEOGRAPHER: Will the court reporter please swear in the witness.

DAVID PYATT,

being first duly sworn in the above cause, was examined and testified as follows:

EXAMINATION

BY MR. PATTON:

Q Please introduce yourself for the record.

A David Pyatt.

Q Mr. Pyatt -- or is it Dr. Pyatt?

A I have a Ph.D. in toxicology.

Q Okay. Dr. Pyatt, you are a toxicology expert, correct?
A Yes, sir.
Q Were you hired by Shell in this case?
A I was hired by. Shell and I think when I first got involved, ConocoPhillips might have been involved, as well.
Q Dr. Pyatt, were you hired by Shell to give an opinion in this case?
A Yes.
Q Have you testified or been retained on behalf of Shell in other benzene exposure cases?
A There was other case for sure that I worked with Mr. Perry on. Shell may have been a defendant in some other cases that I've worked on, along with others, but I don't specifically recall that. But I wouldn't say absolutely that they have not been involved.
Q Can you explain to us your experience in the area of benzene in working with industry?
A Working with industry?
Q And by example, sir, today you're working with industry?
MR. PERRY: Object to form.
Q (By Mr. Patton) Do you agree?

A Today I'm providing my professional opinion on a toxicological issue that Mr. Perry asked me to evaluate. I'm not sure that -- I mean, I have done projects for the American Petroleum Institute. And in that case, I guess you would say that I was working on behalf of industry.

Q Tell us about your experience in working on behalf of industry with the American Petroleum Institute or the API.

A Well, even when I was a graduate student and was doing toxicological research, we -- my -- the laboratory that I worked in had some funding from API so I guess it would have started 15 years ago. And then since then I've had various projects as a consultant for that trade group.

Q Can you estimate how many various projects you've been a consultant for the API on?

A Three.

Q And are any of those active right now?

A I would say no, equivocating a little, but essentially no.

Q What were the three projects you did for the API?

A Probably the longest one was, we conducted a -- and actually, technically, that wasn't API.

They were involved in it, but it was also with the American Chemistry Council. They kind of had this giant group called the benzene consortium. And what we were tasked with doing was conduct a risk assessment for children with regard to benzene exposure for EPA. So EPA put out this -- they called it the VCCEP program. Are you familiar with that? VCCEP stands for Voluntary Chemical Children's Exposure Program.

So they said anyone out there that's making chemicals that could potentially -- that children could potentially be exposed to, we would like for you to conduct a risk assessment specifically for children, using children-specific factors, and things like that. And so this group decided to do it for benzene, and they hired us to do that part. And so my job was the hazard assessment portion of that project as well as some of the children-specific sensitivity issues. So that was one. Then we presented that to EPA.

Q Okay. Finish your thoughts on this project because I want to talk about it before we go to Nos. And 3.

A Okay. And then -- yeah, so we presented to EPA, they came back with comments, we responded to

those comments, and now it's in the public domain.

Q Okay.

A If you go to TERA's Web site, T-E-R-A, you can find what we said, what we did, and what EPA said in response.

Q Okay. So he so if I understand you correctly, EPA told the general public and chemical manufacturers, Hey, if you're manufacturing chemicals that children are going to be exposed to, we'd like you to do a risk assessment; is that accurate?

A That's correct. And that was the voluntary part.

Q Okay. And then the API said, Okay, we're making chemicals, kids are exposed to chemicals, we'll do a study on it, correct?

A API was involved. But like I said, it was this kind of benzene consortium, and I don't know even know if that's still in existence any longer. So it was sort of a collaborative effort between the American Chemical Council and API.

Q It was a collaborative effort initiated by industry, right?

A Oh, yes; yeah, yeah, just I don't know which -- which one of the industry members, you know, took a lead. I don't know how all that worked out.

Q Okay. And you studied benzene exposure and its possible effect on children?

A Right. We did a formal risk assessment for children with regard to environmental benzene exposures.

Q Okay. Kids living near gas stations, things of that nature?

A Correct; attached garages, cigarette smoke, you know, living next to roads, all of the various exposure pathways that could --

Q Did your -- I'm sorry.

A I was thinking. Go ahead. I'm done.

Q Did your consortium group conclude that kids are exposed to benzene by living near gas stations?

A We're all exposed to benzene so they do have background exposures to benzene, just like all of us do. Actually, living next to a gas station or living in proximity to a refinery was a relatively minor contributor. So while it probably did contribute some, just like it would to anyone's background exposures, it was a relatively minor contributor. The biggest contributors were cigarette smoking and having an attached garage.

Q Children are exposed to benzene above

background levels simply by virtue of having an attached garage in their home?

A I wouldn't say their -- I would say that makes -- no. What I would say is that we are describing their background exposures.

Q Fair enough. When you are -- in a risk assessment you are studying exposure to a substance and the incidence of disease; is that a fair statement?

A We were studying -- no. We were doing a risk assessment, so not really -- it wasn't an epidemiology study. We were actually looking at the incidence of disease. It was a formal, EPA based risk assessment where you use their potency factors and their slope factors and their tox values. And you basically multiply that by the exposures, and you come up with a theoretical risk to children. And then they have guidelines as to when that risk becomes unacceptable. And, you know, it's just a basic risk assessment.

Q What diseases were you looking at in that study or evaluation of risk assessment?

A Well, it doesn't work that way. I mean, what you use is, you use EPA's toxicity values, and those are based on specific end points. So, you

know, that -- we can discuss that. But you don't -- it's not a disease specific risk assessment. You're just looking at what EPA says -- you have to kind of understand how a cancer slope factor works and how you would use it in risk assessment. But it is based on -- I mean, ultimately if you go back far enough, it's based on AML, and the noncancer toxicity is based on lymphocytopenias.

Q Did you look at the relationship between benzene exposure to children and the incidence of leukemia in that risk assessment?

A Not per se, no.

Q Okay. In what way -- if not per se, in what way did you evaluate leukemia, childhood leukemia in relation to benzene exposure in that project?

A Well, you didn't do childhood leukemia because there's no -- there's no data that children have gotten leukemia from benzene. So what you have to do is use the cancer slope factor, which basically says by EPA's calculations, here is the risk for developing AML if you are exposed to X amount of benzene. So then at the simplest level, you take what the kids' exposure to benzene is, you put it into that equation, and it spits out a risk number.

As then long as that risk number is below 1 times 10 to the minus 6, everyone ignores it. If it's below 1 times 10 to the minus 4, you discuss it, but then you ignore it. If it's greater than 1 times 10 to the minus 4, then you do something about it.

Q So you did look at AML in relation to exposure in that study?

A Insomuch as that that is the basis for the cancer slope factor.

Q Okay.

A I mean, we weren't doing an epi study. We weren't going out --

Q I understand.

A -- counting diseases.

Q How much benzene did you conclude that the children were being exposed to? Was there a range?

A There was a range, and you can find it, but I don't remember specifically. I mean, the risk numbers associated with those exposures were less than 1 times 10 to the minus 4, so that I remember.

Q So you didn't find an increased risk?

A Well, it depends on your definition of "increased." I mean, if your definition of "increased" is zero, then sure, it was increased. If your definition of "increased" is something that the

federal government feels like is an issue, then no, we didn't find one. Because the federal government, EPA, did not consider those exposures, which they agreed with, to pose an undue risk to children.

Q What were the second and third studies you did?

A The second study had to do with chemical sensitivity, which was connected to that. And then the third study, third project, if you will, was a re-re-revaluation of the Pliofilm cohort. And we didn't -- I've only done a little on it. We conducted Phase 1, as it were, and we haven't done anything else. We haven't been able to do the interesting stuff yet. I'm trying to think of everything that I've done for them.

Q For the API?

A Correct, yes. So there was that little Pliofilm one. There was the children's risk assessment. There was the sensitivity paper.

Q Let me ask you this. In the first study you did, the API one about benzene in children with the benzene consortium, were any of your -- did the results of any of your work in that risk assessment then translate into additional measures that would protect people?

A No.

Q The chemical sensitivity -- well, let me ask you. The reevaluation of the Pliofilm cohort, did any of your work in that project then translate into other work which would go on to further protect workers?

MR. PERRY: Object to form.

A Well, I'm not quite sure about the way you worded that question. But if that were completed, once we complete that project -- and I can explain to what you we're doing, if you'd like. But once we complete that project, it could impact on regulatory -- a decision that involves the regulation of benzene. The childhood leukemia risk assessment could have if there was a need.

Q (By Mr. Patton) What is the chemical sensitivity project you worked on, No. 2?

A That one had to do with the -- what I did essentially was say, you know, there are other things in the world that give people leukemia. And two of those things that we know give people leukemia are chemotherapeutic agents. And they give adults leukemia, and they give children leukemia. And the leukemias that children get are remarkably similar to the leukemias that adults get and the doses.

So with that as kind of a theoretical framework, what I went and did was looked at all of the clinical literature that I could find, 250 some studies, and evaluated on a study-by-study basis whether or not they had separated out the various ages. And the idea was, does age, as an independent variable, affect a person's risk of developing leukemia from these chemicals. And it did not appear that the children, just by virtue of being children, were any more susceptible to developing AML following chemotherapy than adults were. In fact, most of the studies would suggest that they were less susceptible.

Q So the chemical sensitivity project you did was also relevant to children?

A Yes.

Q It dealt with children?

A Yes.

Q It focused on children?

A Correct.

Am I providing too much detail. I can --

Q It's all right.

Are you involved in -- where is it that you work, the University of Colorado Health Sciences Center in Denver?

A I work for Summit Toxicology, which is my consulting firm. But I have appointments at CU, in various departments at CU.

Q Are you familiar with an ongoing benzene study in Shanghai?

A Yes.

Q Are you involved in that?

A No.

Q Do you know why you're not involved?

MR. PERRY: Object to form.

A No.

Q (By Mr. Patton) It's a weird question.

A Well, I mean, I'm probably on -- to answer that question, no. Let me just tell you the story. I mean, Rich Irons was my thesis advisor, and he's the PI of that study. He's the principal investigator of that study. When I was still at the university and still working with him, he was conducting the feasibility portion of that study, and I was peripherally involved at that point. In fact, I taught an immunology class on his behalf so that he could go spend time in China to figure out whether or not they could actually do this study. After that I had my own grants funded. I was doing my own thing. We kind of parted ways, like every graduate student

and their advisor does, and so we had separate career paths.

Q Rich Irons -- Richard Irons, his career path has involved a tremendous amount of work on behalf of industry related to benzene; do you agree?

A I wouldn't agree that that's the only thing that he's done.

Q I didn't say it was the only thing, sir. I said a large amount. Would you agree that a large amount of his work has been on behalf of industry in relation to benzene?

MR. PERRY: Object to form.

A Yes, yes.

Q (By Mr. Patton) And in fact, it sounds like that's how you got started in doing benzene work on behalf of industry; is that a fair statement?

A No, no. I mean, I said that there was funding from API in the lab where I started with Dr. Irons. But we also had a really big NIH grant, and I think I was working on the NIH grant. I'm not positive about that.

Q What is -- you're a toxicologist, correct?

A Yes.

Q What is toxicology? What is it the study of?

A Toxicity, poisons. I mean, if you look at Casserett and Doull, which is our primary textbook, it says Toxicology, the basic study of poisons.

Q Is benzene a poison?

A Benzene can be toxic.

Q What is the objective of studying poisons, of being a toxicologist?

A That's a good question. It depends. I mean, there's lots of objectives. There are -- you want to understand the dose response relationship. You would like to know how much of the chemical can actually make a person ill, can exert a toxic effect. You would like to understand, if that occurs, is there some way to treat it, is there a way to mitigate that toxicity.

Every toxicologist that I'm aware of -- and certainly in my own research -- we all have a keen interest in the underlying disease process. So while we were looking at benzene and its ability to cause AML, we were also researching the basic pathology of that disease. So, I mean -- and people that look at kidney toxicity, they are also uncovering basic physiological mechanisms for kidney function.

Q Is there something of a loftier objective

in the study of toxicology? That's to say, there's an end goal to help people? Would you agree with that?

A That's pretty lofty. Yes. I mean, it certainly isn't the other way, at least today. In medieval Europe, all the courts had toxicologists. The kings employed toxicologists. And in that situation, those individuals were not necessarily employed to help people.

Q As a toxicologist do you personally consider yourself somebody who is in the business to help people, make people safer?

A I've never really thought about it in those terms, but yes.

Q I mean, do you want the end work of your body of work, all the studies that you've done, all the information that has went into your curriculum vitae, do you want that ultimately to go to help people, make the world a better place and make people safer? Is that part of your sort of mission, as a toxicologist?

A I've never really thought about it that way.

Q So you don't know one way or the other? That's your --

A I certainly wouldn't say that it's the other way, to make people less safe.

Q Okay.

A So sure, I would agree with that. Although I'm just telling you, I've never looked at myself in the mirror and said, Okay, today we're going to go out there and make the world a safer place. I just haven't done that. But I do agree with you. I mean, ultimately by improving our understanding of chemical toxicity, that is ultimately the goal.

Q Improving our understanding of chemical toxicity can help to protect people, can help to protect certain types of workers; fair statement?

A Sure.

Q You said that part of the study of poisons or part of toxicology is to evaluate ways to mitigate tox -- to mitigate toxicity. That's to say, part of toxicology is to evaluate and study ways so that people aren't exposed as toxically. Is that fair, or am I saying that wrong?

A Well, I understand what you're saying. I think toxically is --

Q It's probably not even a word?

A Yeah, but that's all right. I understand

where you're going. No. I would say that part of it would be industrial hygiene, where you're actually figuring out engineering ways to limit, reduce exposure. What I meant -- and that's really the dose response relationship, how much can a person be exposed to, without any anticipated adverse effects. And that is certainly an important part of what we all do.

Q Industrial hygienists, they have the -- part of their job involves the ability, to some extent, to put engineering controls in place, do things to reduce workers' exposure to benzene to mitigate toxicity; do you agree?

A I do agree. And they would base that decision on our toxicology --

Q Right.

A -- dose response information.

Q Right. So you could give those more engineering type folks the scientific and medical information so that they could go do things to mitigate exposure and protect workers. Do you agree with that?

A Yes.

(Exhibits 1 through 3 marked.)

Q (By Mr. Patton) Okay. We've got your CV,

which I've marked as Exhibit 2. It likes like it's 5 or 10 pages. It talks about your professional, studies you've done, consulting work, editorial review, et cetera. We have Exhibit 3. This is a list of all the testimony you've given in lawsuits. Is there anywhere on Exhibit 2 or Exhibit 3 where you did something in your body of work as a toxicologist that contributed to industrial hygienists or other folks actively doing something to mitigate toxicity?

MR. PERRY: Object to form.

A It would be a pretty crooked line. I mean, most of the research that I was doing in the lab would -- you would not expect that exposures typically encountered today that you would expect those toxicities to occur.

Q (By Mr. Patton) Sir, maybe my question wasn't clear. Are you a health scientist?

A Well, I'm a toxicologist.

Q My question is this, sir. In Exhibit 2 and Exhibit 3, is there anywhere in there where you can point to your personal work that resulted in changes down the road, so to speak, that helped to mitigate toxicity such that workers were directly in safer conditions?

MR. PERRY: Object to form.

A I -- I don't know whether that's happened or not.

Q (By Mr. Patton) Okay. You mentioned earlier -- well, hold on a second. Does any part of your -- how old are you?

A I'm 51.

Q Does any part of your remaining career -- does it interest you to do things that would make the world a safer place or maybe protect certain classes of workers?

MR. PERRY: Object to form.

A Well, yeah. That interests me. I mean, I worked at EPA for two years. So that was -- you know, at a lofty level that was to protect the public, to protect the environment. So, I mean, there have been -- maybe I'm being too literal, but I'm trying to think of a specific experimental work that I've come up with myself in the lab that I can draw a straight line to a change in regulation on benzene exposure, and I can't do that.

Q (By Mr. Patton) Or any other exposure, for that matter, correct?

A I can't do that. That's not to say that things that I have published have not been part of a decision-making process. But I can't point to a

straight line and say, okay, because of this study that I did, people went out there and said, yeah, these exposures are too high, and people are getting sick from it.

Q Right. Is it fair to say that you've kind of done the opposite? For instance, Exhibit 3, your deposition testimony, in each of these cases did you testify that the chemical agent did not cause a person's disease?

MR. PERRY: Object to form.

A I wouldn't say that's the opposite.

Q (By Mr. Patton) Okay. Sir, on Exhibit 3 you've testified in 20 depositions involving chemical exposure as the cause of disease, correct?

A Say that again.

Q Chemical exposure as a cause of disease or illness.

MR. PERRY: You're saying all of those on Exhibit 3?

A I'm not --

Q (By Mr. Patton) Let me be a little more clear.

A Please.

Q Dr. Pyatt, the deposition testimony marked as Exhibit 3, these are all the cases you've

testified in, correct?

A Yes, sir.

Q Okay. Have you ever been hired by an injured worker or lawyers or families, people involving an injured worker?

A I have not yet, no.

Q Have you always been retained on behalf of industry or chemical companies?

A So far, yes.

Q Okay. Do all -- excuse me. All of these cases that you've testified in as a toxicology expert, you testified on behalf of industry or chemical companies, correct?

A That's right.

Q In each of these cases -- in any of these cases -- let me ask a better question.

How many of these cases involve benzene?

A They all do.

Q All 20 of them?

A Yes, sir.

Q In any of these cases did you testify that benzene exposure caused the person's disease?

A No; not in those 20, no.

Q Okay. Is it leukemia in all these 20?

A No, sir.

Q Are there -- what are all the diseases reflected in this list?

A Can I see it? I'd have to go through it.

Q Let's go through and just write what disease it is right next to each case.

A Right now?

Q Sure.

A Okay.

Q You can write it next --

A You want me to do it out loud, or you just want me to do it quietly?

Q Let's do it out loud.

A Okay. Wilkerson, this was a nonHodgkin's lymphoma. Remboldt, this was a nonHodgkin's lymphoma. Barker, this was an AML. Detel, this was an ALL. Reed was a Hodgkin's lymphoma. Solis was a nonHodgkin's lymphoma. Behymer, this was a chronic lymphocytic leukemia and a multiple myeloma. Henrickson was an AML, and that actually was a gasoline case.

Q Yeah. If there are any others in there that involve gasoline, please mark them.

A Barker was a gasoline case. Saltonstall, that's an NHL. Baker, this was a monoclonal gammopathy of undefined significance.

THE REPORTER: Say that again.

MR. PATTON: Worry about it later.

A A monoclonal gammopathy of undefined significance. This was an idiopathic myelofibrosis. This was an AML. This was an AML. This was an APL.

Q (By Mr. Patton) APL, acute promyelocytic leukemia, right?

A Right.

Q It's still a myeloid disease, right?

A Yes. This was a nonHodgkin's lymphoma. Bishop was multiple myeloma. Knapper was an AML. This was an AML. We've already done that one. This was an APL/AML. And this one was paints. And this one, Remboldt, this was crude oil. So was this one. So saying these are all benzene-related, it's not totally accurate. The hearing stuff, you want those, too?

Q Sure.

A Okay. Cantu, that was a nonHodgkin's lymphoma.

THE REPORTER: I can't hear you.

THE DEPONENT: I'm sorry. I'm talking to myself.

A Milward, we already talked about that one. So there was a deposition, and then there was a

hearing so it was really the same thing; and Wolfe, the same.

Q (By Mr. Patton) Dr. Pyatt, we just took some time, and you went through Exhibit 3 and you marked next to each of these cases in which you testified on behalf of industry or you were hired by industry. You testified in some 20 cases involving benzene or benzene-containing products which the plaintiffs alleged caused a form of myeloid or lymphocytic leukemia, fair statement?

A Sometimes, not always.

Q What do you mean, "not always"?

A Well, like, for example, the idiopathic myelofibrosis, which was Stromberg, they were claiming that that was toluene. And while they discussed the possibility of a minute amount of benzene in the toluene, they still said, We think it's toluene that's doing it. Thorpe was mineral spirits.

Q Mineral spirits, do those contain benzene?

A At low levels, yes.

Q Does toluene contain benzene?

A It depends. I mean, even if you hydro treat it, you can probably find a molecule or two.

Q Crude oil contains benzene, yes?

A Crude oil has benzene in it.
Q Gasoline contains benzene, correct?
A Yes.
Q You mentioned earlier part of your work as a toxicologist involves an interest in underlying disease progress. You remember saying that?
A Process.
Q Underlying disease process. Okay. Would you agree with me that diseases such as the myeloid leukemias, diseases -- I only want to talk about the myeloid leukemias, okay?
A Okay.
Q Would you agree with me, Dr. Pyatt, that the myeloid leukemias, they do progress through a series of different myeloid disorders before they ultimately arrive at a firm diagnosis? And then once that happens, they might progress to a different myeloid firm diagnosis? Would you agree with that?
MR. PERRY: Object to form.
Q (By Mr. Patton) I probably didn't phrase that real good, but do you understand what I'm asking?
A Kind of.
Q Well, let me ask it in a different way.
A Okay.

Q You issued a report in this case. We'll mark it as an exhibit, right?

A It is. Oh, it's not, but we will sure.

Q Okay. And you talk about my client, Raul Zendejas, having, CML or atypical CML. And I think -- do you say in your report about maybe he has MDS?

A I don't think so, no.

Q Let's get your report, just to be fair. We'll mark that one as an exhibit.

(Exhibit 4 marked.)

A "I have been asked to evaluate the allegation that Mr. Zendejas' atypical CML was caused by gasoline, diesel, or other chemical exposure, including benzene."

Q Okay. As we sit here today, do you understand what disease Mr. Zendejas has?

A I think I do, yes.

Q What does he have?

A I think he has atypical CML.

Q As we sit here today?

A Well, as we sit here today, it went -- as we sit here today, I think he's in remission. But it went through a blast crisis.

Q Okay. Did you review the deposition of

Dr. Carroll, who is -- who was Raul's main treating doctor?

A Yes.

Q Did you read the deposition of Dr. Gore in this case?

A Yes.

Q You understand Dr. Gore is a world renowned hematologist oncologist at Hopkins? Do you understand that?

A No.

MR. PERRY: Object to form.

Q (By Mr. Patton) Are you a hematologist or an oncologist?

A No.

Q Have you ever diagnosed anybody with a type of leukemia?

A No, sir.

Q Have you ever saw someone in a clinic or looked at pathological findings and said, Hey, you have this specific type of leukemia? Have you ever done that with a patient or person?

A Well, I did that a lot through my training, but it was not the formal diagnosis that went on people's charts.

Q Have you ever formally diagnosed anyone

with any type of leukemia?

A No.

Q Okay. Dr. Carroll, do you understand he held some type of leadership position at the Arizona -- University of Arizona Cancer Center? Do you understand that?

A No.

Q In his deposition he says that Raul Zendejas went through atypical CML, ultimately was treated, and then came back with AML. He calls it AML. Did you see that in his deposition?

A I recall that.

Q Okay. Dr. Carroll is a bone marrow transplant doctor, and he saves people's lives. And he saved Mr. Zendejas' life.

A I did see that, yes.

Q Okay. And he says that Raul had AML. As we sit here today, are you telling this judge and this jury that Raul did not have AML?

A Well, what I'm saying is that when you have CML first, atypical or otherwise, and it goes into blast crisis, that is usually what most hematologists will say, that he's in blast crisis. It can look like AML. It can be indistinguishable from AML, but it doesn't always look like AML.

Sometimes it's a lymphoid malignancy. Whether or not he called it AML, I'm calling it blast crisis, it was an acute manifestation of his disease. So it was, clearly, an acute myeloid disease.

Q He's got AML, right?

MR. PERRY: Object to form.

A He would have AML if there wasn't the preceding process. With the preceding process, most hematologists in the field would describe that as blast crisis.

Q (By Mr. Patton) Can you hold up this notebook and show it to the camera.

A (Deponent complied.)

Q Okay. Can you hold up this notebook and show it to the camera.

A I think they probably saw it when you handed it to me. Okay.

Q Can you hold up this notebook and show it to the camera.

A Okay.

Q Doctor, all these papers in front of you, these are some of the studies that you're relying on in giving your opinions in this case, correct?

A Yes.

Q Is it your opinion in this case that

benzene did not cause Mr. Zendejas' form of leukemia?

A Yes.

Q Okay. And are you testifying that based on all this literature, you therefore feel that benzene played zero role in Raul's leukemia? Is that your testimony?

A Well, I don't believe the scientific literature supports that benzene can cause atypical CML, A. B, I don't believe Mr. Zendejas ever used pure benzene. So the literature that's far more appropriate to evaluate a causal relationship with Mr. Zendejas is gasoline because that is ostensibly the thing that he was the most exposed to. And I think that literature is pretty clear that gasoline exposure does not cause CML or AML.

Q So your testimony is yeah, he has leukemia, but he doesn't have AML, right?

A Well, now you're asking a different question.

Q I am.

A Okay. My testimony is that he has atypical CML. And when chronic myelogenous leukemias progress, they will frequently go into what is usually termed blast crisis, which is an acute manifestation of their disease.

Q All these studies that you just showed the camera, did any of those people in those studies with leukemia go through an acute blast crisis before they arrived at their AML?

A You would have to go through the studies specifically to answer that question.

Q Have you went through any of these studies to determine if some of these patients -- before they were classified as an AML in the study, to determine whether or not they went through an MDS phase or they went through a CML phase or an atypical CML phase? Have you done that evaluation?

A Yes.

Q And what did you find?

A I didn't see any evidence of that. I mean, CML -- the only way --

Q Hold on. Hold on.

MR. PERRY: Let him finish.

A They only way that could occur is that someone does not ever get -- seek medical treatment for the first part of that disease. So they walk around with CML for however long it takes, their spleen keeps getting bigger, their white count keeps increasing. They ignore all that symptomatology until it goes into blast crisis, and then they show

up and say, Yeah, you're in blast crisis or, you have AML.

So I guess theoretically, if you took a study that was just purely a retrospective mortality study and all they did was look at death certificates and they said, okay, this person died from AML, there's no way to know what happened prior to that diagnosis. But that is not -- or prior to what the funeral director -- or whoever filled out the death certificate, because those things can be fairly inaccurate. But many of the studies, that's not the way they work. I mean, they knew the person. They could do a work history on the person. There's no evidence that that person had a chronic myeloproliferative disorder before they developed AML.

Q (By Mr. Patton) Do you believe that any of the folks with AML in any of these studies in front of you ever experienced a myeloproliferative disease or form of MDS or CML before they were designated in the study as AML?

MR. PERRY: Object to form.

A Ask that again.

Q (By Mr. Patton) Do you believe that any of the individuals in any of these studies who were

called persons with AML, do you believe, as a scientist, that none of them went through phases where they had atypical CML or MDS or myeloproliferative disease or aplastic anemia or anything else?

A There's no evidence that that occurred, but I can't say unequivocally that it has not.

Q Sir, as a scientist you would respect and acknowledge and you would agree with me that it is certainly possible, if not likely, that these folks with AML, they had other specific disease characteristics and disease precursors, other forms of myeloid diseases before they were called AMLs in a study?

MR. PERRY: Object to form.

Q (By Mr. Patton) Would you agree with that?

A No.

THE REPORTER: Excuse me. You need to slow down a little bit. I have you saying, "Object to form" and then "No." Give him a little time.

THE DEPONENT: Sure, you bet. Sorry.

MR. PERRY: Object to form.

Q (By Mr. Patton) Let me ask --

A No --

Q -- the question again, Doctor.
A -- because many times AML is diagnosed de novo. Sometimes there is a -- sometimes there is a progression. I'm not arguing that the progression can occur. And frequently people that are diagnosed with myelodysplastic syndrome, based on the form, some percentage will convert to AML. But I think those people will show up in the studies as MDSs.
Q Why do you believe that?
A Because that's when they would have gone to the doctor, and that's when they would have been pulled into these studies as a myelodysplasia.
Q So let's say somebody goes into the study as an MDS. The study's published. When the person goes back to the doctor two months later, they're diagnosed with AML.
A That's possible.
Q Do the study authors ever go back to the study and redraft and say, Hey, by the way, you know, we just looked at these people who were subjects of the study. They're not MDSs anymore. They're AMLs. Has that ever happened?
A I don't think it's needed.
Q Okay. What about CML? Would you agree that some patients get CML and then they end up with

AML?

A Well, they end up in blast crisis. Almost all of them will end up in blast crisis, I mean, unless they get cured or with a bone marrow transplant and you end the disease process. But that is historically the way people with CML die. They went with their CML up to the point where they went into blast crisis; AML, if you want, fine. With acute manifestation, blast cells pour out of the marrow into the periphery, and then that ends up being insurmountable. They don't survive that.

Q Most people with CML, their CML eventually evolves into a blast crisis or an AML?

MR. PERRY: Object to form.

Q (By Mr. Patton) Right? Isn't that what you just said?

A Historically.

Q If a patient in the study like a mortality study where you're looking at death certificates, okay, if they're called an AML on their death certificate, they're designated as an AML in the mortality study, correct? It's a mortality study. It looks at death certificates, right?

A Well, some only look at death certificates. Some may be more involved. They may

have hospital records. They may have interviews with treaters. I mean, there are -- there is a spectrum of specificity with how epidemiologists go about doing the studies. Some, yes, just look purely at death certificates or cancer registry data. And then they really -- there's no way to know what happened before. But that's not the case in all of them.

Q Had Raul Zendejas died during his second bone marrow transplant, do you know what he would have been called on a death certificate?

MR. PERRY: Object to form.

A I would assume that he would have been called atypical CML.

Q (By Mr. Patton) He could have been called blast crisis, though, correct?

A They might have put that in there, yes.

Q And they might have put AML on there, correct?

A That, I don't know.

Q Would they be wrong if they put AML or blast crisis on there?

MR. PERRY: Object to form.

A They wouldn't be wrong if they put blast crisis. I think if they listed AML, that would really not be enough information to understand what

Mr. Zendejas actually went through.

Q (By Mr. Patton) Consistent with your other experience in industry, would you agree with me that it is easier for you to testify on behalf of industry in this case because Raul didn't die? He's not called an AML; therefore you can call him an atypical CML and stratify him out of the literature? Is that accurate?

MR. PERRY: Hold on. Objection; form, unfounded, argumentative. Go ahead.

A No, no. I mean, because he had atypical CMA, that brings in a different body of literature that is appropriate and relevant to discuss. And most of that is in this binder, the CML literature. But he was exposed to gasoline and diesel fuel. Gasoline is not considered to be a carcinogen by any regulatory body. Neither is diesel fuel.

So whether he had AML or blast crisis or CML or myelodysplasia or any other myeloid disease that you would like to categorize him as, I do not believe that gasoline -- that there is sufficient literature for the gasoline to have caused that malignancy. So no, what you're saying is incorrect. The CML brings in a different body of literature. If it were a pure AML case, no one ever said that he had

CML, it was just straight AML, then that would be a different body of literature. But it would still be related to gasoline.

Q (By Mr. Patton) Some of the citations or the references in support of your report talk about refinery workers and talk about benzene exposure and talk about leukemia, correct?

A I would have to look. I suspect that's true, yes. I mean, I printed out a few refinery worker studies.

Q Are those guys exposed to all benzene all the time? Is it all 100 percent benzene?

A Not at all.

Q What do they make in refineries? Do you know?

MR. PERRY: Object to form.

A I think that depends on the refinery.

Q (By Mr. Patton) Would you agree with me that some oil refineries make gasoline?

A That's a good question. I don't -- I think some might. I actually thought they all did. But I was informed just recently at the Munich meeting, talking to a toxicologist for a refinery, and he said they don't make gasoline. So I thought they all did, but apparently that's not true.

Q The studies that connect -- first of all, the Pliofilm cohort, you talked about you're currently doing a re-revaluation of the Pliofilm cohort, correct?

A Kind of tongue in cheek because it's been evaluated to death, but yes.

Q Okay. So the Pliofilm cohort, what was that?

A The Pliofilm cohort is the most important cohort in the U.S. with regard to benzene exposures and the risk of AML.

Q Who performed that study?

A Which study?

Q The Pliofilm cohort.

A The first one?

Q Yes.

A I mean, there's like 30.

Q Dr. Infante was involved in that, correct?

A Peter Infante was the first person who responded from NIOSH to Dr. Sakol's observations that we're seeing too many of these erythroleukemias in this population. But the first quantitative study, the first quantitative epi, was not done by Dr. Infante. It was done by Dr. Rinsky.

Q In any event, the Pliofilm cohort helped

lead to the increased -- or to the new benzene standard in the late '70s, correct?

A That is my understanding, yes.

Q It's my understanding, too.

And is Pliofilm benzene?

A Pliofilm is rubber hydrochloride.

Q It doesn't -- it's not pure benzene, is it?

A Well, Pliofilm, that's a thing. That's a material. And it's kind of like Saran Wrap. When they were making this Saran Wrap, thick Saran Wrap, which is called Pliofilm, which is rubber hydrochloride, they used pure benzene as a solvent. So they dissolved this stuff in pure benzene. They spread this out on this big table in pure benzene. They allowed the benzene to evaporate up. They tried to collect it overhead in tanks. That's why it gives us such good data on the benzene/AML association.

Q But weren't the folks who were making that Pliofilm, weren't they exposed to other chemicals besides just the benzene?

A No. That's the reason why the US EPA, OSHA, all these people, all the regulatory groups in the US rely on Pliofilm to set our regulatory standards, because there was not a lot of confounding

chemical exposures in those workers. And we had a reasonable dose response. So we knew some people were dry side workers. They weren't exposed very much. There were wet side workers, five or six job categories. They had varying levels of exposures so it allowed us to do a pretty detailed dose response analysis.

Q You bring up dose response, which is an interesting point. Does everyone respond the same way to the same dose of a given substance?

MR. PERRY: Object to form.

A It depends on the substance. It depends on the dose. I mean, I think what you're asking me is, is there individual susceptible?

Q (By Mr. Patton) Yes, sir.

A And the answer is, or course.

Q Of course there is individual susceptible?

A Yes. Now, whether or not that impacts on any observation that you might see epidemiologically or pharmacologically or toxicologically, that's going to be a discussion for the chemical and the end point and what we know about that scenario. But yes, as a general rule, we all respond differently to different chemicals.

Q Because we all respond differently to

different chemicals, and specifically to benzene, would you agree with me that different workers will respond differently to benzene?

MR. PERRY: Object to form.

A I was talking more generally.

Q (By Mr. Patton) I want to talk a little more specifically.

A About benzene specifically?

Q Yes, sir. Will you agree with me when we're talking about benzene that all workers respond to benzene in the same way (sic)?

MR. PERRY: Object to form.

A It depends on the end point.

Q (By Mr. Patton) What do you mean, "end point"?

A Well, like we know that people who were exposed to thousands of part per million in a short term benzene suffer CNS, depression, and they lose consciousness and pass out. There doesn't appear -- I mean, there's not many people that have ever been exposed like that, but there doesn't appear to be a clear-cut distinction in one person versus another. So I think there is no evidence to say that there would be individual susceptibility with regard to that end point. So that's what I meant by end point.

Now, if you're talking about cytopenias or you're talking about aplastic anemia, then that's a different question.

Q If benzene is going to hurt someone, what organ of the body would it hurt?

MR. PERRY: Object to form.

A That would depend on the dose.

Q (By Mr. Patton) Does benzene --

A It would depend on the exposure duration.

MR. PATTON: Objection, nonresponsive.

Q (By Mr. Patton) Does benzene affect the bone marrow?

A It can.

Q It can. And what does it do to the bone marrow?

A That depends on the dose.

Q Okay. And what dose does it take of benzene to affect the bone marrow, to create a harmful effect on the bone marrow?

A In experimental animals, it depends -- you know, I could quibble. It would depend on your definition of a harmful effect. But in experimental animals, you know, the levels were pretty high, 300 PPM, five days a week, et cetera, and then you saw pretty severe toxicity in their bone marrow.

More subtle changes you could see at about 10 part per million.

And as far as people go, if that's the end point, which most people would agree is the most sensitive end point for benzene toxicity, that being a cytopenia, it appears to be happening somewhere between, say, 5 and 10 part per million.

Q Okay. So let's kind of start over based on what you started to explain. Benzene can affect the bone marrow, correct?

A Yes.

Q Okay. And benzene can create harmful effects on the bone marrow, right?

A It can if it's high enough and long enough.

Q Sure. And benzene can cause -- you said cytopenia?

A Correct.

Q What is cytopenia?

A The penia means there's too few. So a cyto is a reduction in cells. So you can have a lymphocytopenia, which would be something you would measure in the peripheral circulation, and you would see a decrease in the lymphocytes. And EPA's reference concentration, which is their regulatory

standard for noncancer end points, is based on lymphocytopenia. So according to the EPA and Nate Rothman's study, that is the most sensitive end point for humans exposed to benzene, lymphocytopenia.

Q And that is observed at what amount of benzene exposure?

A In that study it was 7.2 PPM --

Q Okay.

A -- over an eight-hour time weighted average.

Q Okay. So let's say I go work all day, and I'm loading gasoline. Let's say Raul Zendejas is working all day, and he's loading gasoline, okay? You agree with me that gasoline contains benzene, correct?

A Yes.

Q You agree with me that workers -- gasoline distribution workers can and sometimes are exposed to measurable amounts of benzene while performing certain loading and unloading operations? Do you agree with that?

MR. PERRY: Object to form.

A We can measure benzene in really low levels so we're all going to be exposed to measurable amounts of benzene.

Q (By Mr. Patton) Would you agree with me --

A So I'm not sure that's a good adjective to use.

Q Okay. Would you agree with me that gasoline distribution workers at least had the potential, if they're not, in fact, sometimes exposed to measurable amounts of benzene around or -- above, actually, background levels? Would you agree with that?

MR. PERRY: Object to form.

A For the period when they're loading, I think that seems to be borne out in the literature, yes.

Q (By Mr. Patton) Would you agree with me, Dr. Pyatt, that the literature tells us that gasoline distribution workers, when they're loading gasoline, they are exposed to measurable amounts of benzene above background levels, correct?

MR. PERRY: Object to form.

A Yes.

Q (By Mr. Patton) And they are sometimes or can sometimes be exposed to benzene above or close to or around the amount that is a PEL or a TLV, correct? Does the literature show us that?

MR. PERRY: TLV or PEL for benzene or gasoline?

MR. PATTON: Benzene.

MR. PERRY: Because there's one for both.

MR. PATTON: Benzene, sir.

MR. PERRY: Object to form, unfounded.

A That, you've exceeded my knowledge of the industrial hygiene for distribution workers.

MR. PATTON: Let's take a break. Off the record.

THE VIDEOGRAPHER: The time is 10:28. We're going off the record.

(Recess from 10:28 a.m. to 10:39 a.m.)

THE VIDEOGRAPHER: We're on the record at 10:39. This is the beginning of Tape 2.

Q (By Mr. Patton) Dr. Pyatt, must someone be exposed to pure benzene to be exposed to benzene?

A No.

Q Okay. Must I be exposed -- well, first of all, alcohol can make someone intoxicated, right?

A Ethanol. I mean, there's lots of kinds of alcohol.

Q Okay. But as far as --

A Ethanol alcohol can.

Q Okay. If somebody's going to go out to

dinner and have a couple cocktails, okay, that can cause intoxication, right?

A That's true.

Q What is it in the cocktails that causes intoxication?

A Ethanol.

Q Ethanol, okay. So must someone be exposed to pure ethanol to be intoxicated, or can the ethanol be diluted with other products like in a cocktail to make someone intoxicated?

A Both.

Q Okay. But you would agree with me that someone who is exposed to alcohol or ethanol in a cocktail is ultimately being exposed to alcohol or ethanol, correct?

A Yes.

Q The fact that the alcohol or ethanol is in the cocktail doesn't change the fact that they're still exposed, right?

A No.

Q Okay. You talked about individual susceptibility and dose response. Is there a dose of alcohol or ethanol in cocktails that causes intoxication?

A You'll have to define "intoxication."

Q Well, does it -- my point, sir, is doesn't that differ among the individuals?

A It does down at the low end. I'm not sure that it does up at the high end. I mean, people -- if you drink enough alcohol and you go into a coma and respiratory arrest, I don't know that that happens at different levels. But certainly down at the low end of that dose response, the Asian population, they have this so-called flushing reaction. And they get a profound metabolic change at really, really low levels of ethanol that I don't get. So there are some differences. That's what I mean. You just have to define what you mean by "intoxication."

Q Okay. Well, let's take, for example, drunk driving, okay?

A Okay.

Q The blood alcohol content I think in most of the U.S. is .8; is that right?

A I thought it was 1.0, but maybe it has dropped. I don't know.

Q Let's say it's 1.0.

A Okay.

Q How much alcohol must someone drink to reach a 1.0 blood alcohol level such that they are

drunk, according to the police?

A I don't know the answer to that. It would depend on like what they ate prior, their size, the content, how quickly they did it. I mean, there's a lot of variables, and there are charts that can predict that. But I'm -- I don't know what those are.

Q So different folks are going to -- their blood alcohol level is going to respond differently or arrive at a different number depending on various factors, correct?

A That I think is fair, yes.

Q Okay. If someone's exposed to benzene, okay, would you agree with me that there are different various factors that would affect that person's uptake or metabolic effect, so to speak, of benzene? Or is it just uniform across the board?

A Well, there's a lot involved in that. The uptake -- which I'm assuming you man absorption?

Q Yes, sir.

A There doesn't appear to be any differences in absorption.

Q Is it your opinion, Doctor, that all people absorb benzene at the same rate?

A There doesn't appear to be much

difference. So, I mean, you're going to have differences in breathing rates. So differences in breathing rates will affect the absorption. But on a given amount of benzene that's in the lungs, there doesn't appear to be differences in the ability of a molecule of benzene to cross those membranes and get into the blood. That's absorption. So that, there doesn't appear to be -- or I certainly can't think of a biological reason why that would be different.

The next part of your question, the metabolic, I think -- what did you say? Behavior?

Q Let me stop you real quick, and then I want you to start with the metabolic --

A Sure.

Q For absorption of benzene purposes, is it your opinion that all human beings absorb benzene at the same rate regardless of race, age, gender, weight, what they had for breakfast, things of that nature?

A We're talking about inhalation, breathing --

Q Sure.

A -- breathing it in? I've not seen any information that would indicate that people absorb benzene differently. The amount of benzene that you

can pull into your lungs, which would then dictate how much gets into your -- the bloodstream, that could vary. Children breathe quicker. Workers working harder, they're breathing in more air. So those things can change.

But, I mean, if you said here's 100 people, here's 100 molecules of benzene into each one of those people's lungs, how much of that 100 molecules of benzene are to get into each of those people's bloodstreams, I've never seen any data to indicate that there would be any difference in that.

Q Okay. Is it your opinion that the literature says -- does, in fact, support your opinion, that the benzene absorption rate is the same for pretty much everyone across the board?

A I've never seen anything contrary to that.

Q Okay. So the next step is metabolism of benzene?

A Correct.

Q Okay. What is metabolism of benzene when it's affecting the body?

A The first thing that benzene's going to do -- and it would be irrespective of the route of administration, whether it's inhalation or

whatever -- it goes to the liver. And it is oxidized by a variety of enzymes in the liver, and it produces a variety of metabolites that I can describe for you, if you care. But some of those are excreted right away. Some are exhaled. Benzene is exhaled right away so it doesn't get metabolized. You know, kind of like ethanol goes right back out, and people can detect it in these Breathalyzer tests. So benzene does the same thing.

Some of the metabolites that are formed in the liver then travel through the bloodstream to the bone marrow where additional metabolic processes take place. And that is thought to be underlying some of the toxicity associated with benzene in the bone marrow.

Q Okay. Let's go through those in a little more detail. So someone absorbs benzene at a given rate that is shown to be clear, across the board, right?

A There hasn't been information that I'm aware of that shows that it's different.

Q I'm not challenging you on that.

A Well, yeah. I just want to make sure. I'm not saying that everyone -- that we know definitively, but that there's not been any evidence

to show that it's different.

Q Okay. And then benzene is being metabolized by the body. That means that the body's taking it in, kind of like someone drinking a cocktail is metabolizing the alcohol; is that fair?

A It's exactly like that.

Q Okay. And so the benzene's being metabolized in the body. It goes through the kidneys or liver?

A The liver is the first part where it is metabolized, but the kidneys certainly play an important role in the excretion of those metabolites.

Q Got it. So the benzene's being processed through the liver. Some of it makes its way to the kidneys. People urinate it out, correct?

A Yes.

Q And then some of it makes its way to the lungs, and then you exhale it out, correct?

A More it's still in the lungs from breathing it in, but yes.

Q Okay.

A It comes back out of the blood, goes into the lungs, you exhale it, yes.

Q Got it. And then some of it goes to the bone marrow, correct?

A Correct.
Q Before we talk about the bone marrow, does some of it go anywhere else?
A Well, it's in the systemic circulation so it's going to go anywhere the blood's going.
Q Okay. So by that time, benzene has metabolized into the blood and into the bone marrow, correct?
A By that time, the metabolites have left the liver. Some of those have been conjugated, and they're being excreted. That's part of this process because that's how they get transported to the liver -- to the kidneys and throughout. Some of them are in transit anywhere that the blood is taking them. And then there are specific enzymes in the bone marrow that can take those metabolites and further metabolize them.
Q Okay. And so is there a uniform or a -- is it the same rate of benzene metabolism among everyone?
A No.
Q Okay. That differs?
A Absolutely.
Q Okay. A kid who weighs 20 pounds at infant age compared to a pro football linebacker,

they're going to metabolize benzene differently, right?

A Probably not; probably not.

Q Probably not?

A No.

Q Not differently?

A No.

Q Where are we missing each other?

A Well, I said that people can be different, and they can. Your question posed, is it because of age or because of weight. And those two things -- after a certain age, children express the same profile of enzymes that the pro football players are going to express on a per gram basis. And so they're going to be, on a per gram basis, equally -- they have an equal ability to metabolize benzene. So based on those two, on the information you provided, there's no evidence to think that they would handle it any differently.

Q Okay. What about men who are in their 20s and 30s and 40s, they have different diets, they're different weights, they're different races, are there similarities or are there differences in the metabolic rates of benzene for those individuals?

A There's not -- let's try to talk a little

more specifically. The first metabolic step and the most important step in the so-called bioactivation of benzene -- because benzene is not toxic. You understand that? It has to be metabolized to be toxic. Okay. That first metabolic step, that first step that it takes is catalyzed by an enzyme 2E1. It doesn't matter what it does, why it's doing it. But about 2E1 expression does not seem to change from 20 to 30 to 40.

Now, there are other enzymes in the process of how benzene is metabolized. It's a very complicated process. Some of those steps we have found. "We" being the scientific community -- that there are racial differences. African-American is not one of them, but there are some differences. Asian populations have a different expression of those enzymes and, as such, they will metabolize benzene -- potentially metabolize it differently. So it certainly can happen. Your examples just aren't good examples of where it would happen.

Q Okay. Do Asians metabolize benzene such that -- how do you explain it as far as more harmful or less harmful when comparing Asian to white Americans, let's say?

A There is an enzyme called NQ01, and NQ01

is something that is thought to be a detoxification step. So 2E1 is making toxic metabolites. NQ01 detoxifies those metabolites. If you have a mutation in NQ01, then you have a reduced ability to detoxify the metabolites.

Q Okay. So --

A As such --

Q Go ahead.

A -- then it's going to ostensibly be more toxic. That mutation is present in a higher percent of the Chinese population than it is in the U.S. population. So based on that one end point alone, you would say that Chinese people, the population, they have a reduced ability to detoxify benzene. So ostensibly they could be more susceptible to benzene toxicity.

Q Asian folks are more susceptible to the toxicity of benzene, correct?

A Chinese, if they have that mutation in that enzyme.

Q Depending on little -- depending on individual characteristics in their DNA?

A Sure.

Q Okay. Would you agree with me -- you said earlier that individual susceptibility varies,

correct.

A Yes.

Q Would you agree with me that individuals in the U.S., whether Hispanic or African-American or white, based on other biological factors, DNA, what have you, different people -- their ability to detoxify or get rid of the harmful benzene before it gets into the bone marrow, that's going to differ among people, right?

A You certainly see a kind of a dose response relationship anytime you look at populations.

Q My question isn't --

A I'm getting there.

Q I don't mean to cut you off. Okay.

A So this dose response relationship, say, with benzene toxicity, it is likely due to variations in these various enzymes that we're discussing. We don't know what all those are so I can't tell you definitively, yes, here is a profile of a person who is going to be more susceptible or less susceptible to benzene, with the exception of NQ01. That is one that we sort of have a pretty good handle on.

Q For Asian folks, for Chinese people?

A Well, it would be the same. They just

have a higher population -- they have a percentage of that mutation in their population. In the U.S. it's about 5 percent. So 5 percent of the U.S. population would possess that mutation and based on that one end point alone could be susceptible -- more susceptible to benzene toxicity.

Q Folks in the U.S. who have the NQ1 (sic) mutation are more susceptible to benzene toxicity, correct?

A Benzene toxicity.

Q Okay. And that makes up 5 percent of the U.S. population?

A That's correct.

Q How do you find out if someone has an NQ1 mutation?

A NQ01.

Q NQ01.

A That's fine; get some DNA and look for it.

Q And those people who do have that mutation, benzene is more likely to hurt them than the people without the mutation?

A I mean, there is a study that suggests that. It's not the strongest piece of evidence in the world. But I don't know many people who have done research on benzene that do not believe that

it's meaningful. But the degree of vulnerability does not appear to be very much. I mean, so there's a lot of caveats to it, but it does appear to hold true.

Q It has some merit?

A I think it does, yes.

Q Okay. When did this NQ01 thing first -- when was it first realized or understood by industry?

A By industry? I can't answer that.

Q Ten years --

A I can tell you --

Q -- ago? 20 years ago?

A -- it was first published.

THE REPORTER: Excuse me.

THE DEPONENT: Sorry.

Q (By Mr. Patton) What year was that first published?

A Probably the mid, early '90s is when people started recognizing the importance -- then it was called DT diaphorase, the same enzyme. People started recognizing that there were differences in DT diaphorase that impacted on this ability to handle chemotherapeutic drugs. And then there was a pretty important paper that was published in 1997 from research at the University of Chicago, '96, where

they reported that individuals that had this NQ01 mutation had a higher risk of developing leukemia secondary to chemotherapy.

Q And is that akin to developing leukemia secondary to benzene? Doesn't chemotherapy and benzene act similarly?

A That certainly would be debatable, depending on who you were asking, but I believe there's some -- there is some value in evaluating the chemotherapeutic literature with regard to understanding what benzene is doing.

And then there was one more piece. NCI researchers evaluated -- and this was '99 or 2000 where at high exposures, 30 PPM time weighted average and up, they saw an increased hematotoxicity associated with benzene exposure in those workers. So the final step, does this mutation actually increase a person's ability -- increase their risk of developing AML from benzene, that hasn't been done. But, you know, the pieces are all lining up, and I believe it's a legitimate theory.

Q So in the late '90s there were two studies, and they examined the role of this NQ01 as increasing someone's likelihood of receiving the harmful effects of benzene because they can't

detoxify it as quickly, correct?

A The first study was looking at the risk of secondary leukemia following chemotherapeutic agents, nothing to do with benzene.

Q Okay. And then the second study -- what was it, again?

A The second study was looking at hematotoxicity as evidenced by cytopenias, which we talked about earlier, and whether people that have this mutation, whether they have an increased susceptibility to this cytopenia. And at high exposures, it appeared that they did.

Q Okay. The combination of which increases someone's -- based on their genetic factors, it increases their likelihood of getting a secondary leukemia from benzene or even chemotherapy therapeutic agents, correct?

A The first part of that would be speculation; supportable, but still speculative. The second part of that, I think Richard Larson's group pretty convincingly demonstrated, and that was the chemotherapy. The benzene is speculative. The chemotherapy I think is pretty solid.

Q And you think because they act similarly enough it lends itself to being instructive on

benzene?

A I would think so, yes.

Q It's helpful to determine what folks might be extra susceptible to get a secondary leukemia from benzene exposure, true?

MR. PERRY: Object to form.

A Well, I mean, no. It wouldn't be helpful at all if there's no evidence that anyone is going to be exposed to the levels of benzene that can cause leukemia, so there would be no real value in knowing that. I don't even think -- you know, from an academic point of view, it was very interesting. But even knowing that in the clinic, you've got to treat these patients. So whether you think they have an increased risk of developing leukemia from your treatment or not, they're not not treating them with these chemotherapeutic drugs because they're going to die of the primary malignancy. So it's interesting academically, but I don't know that it has that much bearing.

Q Can you examine a person's breath and evaluate their level of benzene, the level of benzene being metabolized by their body?

A The level of benzene being metabolized?

Q Well, let me ask a better question.

A I don't know the answer to that.
Q If someone is drinking alcoholic beverages, they can take a Breathalyzer and you can evaluate the blood alcohol content of that person, correct?
A Yes.
Q Okay. Can you examine a person's breath and learn anything about benzene?
A You might be able to if you did it really, really quickly. But you blow off benzene really fast. So, you know, if you look in my breath today, you might can say what might be in this room or what I might have been exposed to when I went to the restroom, but you're not going to learn anything about what I was doing yesterday.
Q Okay. But if we took a snapshot of your breath yesterday, we could tell if you were being exposed to more benzene, correct.
A Perhaps.
Q Okay. So like today you're sitting in a hotel conference room. If we were to detect your breath, you certainly wouldn't -- would you -- or would you expect to find any appreciable amount of benzene?
MR. PERRY: Object to form.

A Yeah. I don't know what mean by -- you could measure it.

Q (By Mr. Patton) Okay.

A You certainly could measure it.

Q Okay. And then what if tomorrow you and I go work on a gasoline loading rack and we top-load without vapor recovery? Could we then evaluate our breath and determine how much benzene is being exhaled?

MR. PERRY: Object to form.

A Yes. You could certainly measure the benzene that would come out of a person's breath, no matter what they're doing.

Q (By Mr. Patton) Okay. But you're saying that it happens quickly, such that you have to take the snapshot kind of as it's happening?

A I don't actually know the kinetics so it wouldn't have to be instantaneous. But it's not going to stick around very well.

Q Okay. And then what about someone's blood? Can you evaluate people's blood to determine the benzene metabolism or benzene toxicity being experienced at a given moment in time?

A You asked two questions.

Q Can you answer them both?

A Not together. We actually believe now -- we've looked at urinary metabolites. We've looked at all these different things. They're not specific. There's problems with them. What seems to be the best way at this point to biomonitor people for their exposures is benzene in blood. Because if they have benzene in their blood, then it had to have come from benzene externally. If you see these metabolites in their urine, maybe it came from benzene. Maybe it came from the fact that they ate tuna fish the night before. So there's all kinds of problems interpreting it. So benzene in the blood does seem to be the best biomarker for -- you know, exposure marker with the caveat that it also is very quickly metabolized so you've got to do it fast.

Q Are there -- I don't want to say standards, but are there -- is there information out there indicating the importance of benzene levels when detected in the blood? Like how are they measured?

A That's a great question. The only -- the only chemical that is regulated today based on internal levels is lead. So they say, Okay, you've got too much lead in your blood. We need to kelate you. You need to get out of this apartment complex,

you know, whatever -- whatever the situation is.

All the other chemicals, benzene included, is regulated on external concentrations. So at this point we can't say definitively if you have this much benzene in your blood, you have been overexposed, or here's what your exposures were. All the regulations are external concentrations, how much benzene is in the room. Five years from now, 10 years from now, maybe it changes. We certainly understand the pharmacokinetics -- well, I wouldn't say that. We might at some point be able to understand the pharmacokinetics of benzene to be able to make that extrapolation, but right now we don't. We can't.

Q How did we --

A Sorry -- the scientific community.

Q I'm with you on that.

How did the scientific community ultimately arrive at those lead numbers?

A That's a good question. I don't know the answer. I mean, I would guess that it was through animal data and extensive epidemiological research and looking at various children exposed to these and doing these subtle cognitive tests.

I mean, at a certain height, a hundred micrograms per deciliter, people are sick. So, you

know, at those levels it's easy to say, You've got too much lead. We need to get it out of you.

At 10 micrograms per deciliter, you know, there's not going to be any overt manifestation of that so it's a much harder question to answer.

Q If the oil and gasoline producers or manufacturers and the API, if they wanted to determine the amounts of benzene in blood which correspond with certain levels of exposure, external exposure and safe limits and things of that nature, do you believe the chemical companies and the API have the amount of resources to figure that out?

MR. PERRY: Object to form.

A I'm not sure. I mean, it's not a matter of -- there is a lot of research going on right now on benzene metabolism. There's several people -- that's what we did in this Munich meeting. People from all over are researching benzene and its metabolism. So it's not -- it's just a very complicated -- it's a very hard question.

Q (By Mr. Patton) The scientific community at some point years ago decided to evaluate lead, and reached conclusions -- they performed studies and they reached conclusions, and they decided what a safe amount of exposure to lead was, right, or what

an acceptable amount of exposure is --

A That's --

Q -- in the blood?

A That's true.

Q Okay. Are you aware of any efforts by the oil and gasoline companies and/or the API to evaluate the amount of exposure or the amount of benzene in the blood to determine more acceptable or safer levels of exposure? Are you aware of any efforts by the API right now?

A Yes.

Q With that end goal?

A We're doing it.

Q Are you doing it with the end goal of increasing regulations?

A Well, we're doing it with the end goal of understanding the relationship between benzene and blood and what the external -- what a regulatory limit would look like. What we're doing is calculating what's called -- what we call a biomonitoring equivalent, looking at internal blood levels and how they would correlate with external concentrations.

Q And is it your position that that research is being done to find a regulatory limit? Is that

the purpose of the research?

A We have regulatory limits. We have all kinds of regulatory limits. We have environmental limits. We have occupational limits. There's all kinds of limits for benzene. What we're doing is trying to take those regulatory limits and say, okay, if a person was exposed to what EPA says is safe -- we're all exposed to benzene all the time, right? So if a person was exposed to what EPA says, this is okay, this is -- you're all right with these levels of benzene, how much benzene in the blood would you see. That's what we're trying to determine.

Q Okay.

A So then if you have benzene in the blood, then I could say, Okay, based on this calculation, EPA would say -- so we're doing exactly what you were asking with lead. The internal concentration of lead in blood correlates with an external exposure. That's exactly what we're doing with benzene.

Q Is industry funding that?

A Yes.

Q Okay. Did you -- would you anticipate that the results of all the scientific activity which industry is a part of, do you anticipate that industry would support lower levels of exposure to

benzene at the end of that research some?

MR. PERRY: Object to form.

A Would I -- would they support it?

Q (By Mr. Patton) Correct.

A Well --

MR. PERRY: Object to form.

A I'm trying to think. I don't see how what we're doing could ever --

Q (By Mr. Patton) Let me ask the question a little differently.

A Okay.

Q Let's say the scientific community reaches conclusions about the amount of benzene detectable when found in certain types of workers' blood.

A Okay.

Q And the scientific community says, Hey, these people are being overexposed, okay, and we can quibble about that term. But the scientific community says if someone has X amount of benzene in their blood, they're being exposed to too much benzene, okay?

A Yes.

Q Then let's say the scientific community went to the government and said, Hey, we suggest increased safety limits to better protect workers by

evaluating benzene in the blood. Okay. Are you following me?

A Uh-huh.

Q Would you anticipate that industry would be onboard with those stricter safety requirements, or would they oppose those stricter safety requirements, based on your experience with industry?

A I think that would depend 100 percent on the strength of the science that was presented on behalf of, you know, this scenario that you just described. I mean, if that science is unequivocal and we all agree, yeah, this is -- this is pretty solid, no, I don't think they would -- I don't think they would oppose it.

If it is not unequivocal and there are holes in it, they may not oppose it, but they may say, you know, we don't know that that's true, and we need to do these other things to fill up those data gaps.

Q And what did industry do in that regard in the late 1970s with benzene? Can you explain that process, how it kind of compares to the scenario I set forth?

MR. PERRY: Object to form.

A I can't. I wasn't around then. Sorry.

Q (By Mr. Patton) OSHA wanted to increase workers' safety by decreasing the acceptable amount of benzene exposure in the late 1970s, correct?

MR. PERRY: Object to form.

A That's my general understanding, yes.

Q (By Mr. Patton) You weren't around back then, but that's your understanding, right?

A Yes.

Q And industry opposed those stricter guidelines, correct?

A I think that's a simplistic view, but yes. I mean, ultimately they opposed it for, you know, whatever their scientific reasons were.

Q Okay. Did you know that part of the reason that they opposed it was because they were concerned about the gross national product? Did you know that?

A I did not.

MR. PERRY: Object to form. The Supreme Court of the United States opposed it, too, and rejected it.

Q (By Mr. Patton) I'll show you what I'll mark as Exhibit 5. Have you ever seen this document before?

(Exhibit 5 marked.)

A No.
Q (By Mr. Patton) Can you take a minute to read through it?
A The whole thing?
Q Well, if you want to read the whole thing, that's fine. But I want to point your attention to a couple portions.
A Yeah. If you're going to quiz me on it, I'd like to read it.
Q Okay.
A (Deponent examined exhibit.)
Q Actually, Doctor, I don't -- let me -- I'm not going to ask you a question. I want to give you a chance to read it.
A Okay.
Q But what I'd like to do is if I can give you that document and another document, and then we'll take -- and then I'll let you read them and then ask you some questions after that. Can we do that?
A Sure.
Q Okay. And I will show you what I will mark as Exhibit 6, and this is an article called "Benzene: an historical perspective on the American and European occupational setting." This is a study

by Dr. Infante. Are you familiar with this?

A No.

Q Have you ever seen that article before?

A No.

Q Okay. What I'd ask you to do is, I'd like to go off --

A This isn't an article. This is a book chapter.

Q Okay. What I'd like to do is go off the record, and I'd ask you to do two things. With Exhibit 5, the Shell Views paper, if you could read that.

A Okay.

Q And then with respect to Exhibit 6, if you can go through each paragraph -- and do you consider yourself an expert on benzene?

A I consider myself an expert on benzene toxicology --

Q Okay.

A -- not historical activities associated with the regulation of benzene.

Q Okay. Can you go through this Exhibit 6 and mark next to each paragraph agree, disagree, or no opinion? Can you do that for us?

A I'll try.

Q Okay. Let's go off --
A Each one of the paragraphs?
Q Yes, sir.
A Okay.
MR. PATTON: Let's go off the record.
THE VIDEOGRAPHER: We're off the record at 11:11.
(Recess from 11:11 a.m. to 11:48 a.m.)
THE VIDEOGRAPHER: We're back on the record at 11:48.
Q (By Mr. Patton) Dr. Pyatt, if you could take a look at Exhibit 5, turn to Page 6. Did you have a chance to read this document during the break?
A Yes. I read both of them.
Q Would you look at Page 6, please. It says 6 in the lower left-hand corner right there.
A Okay.
Q Do you see near the top in the left-hand corner --
A What am I looking for?
Q The paragraph beginning "Certainly." Do you see where it says, "Certainly we are not considering maintaining exposures that are clearly unsafe. Such risks are unacceptable and must be eliminated by reducing exposure drastically or, if

the risk cannot be sufficiently reduced, stop manufacture." Do you see that?

A I see that paragraph, yes.

Q And then you see it goes on there, and it talks about an example of the benzene case? And then if you look in the right-hand column near the bottom, the paragraph starting, "The industry's argument goes, if all of society's resources were allocated with the cost-ineffectiveness of the benzene standard, the total gross national product would soon be exhausted before making a dent in the vast array of health and safety problems confronting workers and others in our society."

This is a position by Shell. It's called a Shell Views, this document on the front page. Do you understand that?

A I do. I mean, you skipped over. You kind of picked and choosed (sic) what you wanted to read out loud. You skipped over the paragraph right underneath the "certainly" where it says, We are considering the fact that industries are disrupted and society loses in the long-term when no tangible benefits result from expenditures of large amounts of talent, time, and money. An example of this is the benzene case."

So what they were describing was that benzene was not related to the paragraph that you read, where clearly there was a health hazard and they were going to stop manufacturing. With regard to this other paragraph that you read down here, I don't -- I don't know what the GNP is. I don't know -- I mean, I can't comment on that.

Q Okay. Sir, I'm not implying that they should stop producing benzene or producing gasoline. But my question for you is based on this document, do you believe that Shell's view, as reflected on the front page, is in favor of increased safety or against increased safety, or do you not have an opinion, as a health professional?

A I think in 1980, which is when this document was produced, I read this, and I agreed with -- I agreed with it. I thought it was pretty well written, pretty well considered. I think they would be for increased safety.

Q You think Shell would be for increased safety --

A Correct.

Q -- even though they joined with the API in opposing increased safety?

MR. PERRY: Object to form.

A Well, that's your interpretation of that argument. And I don't know enough about it to say whether you're right or wrong. But you're interpreting that debate as to whether or not we should be reducing the acceptable amounts of benzene based on leukemia risks that went on in the late '70s and early '80s as these guys saying we don't care about safety. And what they said was, we're not convinced that the science supports that reducing the risks -- excuse me -- reducing the concentration is really going to make any difference in the leukemia risk.

Q (By Mr. Patton) As a toxicologist would you agree with me that the less people -- the less workers are exposed to benzene, the less likely they are to suffer leukemia as a result of benzene?

MR. PERRY: Object to form.

A No.

Q (By Mr. Patton) You don't agree with that?

A No.

Q So if the -- if the whole classification of -- let's say workers who work with benzene, okay? If all the benzene workers in the country, whether gasoline distribution workers or they are refinery

workers, if somehow, some way, they are exposed to less benzene, what effect do you think that would have on their risk of getting leukemia as a result of benzene?

A It depends on how you define "risk." It depends on what the concentrations are. If the concentrations for this population of individuals that are exposed to benzene are not going to increase the risk of leukemia to any meaningful degree, cutting it down by a factor of 10 or a factor of 100 or a factor of 10,000 isn't going to impact on the risk because it's not elevated because of those exposures. That's why I disagree with that.

Q Do you believe people who work with and around benzene in refineries have an increased risk of getting leukemia, yes or no?

A I can't answer that yes or no.

Q Do you believe that folks who are gasoline distribution workers who are exposed to benzene on a daily basis have an increased risk of getting leukemia?

A Well, that one, there's actually studies that people have exactly answered that question. And the answer is no. They do not have a meaningful increase in their risk of leukemia in distribution

workers exposed to gasoline.

Q What do you mean, "meaningful increase"?

A Well, it depends on -- that's why I said it depends on your definition of "risk." If you're going to draw a line through zero and you're going to model benzene leukemia risk as a straight, linear line through zero, then by definition any increase in exposure is corresponding with an increased risk of leukemia. Which means if I eat a banana for breakfast, theoretically I now have an increased risk of leukemia from eating that banana because it has benzene in it. Well, I don't agree with that.

But if you are going to take that linear approach, then any exposure, whatever it comes from, Diet Coke, decaffeinated coffee, or loading gasoline into a tanker -- all of those are going to have an increased risk of leukemia just because of your model.

Q Occupationally speaking, do you believe that low benzene exposures cause blood effects?

MR. PERRY: Objection to form.

A Occupationally speaking, do I believe low concentrations of benzene cause blood effects? Okay. What do you mean by "low," and which blood effects are we talking about?

Q (By Mr. Patton) That's what I'm trying to understand. As a toxicologist, how would you define low level benzene exposure?

A We already talked about that. I thought we clarified it. What you're going to say is low or insignificant or toxicologically irrelevant or safe, however you want to describe it, is going to be solely dependent on the end point in question. I mean, what I consider low as far as neurological dysfunction and coma and respiratory distress and passing out, that's a very different level than what I would consider low with regard to cytopenias. So that question, the way you worded it, can't be answered.

Q Have you ever seen documents from -- well, first of all, were you provided documents from Shell in this case as far as actual Shell documents, memorandums, studies, memos, things like that discussing benzene and gasoline? Were you provided those?

A I was provided as part of the discovery response that had a long list of -- I guess these were lots of gasoline produced, tanks of gasoline produced. I don't know exactly what they were. But it listed all of the benzene content of all of that

gasoline that Shell produced. And that was part of the discovery. That's the only thing that I have.

Q Sir, I'll represent to you that some of the documents produced by Shell in this case talk about prolonged exposure to low levels of benzene. Have you ever heard that phrase before, prolonged exposure to low levels or low concentrations of benzene? Have you ever heard that phrase?

A I've heard all those words, sure.

Q Okay. Have you heard the phrase?

A All the words put together, prolonged -- yeah, sure.

Q Okay. Do you know what that means?

MR. PERRY: Object to form.

Q (By Mr. Patton) What is meant by the word "prolonged" in that phrase, and what is meant by the term "low levels of exposure"?

MR. PERRY: Object to form.

A No.

Q (By Mr. Patton) Have you ever asked Shell what they meant by prolonged exposure to low levels of benzene?

A I have had no reason to do that.

Q Do you believe there's an increased risk for AML in persons exposed to more than 20 part per

million years cumulative dose exposure in the workplace?

A I don't believe there's an increased risk of AML at those exposures to gasoline.

MR. PATTON: Objection, nonresponsive.

MR. PERRY: You didn't specify. You didn't say "gasoline" or "benzene."

A You didn't. Sorry.

Q (By Mr. Patton) I know. I'll ask a new question.

A Okay.

Q Sir, do you believe there's an increased risk for AML in individuals exposed to greater than 20 part per million of benzene exposure?

A The way you worded it, yes. At something greater than 20 PPM years, there will be an increased risk. The critical question is at what level above 20 are you going to start seeing that increased risk. So I don't believe that 21 will get you there, but somewhere above 20, yes, you're going to have an increased risk of developing AML.

Q Okay. If someone -- can somebody be exposed to 20 part per million benzene by working in a -- 20 part per million cumulative dose of benzene from working in a refinery with pure benzene?

Is that plausible, that someone might be exposed at that level over their career?

MR. PERRY: Are we talking PPM years or PPM time weighted average?

Q (By Mr. Patton) Let me ask a different question. First of all, let me back up.

Have you seen -- do you have any -- have you seen any information in this case as to how much benzene Raul Zendejas was exposed to in the course of his work?

A The only thing that I've seen that was not just the published literature, but -- the only thing that I've seen is a report written by your -- by Stephen Petty.

Q Let me ask a more clear question. Have you seen any information in this case besides the report from Steve Petty which discusses specific to Raul Zendejas how much benzene he was exposed to?

A Well, I mean, we know -- we know -- "we" being the scientific community -- there are papers out there regarding what people that are loading trucks with gasoline, what their potential exposures could be. We know what the background exposures are. I mean, there's information, but I haven't seen anything specific to Mr. Zendejas.

Q Besides the report from Petty?
A Right.
Q As we sit here today, do you have any opinions on the range of exposure to benzene commonly experienced by gasoline distribution workers?
A That is outside of my area of expertise. But I will tell you that irrespective of what those end up being, the literature is pretty clear that those workers do not have an increased risk of leukemia. So whatever it is, it's going to be something less than a threshold amount where you're going to start seeing an increased risk of leukemia, AML, in the literature.
Q The report by Steve Petty indicates that Raul Zendejas was exposed to an estimated amount of 25 PPM year cumulative dose to benzene. Is that your understanding of his report?
A That's my understanding of his report.
Q Do you have any reason to disagree with his report?
A Yes.
Q What are those -- what are your opinions in that regard?
A Well, in order for that to have occurred, since most of that exposure that I could tell,

reading his report -- in order for that to have occurred, most of that is going to occur in the last two years of his career when he was driving a truck, which means that his time weighted average exposures would have had to have been 10 part per million over an eight-hour time weighted average.

That's unbelievable. That just is not scientifically defendable. He wouldn't be able to defend it. It's not defendable. Those levels of exposure are higher than most people working in the Pliofilm cohort where they were actually using pure benzene, 100 percent benzene, as a solvent, spreading it out on a table, letting it evaporate. Those people didn't have exposures like that, the vast majority of them. So to think that he did it from loading gasoline in a tanker truck three times a day, twice a day, however often he did it, is just not scientifically believable.

Then when you go and say, Okay, well, that clearly is exaggerated, why is it exaggerated? Well, the math I don't know. I mean, that's -- somebody else is going to deal with that. But he clearly has exaggerated on the amount of benzene that is in the gasoline. He assumes 2.4 percent. And the data that I looked at, that's incorrect. This was part of the

discovery so I'm assuming that it was available for everyone. I mean, I didn't see but just one or two where it was over 1 percent; most of the gasoline that was produced for this area, .7, .6, .5 percent benzene. So if it's .5 and he's estimating 2.5, there's a -- there's a 20 percent -- an 80 percent exaggeration of what it could possibly be.

Then he mistook -- I'm doing this from memory. But he read something that Mr. Horton said that he had done in his career, and he extrapolated and said that Mr. Zendejas had done that same thing, spilled gasoline on himself a hundred times. That's incorrect. And at least in one of the calculations that he had done, he did not account for the fact that Mr. Zendejas was not working with gasoline 100 percent of the time, but only -- I think by his own estimation, only 20 percent of the time, and the rest of it was diesel fuel.

So all of those things combined, plus whatever was going on in the Monte Carlo part that I didn't evaluate, it is a gross overestimation of what his likely exposures were.

Q Your opinion is that Dr. Petty's opinions are -- Steve Petty's opinions are a gross overestimate of Raul's exposure?

A Yes.
Q Do you have any opinions on what would be a fair estimate of Raul's exposure?
A Something much less than that. I mean, even if you just take the factors that I just told you, you know, you divide that by 5; okay, 5 PPM years, even that's really high. That's a lot. Over an eight-hour time weighted average, it takes a lot of benzene to get those numbers up. But even that I think is probably too high.
Q Have you seen any data from Shell estimating the amount of cumulative dose of benzene Raul may have experienced in his work?
A From Shell?
Q Yes, sir.
A I have not, no.
Q Have you seen any evidence or any documents from Shell of any air monitoring performed at facilities similar to the McNeece Brothers facility?
A That I couldn't tell you. I haven't seen it, but I think it exists.
Q Have any Shell experts provided you with any numbers or estimates of how much benzene Raul was exposed to in his work?

A Not at this point. I haven't specifically asked for that.
Q Are you an industrial hygienist?
A No, sir.
Q Have you ever performed an exposure analysis where you've estimated how much benzene someone is exposed to while working with gasoline?
A Yes. Actually, I have.
Q Tell us about that.
A Well, I mean, I was kind of on the sideline, but it was looking at what people using chainsaws -- tree trimmers using chainsaws with gasoline -- gasoline-driven chainsaws, what their personal exposures to benzene would be from using a gasoline-driven chainsaw. So we estimated it, they -- we calibrated pumps. We looked at the data. So yes, at least once, but I'm not an industrial hygienist. I don't -- I'm not pretending to be one.
Q Have you ever -- are you familiar with Dr. Neitzel?
A No.
Q Have you ever worked with him before?
A No. I'm not familiar with him.
Q Have you ever seen any of his work for this case?

A No.
Q Do you have any understanding of what he is going to -- what the purpose of his role in this case is?
A Mr. Perry said something to me about it yesterday, but I honestly don't remember, so no.
Q Dr. Ijazz Jamall, do you know him?
A I do not.
Q Have you ever worked with him before?
A No.
Q Have you seen any of his work in this case?
A No.
Q Do you have an understanding of what he -- the subject of his testimony will be?
A Yes.
Q What is that?
A My understanding is that he will talk -- kind of follow the same lines that he wrote in his paper about why gasoline is not just a vehicle for distributing benzene to people but it is its own unique chemical, and it has its own unique toxicology. And I think he's going to, on a more quantitative basis, evaluate Mr. Petty's report.
Q Have you seen any more specific

evaluations from Dr. Jamall as to Petty's report?

A I just understand that he's doing it.

Q Did you ask him for that information?

A I've never spoken with him.

Q What if Raul was exposed to 5 PPM years? What if based on your calculations you cut his benzene exposure from 25 to 5 PPM years? Would he still be at an increased risk of getting leukemia?

A Well, I mean, even if you accepted the first number, the 25 PPM years, which I don't, but even if you were to accept that number, that number of benzene doesn't cause CML. So even if we said, This is fine, this is okay, we agree with Mr. Petty, this is what his exposures were likely to be, there's clear literature that those exposures and exposures much higher than that does not increase one's risk of developing CML.

Q If I understand your opinions in this case, you're saying we're not talking about AML. You're choosing to talk about CML, correct?

MR. PERRY: Object to form. That's the medical diagnosis in the lawsuit. He didn't choose anything. That's the -- every medical doctor agreed the man had atypical CML.

Q (By Mr. Patton) Dr. Pyatt, in defending

industry in this case you have chosen to classify and analyze Dr. (sic) Raul Zendejas' disease as CML or atypical CML and not consider AML in forming your opinions, correct?

MR. PERRY: Object.

A Well, I always -- I mean, he didn't have AML. He had CML. And that was the consistent diagnosis your experts, our experts, the medical records -- everything says he had CML.

Q (By Mr. Patton) Would it be harder for you to defend this case on behalf of industry if it was AML, if it was clear AML?

A No.

MR. PERRY: Object to form.

Q (By Mr. Patton) Why not?

A Because gasoline doesn't cause leukemia of any kind, any flavor; AML, CML, ALL, CLL, it doesn't happen. So it doesn't matter from a gasoline point of view whether he had CML or AML or any other type of leukemia or myelodysplasia. Gasoline is not carcinogenic. No one thinks that it's carcinogenic: IARC, ATSDR, ACGIH, OSHA. None of these large regulatory bodies out there that have evaluated all of this science that was in this binder that you set in front of me a little while ago, none of those

groups think gasoline is carcinogenic.

Q Just because gasoline is not carcinogenic, does that mean that gasoline distribution workers who are exposed to higher levels of benzene in their work and work with gasoline all the time, does that mean that they just can't -- they can't get leukemia from benzene exposure? Is that your testimony?

A Well, my testimony is there's no evidence that they have.

Q Well, hold on a second. First of all, I'm with you that all these government groups, they say what they say, and what they -- they do not classify gasoline as a carcinogen. Okay. I understand that part. I think a jury will understand that part, too.

But what I'm trying to understand is if you're telling this jury that just because gasoline isn't designated as a carcinogen, that folks who work with gasoline on a daily basis under unsafe conditions where they are exposed to a tenfold amount of benzene than compared to other loading operations, are you telling the jury that there's no way they could get ben -- could get leukemia from benzene because gasoline's just not a carcinogen? Is that what you're telling us?

THE DEPONENT: I thought you were going to

object.

MR. PERRY: Objection.

Q (By Mr. Patton) You want him to object because that's a good question, isn't it?

A Well, there was just a whole lot of kind of stuff in there --

Q Well, he had 10 seconds that he could have --

A -- that we have to --

THE REPORTER: Excuse me. I need you to go one at a time.

THE DEPONENT: Sorry.

A I'm not a lawyer. Okay. Lots of things that you said I would have a problem with; whether or not that really represents an unsafe work environment, whether or not they're really -- when you say a tenfold increase, okay, well, tenfold, if the first exposure's really, really low and now you've multiplied it by 10, yeah, it's higher, but it still may be safe. So all of that, you know, I'm not going to agree with.

Your question I think, in essence is, is it possible for someone to get enough benzene exposure from gasoline to get leukemia?

Q (By Mr. Patton) That's my question.

A However you want to phrase it, is that possible? And the answer is, I don't know. But what I can tell you is, the scientific literature that has evaluated gasoline workers of all types and denominations, they do not support that an increased risk of leukemia or any other human cancer follows those exposures. That's why OSHA, ACGIH, all of these regulatory agencies do not regulate gasoline as a carcinogen.

If that wasn't true and under some extreme exposure conditions you could, in fact, get leukemia from gasoline, that would be represented in these studies, and then these agencies would not take that position.

Q Do you have any understanding of whether Raul's exposure to benzene from gasoline was, in fact, extreme conditions? Do you have any understanding of that one way or the other?

A He only did it for two years. It was in 2004, 2005, 2006. So we're not talking about historical exposures back in the '40s and '50s. I don't know what the numbers are.

Q Sir, the man worked from 2000 to 2006.

A Well, he was a truck driver from '04 to '06.

Q Did you -- did you read his deposition?
A I did.
Q Did you read the other depositions in the case?
A Yes.
Q Did you see where he testified that he loaded fuel before he was a driver?
A That's true.
Q Would you agree with me that he's exposed to benzene while loading fuel?
MR. PERRY: Object to form. It depends on the fuel.
MR. PATTON: On gasoline.
A Well, I mean, he's going to be -- there's going to be benzene exposure in any of these things, just like there's benzene exposure from drinking that Diet Coke, so that's a given. But yes, you're right, he did testify that during that four-year period -- that's fine. Six years.
Q (By Mr. Patton) Then why did you say two years earlier?
A Well, because I was thinking about being a truck driver and loading gasoline in those big tanker trucks. That's where -- that's where I thought your question was going.

Q Do you know that he was loading without vapor recovery?

A I've seen that. I don't -- I don't know enough about what that means in terms of his exposures.

Q Of any of the studies that you brought with you today, do they focus on the group of workers who top-loaded without vapor recovery in extreme heat? Do any of those studies focus on that?

A That, I couldn't answer.

Q Wouldn't that be the most relevant body of literature, folks who top-loaded gasoline without vapor recovery under the conditions that they did at small bulk terminals --

A Well, the only --

Q -- and their incidence of leukemia, rather than just looking at, oh, petrol station attendants? Well, Mr. Zendejas wasn't a station attendant, was he?

A Were you done?

Q Yes.

A Okay. No, he wasn't. But the answer to your question is, you only need to do that if you have -- you have some reason to believe that his exposures were so enormous that these studies aren't

going to include those. And even your own expert, Stephen Petty, that's not the case. I mean, his cumulative exposures to benzene are well within those that are represented in this binder of gasoline studies so he clearly was not somewhere so far out that these studies don't apply to him. Your own expert would agree with that.

MR. PATTON: Let's take a break and change the tape.

THE VIDEOGRAPHER: The time is 12:14. We're going off the record. This is the end of Tape 2.

(Recess from 12:14 p.m. to 12:20 p.m.)

THE VIDEOGRAPHER: We're back on the record at 12:20. This is the beginning of Tape 3.

Q (By Mr. Patton) Dr. Pyatt, as a toxicologist and when it comes to testifying in a case like this, do you believe you are biased?

MR. PERRY: Object to form.

A No.

Q (By Mr. Patton) Why not?

A Well, I mean, when I was being trained at the University of Colorado, all of the graduate students in toxicology -- we all knew that to one extent or another, all of us would be faced with

litigation as being part of our professional career, one way or another. And there was a course that several of the faculty taught called toxicology in the courtroom. And I was pretty early in my training, but in that course what they stressed over and over is that irrespective of who hires us, it is our job as toxicologists to give as fair a representation of the science as we can. So I'm not an advocate for you. I'm not an advocate for Mr. Perry. I am an advocate for the science as best as I understand it. And so no, I don't believe I'm biased. I believe the science supports what I'm saying completely.

Q What if you wanted to say the opposite, okay, that benzene did cause or contribute to Raul's leukemia, okay? There's two sides to every argument, right?

A I guess that's how the saying goes.

Q What arguments could you make and what references in the science and references in causation theory and so on and so forth could you point to that support the proposition that Raul's exposure to benzene did cause or contribute to his leukemia?

MR. PERRY: Object to form.

A That's hard. It's a really hard sell. I

mean, there are a lot of cases that I've seen, three or four that I've said, I can't really make a determination here. It's just too close for me to call. But this is not one of them. I mean, it's not a benzene case. It's a gasoline case. His exposures to benzene, wherever they came, were from gasoline. The gasoline literature is completely free of any risks of CML. And even AML, I don't believe you can get there. I don't know what studies you could pull out that would support this position.

Q (By Mr. Patton) Well, besides studies, let's just think of some arguments, okay? First of all, there is some showing that individuals' susceptibility varies and that some people would argue and some studies may show that there is no safe level of exposure to benzene. Do you agree with either of those principles?

A Well, we need to explore that a little. As we discussed earlier, I do believe -- and then provided you with at least one biologically plausible mechanism where that susceptibility could occur. I suspect there are others. We just haven't identified them.

Q Raul could have been more biologically susceptible to benzene exposure or to suffer leukemia

as a result; possible?

MR. PERRY: Objection.

A It is possible that he could have had an NQ01 mutation and thereby increasing his susceptibility to hematotoxicity. But the levels of benzene were really high in that one study so I don't even think that would necessarily hold up.

The other thing that -- the problem with the theory, as I understand your question, is that we now have -- we can explain to some extent this biological susceptibility: NQ01, glutathione transferase, 2E1, epoxy-hydrolase, myeloperoxidase, all these enzymes that have something to do with benzene metabolism and toxicity. So we can start to explain that. But those things are not new. NQ01 is not new. People in the Pliofilm had NQ01 mutations. People in the China study had NQ01 mutations.

So when we set a regulatory limit or when we say this is a safe dose or this is how much it takes in order to see toxicity, the people that are getting sick, that are exhibiting that toxicity, they are the susceptible people. So to then take this number of 40 PPM years or 100 PPM years or whatever and say, well, that's really not protecting a susceptible person, is it, I disagree. I think that

it is. I think that is the basis for those numbers.

MR. PATTON: Objection, nonresponsive.

Q (By Mr. Patton) Have you seen the Hayes study?

A Yes.

Q What does the Hayes study tell us about the amount of benzene shown to cause an increased risk of leukemia?

A It depends on the form of leukemia.

Q You're just going to stratify every question of mine right back out, aren't you?

A Well, we can get out the study. That's the way they do it. They stratify out -- there's no -- well, there is actually one category for leukemia. You're right.

Q Let's get the Hayes study out.

A Sure. This is the whole NCI series.

Q First of all, are there any criticisms that you have the Hayes study as a whole before you look to the certain pages of it?

A Well, you know, this was a complicated process that was ongoing before NCI, Richard Hayes, and these other guys -- before Martha Linet, before they got involved. The Academy -- Chinese Academy of Preventive Medicine had started this study in the

'80s. It was a mess. They came in, tried to make sense of this mess, but it was not an easy task. So before I list any criticisms, it's not of these investigators. It's that this was a really difficult task.

I think the epidemiology is credible as far as the disease end points, but I think the exposure levels are not credible. So to look at this and say this tells us something about how much benzene it takes to do things, I don't think that it's correct. And in fact, EPA agrees. OSHA agrees. Just about everyone agrees that this is not reliable enough from a quantitative point of view to use for risk assessment purposes or for, you know, regulatory limit setting. So that's my criticism.

Q Doctor, I think I can probably shorten your whole deposition here. And I'm going to try to -- try to see to the extent that I can work through it.

First of all, have you read Dr. Infante's deposition?

A No.

Q Why not?

A I don't think I have it.

Q Have you read any of his work in other

cases?

A Yes. I can't say I've read one specifically in a CML case.

Q Have you ever agreed with any of the opinions that Dr. Infante gives in a given case about benzene exposure causing someone's leukemia?

A Say that again.

Q Have you ever agreed with Dr. Infante's opinion that benzene exposure caused a certain person's certain type of leukemia? Have you ever agreed with that?

A That, I don't know. I mean, like I said, there have been some cases where I said, I can't really get involved here. I can't tell you one way or the other. And Dr. Infante may very well have been on the other side. I don't know. To the extent that I have seen his reports or depositions in cases that I've been retained in, no, we have not agreed.

Q Have you ever given the opinion in litigation that benzene caused someone's leukemia?

A No.

Q Have you ever given the opinion in litigation for industry that benzene contributed to someone's leukemia?

MR. PERRY: Object to form.

A I'm not even sure how that would work, so no.

Q (By Mr. Patton) Well, for instance, myeloid leukemia's caused by smoking, radiation, chemotherapy drugs, benzene, right?

A No.

Q You disagree with that?

A Yes.

Q Why do you disagree with that?

A Because myeloid leukemia includes chronic and acute. Chronic leukemia, all of those things that you just mentioned, none of those will work. Chemotherapy doesn't cause chronic leukemia. Cigarette smoking doesn't cause chronic leukemia, the other form of ionizing radiation -- ionizing radiation does so that's one. But the other ones that you mentioned, they don't form, cause, myeloid leukemia. They cause acute myeloid leukemia, not chronic.

Q Dr. Gore in this case, did you read his deposition?

A Yes.

Q What do you agree with in his deposition, and what do you disagree with?

MR. PERRY: Object to form.

A I can't answer -- I can't answer that. I mean --

Q (By Mr. Patton) Well, let me ask you in an easier way. Is there --

A I agree that he had CML. I agree that he went into blast crisis. That's what Dr. Gore said. He said he would call it blast crisis. I'd agree with that. I agree with what he said about treatment. I mean, there's lots of things that I agree with.

Q What do you disagree with?

A I disagree when he said that diesel fuel is a carcinogen. Okay. I'd like to see him prove that in a court of law, that diesel fuel is a carcinogen. So that's one thing I disagree with. I disagree when he says gasoline is a carcinogen. I disagree when he said that all of these things cause CML. No, they don't. So I disagree with the real pertinent issues of his opinion.

Q He gives the opinion that benzene caused Raul's form of leukemia, whatever you might choose to call it. You're not a hematologist, right?

A I am not.

Q Okay. So whatever it is that Dr. Gore chooses to call Raul's complicated disease process,

he concludes that benzene caused it. And you disagree with that, right?

A I do. He calls it the same thing I'm calling it, atypical CML that went into blast crisis. So I agree with him there. There's no disagreement on that front.

Q What about Dr. Carroll? He opined that benzene caused Raul's disease. Do you agree with that?

A He opined that he believed that his occupation had something to do with it. But when pushed for the literature supporting that, there really wasn't very much. And it looked to me like he was kind of backing off that.

Q I believe I saw the Adegoke study in some of your materials.

A Uh-huh. I have that.

Q Does that support plaintiffs' argument, or does that not support our argument in this case?

A Well, it depends on -- that one study shows an increased risk of CML for benzene exposure.

Q But Raul was exposed to gasoline, correct?

A That's correct. And this actually has a specific type for gasoline, and there is not an increased risk of CML for gasoline. So why would

anyone ignore the thing that he was really exposed to, which is gasoline, and focus on this other category which really is irrelevant for that study? That doesn't make sense to me.

Q Have you ever given your -- given the opinion in your career that exposure to any chemical for a certain person caused a certain resultant disease? Have you ever given that opinion in any context?

A Say that again.

Q Have you ever given the opinion in your career as a toxicologist that someone -- we'll call them Jim, that Jim's exposure to Chemical X or Substance X caused Disease Y? Have you ever given that opinion?

A Yes.

Q Can you give us some examples?

A There was a situation -- I mean, it wasn't a formal setting, but the guy had -- how did it work? He was having something done in his mouth, and they were rooting out a tooth with hydrochloric acid. And the little dam thing broke, and the hydrochloric acid that they were using on his tooth flooded his mouth, and then he said, I can't taste anything anymore.

Q That's pretty simple.

A Well, you asked for an example.
Q No. I --
A I'm giving you an example.
Q I'm not --
A I think --
THE REPORTER: Excuse me.
A I think the guy's hydrochloric acid exposure caused his loss of taste.
Q (By Mr. Patton) Okay. Give me some other examples where you've concluded that a man's or woman's exposure to Chemical or Substance X caused Disease Y.
A I'm trying to think of when I've been asked. That's the only time that I can think that anyone has specifically asked. Now, there have been some cases, examples, where people have called me and said, Will you take a look at this, and I say yes. I take a look at it, and they send me the information. And they call me back, and I say, I don't know. It -- maybe it did, maybe it didn't. I don't -- I cannot defend that it did not. And then so -- to my way of thinking I said, yeah, it could be. It could be. So that's happened several times in my career, but it did not happen in this case.
Q So if I understand your testimony,

you've -- besides the hydrochloric dentist example, you've never had an occasion as a toxicologist to conclude that someone's exposure to Chemical X caused -- a 51 percent chance, okay, more likely than not, caused Disease Y?

A I haven't. That's just not how my career has progressed. I mean, I was in the lab doing that kind of work, doing risk assessment work, consulting work. It just never has -- you know, I think maybe medical toxicologists or clinical toxicologists like Richard Dart at the Rocky Mountain Poison Control Center, he probably makes those determinations all the time. But I haven't ever been asked to.

Q How do you -- can you explain to the jury why you think it is fair for you to give the opinion in this case or as you have in 20 other cases where benzene or other chemical exposure didn't cause this person's certain type of leukemia or other disease when throughout your career you've never given opinions otherwise? How is that fair?

A Well, I disagree with that. I have given that opinion otherwise, and I think we just went over that. But what I would tell the jury is, they don't have to take my word for it. We're going to show them the science. We are going to show them what

ATSDR, what IARC, what all these people say about gasoline. I'm going to show them the studies where people have specifically looked at the kind of disease that Mr. Zendejas has and what exposures are or are not linked to that disease. They don't have to take my word for it. This -- this (indicating) speaks for itself. And that's what my job will be if this goes to trial, to explain to the jury here is this huge body of scientific evidence that does not support that Mr. Zendejas' disease was caused by his occupational exposures. That's what my job will be.

Q Do you feel good about your job?

A Yes.

Q Do you have any opinion as to what occupations outside of a refinery are exposed to the highest levels of benzene?

A Today?

Q Yes, sir.

A Working today, '09, no.

Q You don't have any opinions?

A I don't know.

Q Do you have any opinions as to what occupations do lend themselves to an increased risk of AML or another form of myeloid leukemia?

MR. PERRY: Object to form.

A Well, I don't think the occupation is really the question. So yes, the Pliofilm occupation, for some of those workers clearly increased their risk of developing AML. Some of the shoe workers clearly had an increased risk of developing AML. Rubber workers in some of the older studies clearly had an increased risk of developing AML.

MR. PATTON: Objection, nonresponsive.

Q (By Mr. Patton) My questions is, as we sit here today do you have an opinion as to what occupations lend themselves to the highest exposures to benzene? Do you have an opinion?

A It would be pretty speculative on my part.

Q Okay. Do you have an opinion as we sit here today what occupations that people could perform today lend themselves to the highest increased risk of development of AML?

A As associated with a benzene exposure?

Q Yes, sir.

A They would really be essentially the same question.

Q And you don't know?

A I'm not an industrial hygienist. I just can't really answer that.

Q Do you have an opinion as to what amount of exposure to benzene is necessary for it to be a causative factor in someone's AML?
A Yes.
Q Tell us that.
A I think the quantitative epidemiology supports 40 part per million years at the low end.
Q Okay. Must those people exposed to 40 PPM years be exposed to pure benzene?
A No.
Q They can be exposed to benzene as a constituent in other chemicals?
A Yes.
Q But not gasoline, right?
A Well --
Q Is that your opinion?
A I don't know that you could achieve those with gasoline, quite honestly, without the other chemicals in gasoline being acutely toxic and someone passing out or dying from it. But gasoline has its own set of epidemiology. So while gasoline has benzene in it, I don't think anyone is ever going to argue that it doesn't. It seems counterproductive to ignore this binder -- wherever it went -- this binder of gasoline studies and say, Well, we're going to

ignore all this, and we're going to ignore what all these federal agencies said about gasoline, and we're just going to focus on the benzene part of the equation. That doesn't make sense to me.

Q What does chronic alcoholism cause in the body? Do you know?

MR. PERRY: Object to form.

A It can cause cirrhosis of the liver as well as other, you know, nutritional problems and pernicious anemia.

Q (By Mr. Patton) If someone were to be diagnosed with cirrhosis of the liver and you found out that they weren't alcoholic, would you be able to give the opinion that their alcoholism caused or contributed to their cirrhosis?

A Say that again.

Q If someone were --

A Go ahead.

Q If someone were diagnosed with cirrhosis of the liver, okay, and you asked them what about their exposure to -- I'm sorry -- what about their drinking habits and their alcoholism, and they explained to you that they considered themselves an alcoholic and that they were drinking alcohol on a quite frequent basis, would you then draw the

conclusion or give the opinion 51 percent that their alcoholism caused or contributed to their cirrhosis? Would you give that opinion?

A Probably. But without having reviewed that literature -- I mean, I don't know if there's a clear-cut dose response relationship. I don't know. So I don't know whether there's -- you know, maybe someone can be an alcoholic their whole life but not consume enough alcohol to give them cirrhosis. I don't -- you know, the term "alcoholic," okay, that implies some kind of psychological dependency on the alcohol. Maybe that occurs at lower levels. I don't know the answer to that. But I do know that alcohol at sufficient quantities can cause cirrhosis of the liver, so certainly they have causation.

Q Okay. What if they told you that they were drinking only Jack Daniels?

A That wouldn't matter.

Q Why not?

A Well, because my understanding of the alcohol literature, which is very different than gasoline with benzene in it, but my understanding of the alcohol literature is that we can see that people who live in Germany and who drink beer, they get cirrhosis. We can see people that live in Italy and

drink mostly wine, they can get cirrhosis. We can see people in Russia that drink mostly vodka, they end up with cirrhosis. So it's not the water or the orange juice or the hops or the barley or the grapes. It's the ethanol that's doing it. That's a very different situation than the benzene in gasoline.

Q Can you tell us as we sit here what the range of benzene exposure estimated in these gasoline studies is?

A What the range of benzene -- no, not sitting here. I mean, it's in some of them. We could pull them out and look.

Q You just believe that Raul was not exposed to enough gasoline to cause significant benzene exposures that would cause his leukemia? Is that your opinion?

MR. PERRY: Object to form.

A Well, what I believe is the scientific literature does not support that gasoline exposure at any level causes CML.

Q (By Mr. Patton) Have you seen scientific literature that studies benzene exposure in the context of gasoline distribution workers at small bulk terminals?

A I think I have, but I would have to look

through the literature to see. I mean, there's -- Otto Wong looked at 18,000 distribution workers. Lesley Rushton looked at 20,000 distribution workers. Rob Schnatter looked at distribution workers. Sorahan looked at distribution workers. I've got to believe that a lot of those people that they pulled into those studies would fit into that category.

Q Are there any criticisms of the Wong study that you just mentioned?

A I think you can probably find criticisms of every paper.

Q Do you believe that the less a gasoline distribution worker is exposed to benzene, the safer he is? The better off he is?

MR. PERRY: Object to form.

A That the less a gasoline distribution worker is exposed to benzene, the better off he is?

Q (By Mr. Patton) Let me ask it in a different way.

A Okay.

Q Let's say we have two gasoline distribution terminals 10 miles away from each other, okay, and at one of the terminals the men are loading in -- they're loading with vapor recovery, okay. That's to mean they are exposed to fewer gasoline

hydrocarbon and benzene vapors than the guys working at Terminal No. 2, okay? At Terminal No. 2 they're top-loading without vapor recovery. They're being exposed to more benzene and hydrocarbon vapors. And I'll represent to you, sir, that the evidence from Shell in this case shows that the folks at Terminal No. 2 are exposed to more benzene, generally speaking.

A Okay.

Q Would it -- do you believe it's safer to work at Terminal 1 or safer to work at Terminal 2?

MR. PERRY: Objection.

Q (By Mr. Patton) From a general sense, can you give us any answer on that?

MR. PERRY: Objection to form.

A With the situation as you described it, there would be no way for me to answer it. We're all exposed to benzene. If the benzene exposures in this terminal over here, the one with the lack of vapor recovery, top-loading, whatever you said, if those exposures are not harmful and there's no evidence that those exposures matter from a toxicological point of view, then they're just as safe as these other people.

Q (By Mr. Patton) Right. That's what I

thought your answer would be. Your answer is that under no circumstances can gasoline -- can benzene exposure from gasoline cause someone's leukemia; is that your opinion?

A Well, my opinion -- and you've already asked that. My opinion is the scientific literature, as illustrated by this binder -- and I don't think there's any other one -- does not support that gasoline is a risk factor for leukemia.

Q Right. So it doesn't matter how much benzene the guys at Terminal 2 are being exposed to? It's just not a factor? That's your opinion, correct?

A That's what the literature would state, yes.

Q I'm asking you what the literature states, sir. I'm asking you your opinion in this case.

A But my opinion is based on the literature. They're not separate.

Q Then tell me that. Say my opinion is that under no circumstances can someone's exposure to benzene via gasoline be a factor in their leukemia. Can you tell me that?

MR. PERRY: Objection. He's already done it.

Go ahead and do it again.

A My opinion is, the scientific literature does not support that gasoline is an established risk factor for leukemia, period.

Q (By Mr. Patton) Is it your opinion, then, that under no circumstances can a worker's exposure to benzene from gasoline be a causative factor in his leukemia? That's my question.

A We've done this three times now.

Q And you keep going back to, Oh, I think the literature says this. I'm not asking you what the literature says, sir.

A Well, no.

MR. PERRY: Let me object, Keith. This is not where experts make up what they think. And maybe some of your guys have done that. This guy's based on the science and the literature. This is not a, come in and tell me what you believe. It's what does the scientific, medical literature support. That's what he's going to testify to. This is not the time for personal opinions.

MR. PATTON: Objection, nonresponsive.

MR. PERRY: Objection, nonresponsive.

Q (By Mr. Patton) Dr. Pyatt, is it --

A I'll try. I'll try. I mean, we did it

once before, and what I said --

Q Let me try to ask it differently. I understood what you said before, okay?

A Okay.

Q I just want to go through this, get this, and go to lunch, okay?

A Take as long as you need.

Q Is it your opinion that exposure to benzene above 40 PPM years can cause someone's AML?

A That's what the literature would support, yes.

Q Is it that -- is it therefore your opinion?

A My opinion is that that's what the literature supports, yes.

Q So if someone came to you with solid evidence that a worker was exposed to benzene above 40 PPM years, would you then be willing to give the opinion that that benzene exposure was a causative factor in his AML? Would you be willing to do that?

A Perhaps; other considerations, but perhaps.

Q Is it your opinion that the literature does not support benzene exposure via gasoline as a causative factor in AML? Is that your opinion, yes

or no?

A Yes. That is my opinion --

Q Okay.

A -- as based on the scientific literature.

Q Is it therefore your opinion that under no circumstances, based on the literature, could someone's exposure to benzene via gasoline, that whatever amounts they are, they could not be a causative factor in someone's AML?

A See, I can't answer that. I mean, I can't say what is possible, what's not possible. All I can tell you -- and we did talk about this. I said what you're really asking me is, is it possible for a person to get exposed to enough benzene to give them leukemia from gasoline. And I don't know that it is because, A, I don't believe that you can take the amount of benzene that a person gets from gasoline and just extrapolate it directly to someone being exposed to pure benzene. That's because of competitive inhibition and all there other chemicals going in. So that is an uncertainty.

Also, I don't know if someone is going to achieve, say, 40 or 50 PPM years of benzene, how much toluene are they going to have? How much n-hexane are they going to have? How much of these other

chemicals are they going to have? I don't know the answer to that. What I can tell you is the literature is clear that gasoline does not cause CML.

Q Does the literature say anything about gasoline causing AML?

A Oh, sure.

Q What does it say?

A It says it doesn't happen.

MR. PATTON: Okay. Off the record.

THE VIDEOGRAPHER: We're going off the record at 12:48.

(The deposition recessed at 12:48 p.m., to be reconvened at 1:30 p.m.)

AFTERNOON SESSION 1:40 p.m.

THE VIDEOGRAPHER: We're back on the record at 1:40.

EXAMINATION (continued)

BY MR. PATTON:

Q Dr. Pyatt, in your experience working with industry, has industry ever asked you to help improve nonindustry, meaning nonAPI member work sites or work environments based on what industry knows about the harmful effects of any chemicals?

MR. PERRY: Object to form.

A I can't think of anything that would fit into that category, no.

Q (By Mr. Patton) Aside from defending industry in litigation and aside from the studies that you mentioned earlier in your deposition, is there any work that you've done on behalf of industry, the API, which would directly help the working man?

MR. PERRY: Object to form.

A Well, I mean, I think you could take a fairly lofty approach to that question and say anything that we've done to understand the toxicity of benzene is to the benefit of the working man, public health. So all of the basic research that I

did and did with Dr. Irons and others, looking at the toxicity of benzene, I think that's useful information.

Q (By Mr. Patton) Have you ever done any work which resulted in more stringent workplace controls on benzene exposure?

A I don't know the answer to that.

Q Do you have any opinions in this case as to whether or not the Shell material safety data sheets -- do you have any opinions on those in this case?

A No.

Q Do you have any opinions on whether or not Shell adequately warned McNeece Brothers about the dangers of benzene? Do you have any opinions?

A No.

Q Do you have any opinions on whether or not Shell adequately warned Raul Zendejas about anything?

A No.

Q Do you have any opinion on whether or not gasoline distribution workers as a whole are adequately warned about the dangers of benzene?

A I have no opinion about that.

Q Do you think they should be warned about anything?

MR. PERRY: Object to form.

A Who?

Q (By Mr. Patton) Gasoline distribution workers.

A Who should warn them? I mean --

Q The manufacturers of the gasoline that they're working with. Do you have any opinions on whether or not the gasoline companies like Shell should even warn gasoline distribution workers about anything?

A Well, I'm not a warnings expert, and in a formal setting like this, no, I'd say I don't have an opinion about that. I would let others argue for or against whatever.

Q As a toxicology expert, do you have any opinions on whether or not gasoline is capable of causing any type of disease?

A In humans --

Q Yes.

A -- or in experimental animals?

Q Humans.

A Humans. The only -- the only toxicological -- well, yes. I mean, if you are exposed to a lot of gasoline, there are neurological symptoms. There is respiratory depression, which

really seems to be true for any organic solvent at very high exposure. So that's a toxic end point associated with gasoline that I think can happen.

Q How much is a lot?

A I think thousands of parts per million, but I'm not sure exactly where the cutoff would be.

Q Do you think gasoline is causally associated with any type of long-term disease process?

MR. PERRY: Object to form.

A The only one that's even been really speculated on or maybe, perhaps, probable, however they've worded with it for gasoline, is kidney cancer. And that's based on the male rat and the studies that have been conducted on these experimental animals. Whether or not that holds up under the epidemiology studies, I'm not prepared to talk about because I haven't evaluated that literature specifically lately. But I think that would be the one that -- I mean, when IARC says gasoline is a probable human carcinogen, that's what they're talking about.

Q (By Mr. Patton) So IARC has designated gasoline as a probable human carcinogen, right?

A That's correct.

Q Have you ever seen industry issue a questionnaire among any group of chemical workers in the United States? Have you ever been involved in any of that activity?

A No.

Q As we sit here today can tell me an example of any research that you know that Shell as done specific to gasoline distribution workers?

A I would have to look, but it seems like Sean Tsai has done some studies for Shell looking at hematopoietic effects in the refinery workers. And I would suspect there are some distribution workers that are included in what he did.

Q And he works for Shell?

A Uh-huh.

Q If he were to conclude as an employee or studying and working on behalf of Shell that there was, indeed, an increased risk, would you suspect that they would publish that information?

A I would suspect that they would, yes. I mean, it would depend on the nature of the study, how reliable the study was, but sure.

Q I mean, if API and/or Shell were to perform a study about gasoline and conclude that there was an increased risk, a statistically

significant increased risk of leukemia related to gasoline or benzene exposure, would you expect them to publish that study?

A It's hard for me to answer, but I think they would, yes.

Q Wouldn't it be incredibly detrimental to them because that study would then link benzene and gasoline exposure with leukemia, such that they could be held responsible?

A I don't know about all that, but I think they have an interest, as do all the rest of the companies and researchers, to publish their results and make sure that we all know what's going on with these exposures.

Q You would agree with me that if Shell were to perform a study and they were to conclude that there was an increased risk related to gasoline, whatever that risk might be, whatever disease process, they would have a problem because they could then be responsible for their product causing someone's disease?

MR. PERRY: Object to form.

A Your question is would they have -- would they be responsible, or would they -- I'm sorry. What was your question?

Q (By Mr. Patton) It's all right.
Do you require scientific certainty in terms of causality for every specific lymphohematopoietic disease related to exposure by benzene by exposure level?
MR. PERRY: Object to form.
A Are you asking whether -- what my criteria would be before I could say that benzene can cause a disease?
Q (By Mr. Patton) I'm saying would you require scientific certainty in terms of causality for every subtype of a leukemia?
A Of leukemia?
Q Of a given -- let me start again.
A Okay.
Q For you to testify that benzene caused someone's leukemia -- and again, you -- besides the dental example you've never testified or given the opinion that Chemical X caused Disease Y, correct?
MR. PERRY: Object to form.
A Well, like I said, there have been three or four times when I've had that opinion, and then they either settled the case or went somewhere else.
Q (By Mr. Patton) You didn't say that earlier, sir.

MR. PERRY: Yes, he did.

A Yes, I did.

Q (By Mr. Patton) Would you require scientific certainty in terms of causality for every specific subtype of myeloid leukemia related to benzene by exposure level before you would give an opinion on that causality?

A No.

Q When have you given the opinion to Shell or to industry that benzene did cause someone's disease?

A Well --

Q Because that's not what I understood you testified to this morning. The transcript will say it, but I just want to understand where our disconnect is.

A Like I said, there have been three times where they called me up and said, We'd like for you to help with this case. And the way I always do it is, okay, what are the issues, what's going on. Send me the medical records.

They did. I looked at all the exposure information they provided, talked to them. We had a follow-up call, and I said I can't -- I can't help you. I mean, I cannot say definitively that these

people's -- that this disease was not caused by their exposure. And that's happened three times that I can think of, sitting here.

Q What were the chemicals in question?

A Benzene.

Q What kind of exposure was it? Pure benzene?

A Working at a benzene unit, yes; pretty much, yes.

Q What were the diseases?

A The diseases were AML.

Q What kind of AML?

A That, I don't recall.

Q Wouldn't that matter to you?

A Not really. I mean, it would matter -- there is one subtype of AML called acute promyelocytic leukemia, which is the M3 designation, and that one is unique enough that it has had epidemiology studies conducted on it by itself. None of the other like erythroleukemias, none of the other subtypes of AML have had that extensive research done on them independently.

Q Do you have any opinions as to what McNeece Brothers should have done differently in this case?

A No.
Q Do you have any opinions as to what Raul should have done differently?
A No.
Q Do you have any criticisms of the way Raul worked with gasoline?
A Well, I mean, just kind of general. If what Stephen Petty said was true and he really did spill gasoline on himself 100 times while he's loading up tanks, I might say you might want to be a little more careful and pay attention to what you're doing.
Q But from a leukemia standpoint, if gasoline can't cause leukemia anyway --
A No, I agree.
Q -- what does it matter if he spills any on himself?
A I agree.
Q Okay. So is it your opinion that it doesn't matter how much Raul -- or how he was working with the gasoline or even if he spilled it on himself, because it still wouldn't cause his form of leukemia? Is that your opinion?
A That's what the literature would support, yes.

Q And therefore that is your opinion?
A Based on the literature, correct.
Q Why do you always qualify it, "based on the literature"?
A Because I want the jury to understand that my opinion is solidly based in the literature. This is not an authority based, look at me, I've got all these publications, I know this much about benzene. My opinions in this case are based on these binders of studies.
(Exhibit 7 marked.)
Q (By Mr. Patton) Let's talk about some of your studies. I've marked as Exhibit 7 a study about occupational hobby or lifestyle exposures associated with Philadelphia chromosome positive CML. Is that what Mr. Zendejas had?
A No.
Q Okay.
A But it was a lot closer to him than AML.
(Exhibit 8 marked.)
Q (By Mr. Patton) Exhibit 8, "Occupation and Leukemia Mortality Among Men in 16 States." Are you familiar with this study, Loomis?
A I can't tell from you holding it way over there. I mean, it was one of my papers that I

referenced in my report so I'm --

Q Does this study show all leukemias combined, there's an increased risk of all leukemias from workers in the petroleum industry?

A The relevant data in this study is on Page 512. I'll give it back to you. And no, it doesn't -- I mean, there is the CNLL which is the disease that Mr. Zendejas had.

Q Hold on a second. I thought you said he had CML?

A Same thing.

Q What is it?

A This is chronic nonlymphocytic leukemia. It's just a different epidemiological classification, but it is chronic myeloid leukemia. So there's petroleum refineries, they had no cases of CML so they couldn't do any math on it. Then there's motor mechanics and service station attendants, which would be relevant to gasoline, and the relative risk of CML was .5. So it actually would suggest that there would be a protective effect, based on that study.

MR. PERRY: Who's the lead author of that study?

THE DEPONENT: It was Loomis and Savit, I think, David Savit at UNC; is that right? Is it

David Savit?

MR. PATTON: Yes.

Q (By Mr. Patton) But this says only petroleum refining and rubber manufacturing had excess deaths for all leukemias combined.

A You know, the pertinent data is on that table. I'm not sure exactly what he's talking about. And you could say that something with a relative risk of 1.2, that there was an excess death. But does that mean that it is meaningful? That's where you have to start applying statistics and see whether it really is different or not.

(Exhibit 9 marked.)

Q (By Mr. Patton) The Jakobsson study, you're familiar with this one, correct?

A Yes.

Q This shows an increased risk of AML among petrol workers, correct?

A Correct.

Q Is Mr. Zendejas a petrol worker?

A Well, I think those were like gasoline station attendants. But I think it has relevance to the gasoline -- I mean, that is certainly one that I consider when I evaluate the gasoline literature.

Q Would you agree with me that someone who

bulk-loads gasoline has more exposure to benzene than a service station attendant? Or do you have an opinion either way?

A I don't have an opinion either way.

Q But nevertheless, your Exhibit 9 shows that the risk of AML was increased for petrol workers, right?

A That's correct.

Q Statistically significant, correct?

A Yes.

Q Why doesn't that study by Jakobsson support your -- I'm sorry. Why doesn't that support the argument that benzene did cause Raul's leukemia?

A Well, because you can always find -- anytime a question has been asked 50 times, a hundred times in the epidemiological literature, you can always find a single positive study here and there. You can't just pull out one positive study or one positive finding from a study and say, okay, this is it. See, I have a positive study; therefore, gasoline causes AML.

When you look collectively at that overall literature, the vast majority of that literature does not support that, which is why it is not generally accepted that gasoline is a human carcinogen,

leukemia being one of the end points that they were evaluating.

Q You said something about -- you mentioned earlier a competitive inhibition. What does that mean?

A When we were discussing earlier this morning about benzene's metabolism -- you remember?

Q Yes.

A Benzene has to be metabolized by the liver, and there are certain enzymes in the liver that take -- that are responsible for that metabolism. Well, there are other chemicals that also go through the liver and are acted on by that same enzyme. So the idea is that if a person -- and there's good data to show this. Whether or not it was happening in Mr. Zendejas or not, I don't know.

But there's clear evidence in the experimental animal literature that if you take an animal and you expose them to benzene, you see effects. You take that same animal and you expose them to benzene and a lot of toluene at the same time, you don't see those same benzene effects because the toluene has prevented the liver from metabolizing the benzene. That is the general consensus. And so that inhibition is what

competitive inhibition is. The competitive means toluene is outcompeting the benzene for that spot in the enzyme.

Q And the position that you're stating there, is that -- is that from gasoline analysis?

A There are certainly studies that say that that does happen with gasoline. There are other studies that say it may not even just be the toluene, but that clearly benzene's toxicity in gasoline is different than benzene's toxicity is by itself.

Q Is it your opinion that therefore, then, benzene's toxicity by itself is different -- I'm probably just rephrasing, saying this over.

Is it your opinion that benzene toxicity in and of itself is much different than benzene toxicity when somebody's exposed to additional other chemicals, toluene being one of them?

A Yeah. You can't just say additional other chemicals. Toluene, it is possible, and there is evidence to support it. You can't -- any chemical is not going to matter, necessarily, because it's only going to be chemicals that are impacting on benzene's metabolism.

Q What other chemicals impact on benzene's metabolism?

A I think all of the aromatic hydrocarbons do --

Q Okay.

A -- xylene, trimethyl benzene, all of them.

Q And that doesn't -- doesn't that defeat the refinery studies? Because those folks are exposed to benzene, and they're exposed to all other aromatic hydrocarbons, generally speaking, and there's still an increased risk of AML for refinery workers, correct?

MR. PERRY: Object.

A Well, I would disagree. I mean, I think if you look at the refinery workers overall as a total body of literature, they do not seem to really be at an increased risk. You can certainly find positive studies here and there, and maybe they were working in a benzene unit. I mean, I don't know exactly why that happens. But the overall literature for refinery workers does not support that those individuals have an increased risk of AML.

Q (By Mr. Patton) Do you know if Shell ever studied its own refinery workers and found an increased risk of AML?

A That, I don't know.

Q If they did, it wouldn't make sense

because of competitive inhibition, right?

A No. I mean, all that competitive inhibition means is if it's occurring, all it's going to do is shift the dose response. And if you would see a certain effect at 10 PPM, maybe you need 15 PPM if they're being co-exposed to toluene to see that same effect. It doesn't ameliorate it. It just changes it.

(Exhibit 10 marked.)

Q (By Mr. Patton) Are you familiar with the Adegoke study, Exhibit 10?

A I'm familiar with that study, yes. I didn't know it was Exhibit 10.

Q Let me read for you a portion real quick, and then I'll let you see this.

A Okay.

Q "The odds ratio among those ever exposed to benzene was 1.7, 95 percent confidence interval, 1.1 to 2.6." So that is statistically significant, correct?

A That's correct.

Q For total leukemia?

A That, I don't remember.

Q I'll let you read it here in a second.

A Okay.

Q So this study also analyzed total leukemia without subsetting it into CML, MDS, AML. Would you agree with that?

A I think they did. They also broke out CML.

Q It shows the ever exposed category had a significantly increased risk for CML. Odds ratio 2.5, a 95 percent confidence interval, 1.3 to 4.9. That's statistically significant, correct? Here you go.

A I think I might have another copy so we can both be looking at it.

Q Is what I just read at the right-hand column of Page 47 incorrect (sic)?

MR. PERRY: Is it correct or incorrect?

Q (By Mr. Patton) Is it correct?

A Let's see. The ever exposed category had a significantly increased risk for CML. Odds ratio 2.5, 95 percent confidence interval, 1.3 to 4.8. So yes, that is -- that one finding is statistically significantly elevated.

Q So if you wanted to make an argument in favor of benzene causing Raul's leukemia, you could cite that study, right?

A No.

Q No? Why not?
A Because when you look where they've divided up the all leukemias, the less than 15 year category, which Mr. Raul would -- I'm sorry -- Mr. Zendejas would fit into, that is not statistically significant or even elevated and for CML or all leukemias. And then like we talked about earlier, the benzene category -- sorry -- the gasoline category, which is the most pertinent to this case and Mr. Zendejas, does not support an increased risk of leukemia.
Q This is kind of a futile exercise, isn't it? I mean, just as you've done in the other 20 cases that you've testified for on behalf of industry, you and I could go through all these studies, and you could just carve it out as you see fit? And that's what you're doing, correct?
MR. PERRY: Object to form.
A I don't think I'm carving it out at all. I mean, like I said, I will show the jury and they will see the word "gasoline" and the risks associated with gasoline exposure. I'm not carving it out. That's what this study supports.
Q (By Mr. Patton) Do you believe that the health impact of exposure to gasoline has been fully

elucidated?

A Say that again.

Q (By Mr. Patton) Do you believe the health impact of exposure to gasoline has been fully elucidated?

A Well, I think we know a lot about it. We've been doing animal studies for some time. We've got a lot of epi. I wouldn't say we know everything there is to know about it.

(Exhibit 11 marked.)

Q (By Mr. Patton) So this person, Lagorio from the National Health Institute in Rome, Exhibit 11, one of your studies, concludes that "Filling station attendants are exposed to gasoline vapors and seem at risk of cancer at various sites. But due to the power limitations of this study, a precise estimate of the risk for many causes of death was not achievable. Further cohort studies of greater size are warranted."

And this is from 1994. Do you agree with that statement?

A I don't have any reason to disagree with that.

(Exhibit 12 marked.)

Q (By Mr. Patton) "Workers in the gasoline

service station industry experienced a leukemia mortality excess. PMR equals 328. N equals 3. P less than 0.05." What does that mean? This is from the Schwartz study.

A Yeah, Schwartz. He did a proportional mortality study which -- you know, it's better than a case report because it is at least analytical, but it isn't much better than a case report. And that means that it was a statistically significant finding. But the problem with the PMR, proportional mortality, is that it takes all of the deaths and forces them into a percentage so that the total number of death has to equal 100. So if you see an increase in one death -- one cause of death, then there have to be subsequent decreases in others so that the total amount ends up being 100. So it's not a very good study design.

(Exhibit 13 marked.)

Q (By Mr. Patton) Rushton 1996, Exhibit 13, he (sic) concludes there's "some suggestion of a relation between exposure to benzene and myeloid leukemia; in particular, for acute myeloid and monocytic leukemia. Peaked exposures seemed to be experienced for this disease." I don't understand that. He says, "Further work is necessary," 1997, correct?

A Yes. And Lesley is a woman.
Q What does it have to do with your opinions, that study, Exhibit 13?
A Well, this is a -- this is a very important study. This was looking at a large number of distribution workers, which I think would be relevant to Mr. Zendejas. They looked at various forms of leukemia. They looked at various forms of leukemia associated with cumulative exposures, peaks, intensities, a variety of exposure metrics associated with those workers. And there is a chart specifically for chronic myeloid leukemia along -- associated with various benzene exposure metrics.
Q What kind of benzene exposure were these workers experiencing via gasoline operations?
A You mean in terms of quantification?
Q Yes, sir.
A I don't -- well, yeah. I mean, there was less than 4.5. There was a 4.5 to something. There was a greater than 45 PPM years. I don't know whether those were the distribution workers or not. But at no point in that dose response was there an increased risk of CML, which I think is directly germane to what we're doing here.
Q So even when it shows like all leukemias,

your response is then to say, yeah, but it doesn't deal with -- but it doesn't help it -- help the case to support CML and gasoline exposure, correct?

A Well, I don't believe -- and, you know, we can talk about this as much as you like. But epidemiologists and hematologists and toxicologists we've known for a long time that the etiological risk factors for different kinds of leukemia are clearly different.

Q Do you believe that there's no reason for these researchers to even mention all leukemias? Like what's the point in that? Because if you can't connect it with the disease until you cut it down to the subtype or the division, what's the point in these researchers, in their published studies, to even mention what the odds ratio or what the risk is for all leukemias?

A Well, I mean, that's a fair -- that's a fair question. And I don't know the answer to it.

Q There must be some value because they do it, right?

A Well, I mean, it can go both ways. It can hurt both ways. For example, let's say that you do not find an increased risk of total leukemia, but then when you subdivide it out you see that there, is

in fact, an increased risk of AML, but there's no increased risk of these others so that it kind of dilutes that finding. And so the overall risk, you don't see it elevated. I think it hurts both ways.

Q Have you ever wrote to any of these researchers and said, Hey, you shouldn't deal with all leukemias because it doesn't really matter?

A I've actually had that question with Lesley Rushton, but epidemiologists are going to do what they're going to do.

(Exhibit 14 marked.)

Q (By Mr. Patton) Exhibit 14 is a follow-up from Rushton. It concludes, "Mortality from leukaemia was high at one company and in drivers overall." This is specific to gasoline distribution, right?

A I would have to look at it, but that -- I think that's correct.

Q It shows here that "Several studies have carried out personal air sampling or taken urine samples to investigate the exposure of workers to benzene at bulk marketing terminals or service stations." You see that where I circled that on Page 14?

A Yes.

Q Are you aware of Shell ever performing that type of investigation for gasoline distribution workers?

A I don't know. Are these studies Shell studies?

Q I don't think that one is. My question is a little different.

Are you aware of Shell ever conducting air monitoring, blood samples, urine samples, for gasoline workers outside of Shell's own refineries? Do you know?

A Well, I mean, what I'm saying is, she referenced several studies in support of that sentence. And perhaps they were Shell, but I don't know. You'd have to go and pull these studies and look to see where the people worked and where they were doing the measurements.

Q In this case that we're here for today, has Shell shown you any information indicating that they carried any of those that types of investigation for workers?

A No.

Q Okay. Do you think it would be helpful so that we could be better elucidated when it comes to the relationship between benzene via gasoline and

various forms of leukemia? Do you think it would be helpful for Shell to perform some tests in that manner?

MR. PERRY: Objection, form and vague.

A Well, I think there is a lot of data on what the potential benzene exposures are in these various tasks. I don't think they're very high, but there are plenty of studies, and that's what others in this case will discuss.

THE VIDEOGRAPHER: One minute, Counsel.

MR. PATTON: Off the record.

THE VIDEOGRAPHER: We're off the record at 2:10. This is the end of Tape 3.

(Recess from 2:10 p.m. to 2:14 p.m.)

THE VIDEOGRAPHER: We're back on the record at 2:14. This is the beginning of Tape 4.

Q (By Mr. Patton) I'll mark as Exhibit 15 the Schnatter study. What does this have to do with your opinions?

A Rob Schnatter did the same basic design that we were just talking about with Lesley Rushton where they looked at a variety of worker types in refineries, distribution workers, et cetera.

Q Schnatter worked for Exxon, right?

A Yes.

Q If the results of the Schnatter study would have been such that, Hey, it looks like benzene causes an excess risk of leukemia, that could have been bad for industry, would you agree?

MR. PERRY: Object to form.

A I know Rob Schnatter personally, and he would publish his findings. And, in fact, Jeff Lewis, Dr. Lewis, who is a colleague of Dr. Schnatter, has published papers that show an increased risk of this or that in various refineries, you know, because that's what the science said.

Q (By Mr. Patton) Schnatter performed another study -- I'll mark it as Exhibit 16 -- "The Relationship Between Low-Level Benzene Exposure and Leukemia in Canadian Petroleum Workers." Are you familiar with this?

A Yes.

Q It says here, "This study" -- and this is 1996. "This study is consistent with other data in that it was unable to demonstrate a relationship between leukemia and long-term low level benzene exposure. The power of the study was limited. Thus, further study on benzene exposures in this concentration are warranted."

My question for you, sir, is, doesn't it

benefit industry when the studies specific to gasoline are inconclusive?

MR. PERRY: Object to form.

Q (By Mr. Patton) I mean, as far as responsibility and liability goes, industry is better off when there are inconclusive studies rather than positive studies, true?

MR. PERRY: Object to form.

A I mean, I think we want to know the right answer.

Q (By Mr. Patton) Did you want to know the right answer in this case?

A Absolutely.

Q Is there anything you asked Shell or its attorneys to do to provide you different -- better information so that you could give a right answer?

A I think I had everything I needed.

Q But you don't believe Steve Petty's opinions on how much benzene Raul was exposed to, you don't believe those are accurate, correct?

A No.

Q So wouldn't you require accurate estimates for you to be able to give your opinions?

A I don't understand why I would. Even if you accept his number, that's not going to get you to

an increased risk of CML and really not even going to get you to an increased risk of AML, even if you wanted to accept that this is an AML case. I just don't think those are scientifically defendable.

Q (By Mr. Patton) Gerhard Raabe did a study on -- a mortality study of workers at a refinery in Beaumont. Do you know what he found?

A Gerry Raabe, no. I mean, I'd have to review that study. He's done several.

Q He works for industry, right, works for Mobil, or worked for Mobil?

A He did, yes. I think you'll find through the literature that most of the epidemiology studies for refineries were done by people that had a vested interest in those refineries.

Q Because refiners are the ones who can afford to do those studies, correct?

MR. PERRY: Object to form.

Q (By Mr. Patton) And they have the access to the people and the information, right?

MR. PERRY: Object.

A I think all of that is probably true to some extent.

Q (By Mr. Patton) Would you agree with me that the body of literature you brought with you

today is at least suggestive of a responsibility or of a relationship between benzene exposure and leukemia, or you just -- you just disagree?

A Well, I think the literature that we've been discussing and others is clear that at certain exposure levels to benzene, you get an increased risk of AML.

Q I guess I just don't understand why there are still studies into the '90s and into the 2000s about gasoline, benzene, and leukemia if the question's already settled. Can you explain that, or do you have any understanding?

A The question's not settled. There's a whole lot of information that we would like to know about the process and characteristics and morphology and progression and treatment and response and all kinds of things that aren't settled.

(Exhibit 17 marked.)

Q (By Mr. Patton) Exhibit 17 is the Tsai study, T-s-a-i. What does this have to do with your opinions?

A Sean Tsai I just mentioned earlier. He works for Shell so I think the studies that Sean Tsai has published are going to probably be on Shell employees. But that particular study, I don't --

you'll have to show it to me.

Q I'm handing you Exhibit 17. What does that have to do with your opinions in this case?

A I think I referenced this to just illustrate what we were talking about earlier, and that is that the majority of the literature on refinery workers does not support that they have an increased risk of AML or leukemia.

Q Do you know if Raul Zendejas had any chromosome damage?

A My understanding is no, he did not.

Q Do you believe if someone has chromosome damage, that increases the likelihood that they have a benzene-related leukemia?

A I think that based on our understanding of what benzene can do, based on our understanding of what the AML's following chemotherapeutic agents look like, based on our understanding of what AML following radiation exposure looks like, that yes, cytogenetic changes are certainly consistent with a chemical etiology. Cytogenetically normal AMLs would be inconsistent with a chemical etiology.

Q Are you aware of -- in your opinion are there any specific cytogenetic abnormalities that tend to be -- that are really high on the list of

chemically related, so to speak?

A I mean, there's a lot of discussion. It depends on the chemical. If you're talking about alkylating chemotherapeutic agents --

Q I'm talking about benzene.

A Oh.

Q It's benzene, benzene, benzene all day, every day of the week.

A You don't see --

Q That's my life. Let me ask --

A I'm sorry about that.

Q -- the question, sir. Is there a specific cytogenetic abnormality that you, as an expert, attribute to benzene exposure?

A There are some studies that have shown trisomy 8 might have something to do with benzene, involvement of Chromosome 8 with an 8;21 translocation. Martyn Smith's group out of Berkeley published that. It hasn't been reproduced, but nonetheless, that data's out there. They have shown Chromosome 5, some involvement of Chromosome 7. So there are -- there are some cytogenetic changes that people have said, you know, benzene can probably do this.

Q So if someone had a myeloid leukemia and

they had a minus 5 and a minus 7 chromosome, you would believe that, hey, it increases the chances that it was chemically induced or benzene induced?

A If it was an AML and it had a 5 or a 7, then yes, I would say those were both consistent with a chemical etiology.

Q What if it's MDS?

A Same.

Q The same.

A You want this one back?

Q Sure. Let's put it in the pile.

(Exhibit 18 marked.)

Q (By Mr. Patton) Exhibit 18 is the Santos-Mello "Cytogenetic Studies on Gasoline Attendants." I don't have any questions on that one for you right now.

A Okay.

Q What is the amount of benzene that somebody can be exposed to on a cumulative level that would not put them at risk of leukemia? Like if I want to go be exposed to benzene, okay? I just want to do it.

A Okay.

Q But I don't want to get leukemia.

A Okay.

Q What are those numbers that I want to make sure I don't get over?

A If you're concerned about -- you're probably going to get mad at this. But if you're concerned about CML, I don't think it matters.

Q I can just go work with benzene all day?

A That seems to be the case. But if you're concerned about AML, then the epidemiology literature supports that somewhere around 40 PPM years over a cumulative exposure basis is where the risk becomes statistically significant. It doesn't mean you would get it at 40. Lots of people exposed to that much didn't get it. But you at least would be at a statistically significant increased risk.

Q Are you familiar with the work being done at the University of Colorado Health Sciences Center in relation to the Shanghai study?

A Well, I mean, I worked with Dr. Irons, and I'm in that department. But Rich Irons has retired from the university so I'm not sure -- in fact, I think as of November the funding being run through the university has ended. So I don't think there's anything going on at Colorado.

Q Nevertheless, in those activities haven't they concluded that it is accepted that there's an

increased risk for AML above 20 PPM cumulative years of benzene exposure? You're putting it at 40. And what I'm saying to you is I believe others on your side of the -- on your side of the battle, so to speak, have put it down to 20. Do you have any opinion either way on that?

A I haven't seen that -- I haven't seen the data that could be used to support that.

Q The Australian Health Watch study puts that lower number down around 1 PPM, right?

MR. PERRY: Object to form.

A I disagree with that.

Q (By Mr. Patton) You disagree with that?

A Yes.

Q Am I just saying it wrong, or they're saying it and you disagree with them?

A No, you're saying it wrong. What she says for -- okay. For CML, there is no risk.

Q What about for all leukemias?

A I don't remember the numbers for all leukemias. For AML, then the risks are statistically significant at something greater than 8 but less than 57. So to me, that is consistent with many of the other studies that show an increase somewhere around 40 to 50 PPM years.

Q I'm going to mark Exhibit 19. This is Peter Greenwald, "Landmarks in the History of Cancer Epidemiology."

(Exhibit 19 marked.)

A How did that get in there? That was in the causation binder?

Q (By Mr. Patton) Maybe.

A I just printed it out because I thought it was an interesting title.

Q I did, too.

I'm going to read something, and then can you explain it to me?

A I can try.

Q Okay. "Quantitative" -- and this is on Page 2158 --

A Okay.

Q -- of Exhibit 19. "Quantitative assessment of exposure is difficult, posing challenges to epidemiological investigation of occupation/cancer relationships." Do you agree with that?

A Yes.

Q "Whereas individuals usually can describe their smoking histories and eating habits reasonably well, they're often relatively ignorant about their

work exposures." Do you agree with that?

A Not completely. I mean, I think -- well, they could describe their smoking, but I think frequently they don't. But yes, it's hard to measure -- the occupational exposure part of an epi study is frequently the weakest part.

Q Right. I mean, you ask a guy what he ate for lunch or how much he smokes, he can tell you about that?

A Correct.

Q You ask him how many PPM he's exposed to of benzene when he's working with whatever solvent, I mean, you wouldn't expect a guy to know that, would you?

A He wouldn't know, I agree.

Q "Moreover, because of long latency periods between exposure and cancer diagnosis and because working conditions change over time, data on current workplace exposures may not reflect those experienced by past workers," correct?

A Yes.

Q You agree with that?

A Yes.

Q And you would also agree that workplace exposures in past data like some of the numbers we

might find in these studies you're relying on, those might not accurately reflect exposure data for a given situation like Yuma, Arizona, a small bulk terminal, top-loading, no vapor recovery; would you agree with that?

A Well, in terms of your specific scenario with that case, I mean, all I could say is that even if you accepted Stephen Petty's assessment, then yes, that cumulative exposure is absolutely encompassed in these studies. That is not something that's so far out there that we don't have a study that would tell us about the risk associated with those exposures.

Q So you would agree with me that the studies -- some of the studies do show exposures at the level that Petty estimates? However, you're critical of Petty for other reasons?

A I would say that this literature does have people that are exposed at that level but not from working five years and not from being -- well, I'm not sure about the tank truck drivers. But the ones I'm thinking about are the Pliofilm workers, the rubber workers, the shoe workers. These people have really high benzene exposures.

Q "Epidemiologists" -- and I'm still reading from Exhibit 19. "Epidemiologists often have had to

rely on proxy measures of exposure and studies of occupational cancer." What does that mean, "proxy measures"?

A Like duration.

Q Surveys?

A No. Like instead of actually having quantitative air monitoring data that might be lacking or problematic, they would just say, Okay, well, this guy worked there for five years, these guys worked there for ten years, all those people over there worked there for 20 years, so let's see whether or not there's changes in their risk. So in that case, duration would kind of be a proxy for exposure.

Q Reading on in Exhibit 19, "For example, the epidemiological studies which showed that physicians who specialized in radiology prior to 1950 were at an increased risk of leukemia because of high radiation exposure relied primarily on knowledge of typical radiation protection practices in different time periods, rather then on specifics about the exposure of particular individuals." Does that make sense to you?

A No. I'd have to read it again and look at it in the context of that.

Q "Difficulty in obtaining accurate exposure data remains a problem today, hampering recent efforts such as the determination of whether occupational exposure to pesticides and electromagnetics fields influence cancer risk." Do you agree that difficulty in obtaining accurate exposure data remains a problem today?

A It depends on what it is you're looking for. Certainly something like electromagnetic fields, that's really problematic; benzene, less so. But in an epidemiological study, the exposure assessment is always the weakest part.

Q Because the exposure assessment is always the weakest part in these epi studies, doesn't it make a little more sense to look at the group of workers a little more specifically with a little more care, so to speak, to draw conclusions?

A I don't understand. I think they have done that.

Q Well, I mean, I think that's what Dr. Carroll did and Dr. Gore did for Raul. I mean, they look at -- looked at his specific circumstances. It's in the medical records that the guy's exposed to gasoline and hydrocarbons. They considered the unlikelihood that he would get his disease at his

age, and they concluded that benzene caused it. Why isn't that fair?

MR. PERRY: Object to form.

Q (By Mr. Patton) I mean, you've got all these problems with these epi studies, but epi studies are what you're relying on to disprove causation. Why isn't it fair to do what Dr. Gore and Dr. Carroll did?

A It's not just me. Epi studies are what EPA, IARC, OSHA, ATSDR -- I mean, this is the gold standard for what we're doing. These are human studies with toxicity. The difference is whether you're going to have an authority based kind of opinion or whether you're going to have a science based opinion. And I think that will be evident if this goes to trial when they start asking, Well, what is the scientific basis in support of your opinion. And that's going to be lacking.

Q You believe there is a -- that the scientific basis supporting those experts' opinion is lacking?

A Yes.

Q Isn't it easier, though, to use the epidemiology to carve out a certain disease or a certain specific circumstance to say it did not cause

it? Isn't that easier to do than collect the literature to say it did cause it?

MR. PERRY: Object to form.

A I don't -- I don't how to answer that question. I don't think that I have carved out anything. I have brought in the literature that is related to gasoline that is related to CML. That's what he was exposed to. That's what everyone said. That's the disease that he had. That's agreed upon with everyone. I don't -- I don't see how I've carved out something specific out of the literature.

Q (By Mr. Patton) He's got AML. I mean, if he died tomorrow, it's AML.

MR. PERRY: Object to form.

A We've gone through that. I mean, your own expert said that he would call it blast crisis.

Q (By Mr. Patton) Dr. Carroll already called it AML.

A Dr. Gore called it blast crisis, and that's what -- if you pick up any hematology textbook and you're talking about a person who has his disease following atypical or typical CML, they will call it blast crisis.

Q Your job is easier because you can quibble with these -- with calling it atypical CML?

MR. PERRY: Object to form.

A I'm not sure about that. I don't think my job is easy, although -- I take that back. In this case, it was pretty straightforward.

Q (By Mr. Patton) Was it easy for you to conclude that Raul's exposure to benzene via gasoline did not cause his form of leukemia?

A I wouldn't say it's easy, but I think the literature is less schizophrenic with regard to gasoline and CML than it is a lot of other diseases. I think that literature is pretty clear, pretty clearly negative.

(Exhibit 20 marked.)

Q (By Mr. Patton) Exhibit 20, this is one of the medical records you have. I circled the portion about cytogenetics. Does that indicate he had a translocation?

A In one cell, which in a cytogenetic point of view is insufficient. They have to see it in at least three cells, or they don't count it.

Q Are you a cytogeneticist or a pathologist?

A I took a lot of pathology. Pathology and toxicology in my institution were hand in hand. And I wasn't intimately involved with the studies, but we published -- out of the lab that I worked in, we

published several cytogenetic papers on benzene metabolites and various lymphocytes. I know the person who did the cytogenetics. I mean, I'm not a cytogeneticist, but I'm familiar with the techniques.

Q The term "atypical CML" did not exist before 2000, correct?

A I think they called it Philadelphia negative -- Philadelphia chromosome negative. And I don't remember when they stopped doing that.

Q Because the definition atypical CML was not even used until around 2000 or so, you wouldn't expect that term to be reflected in the literature, would you?

A I think that's right, yes.

(Exhibits 21 and 22 marked.)

Q (By Mr. Patton) Exhibit 21 is another medical record.

Exhibit 22, upon diagnosis Dr. Fayssoux and Dr. Abdella, they mentioned occupation and habits: "He works as a delivery man or a truck driver for diesel fuel and gasoline." Why should they even bother to mention that?

A You'll have to ask them. I mean, I think as Dr. Nadelson described in his deposition, that is something that they typically do, take an at least

brief occupational history of people that have leukemia.

(Exhibit 23 marked.)

Q (By Mr. Patton) Exhibit 23, Dr. Fayssoux originally called Raul's disease a myeloid proliferative disorder, MDS, right?

MR. PERRY: Objection, form.

Q (By Mr. Patton) MPD?

A A myelo -- a myeloproliferative disorder is CML. CML is a myeloproliferative disorder.

Q It's a myeloid leukemia, a myeloid illness, right?

A Which one?

Q MPD or CML or MDS, all of them?

A They are -- no. Okay. MDS --

Q Let me ask a better question.

A Okay.

Q Is MDS a bone marrow disease?

A Yes.

Q Is PMD a bone marrow disease?

A Yes.

Q Is CML a bone marrow disease?

A Yes.

Q Is atypical CML a bone marrow disease?

A Correct.

Q Is AML a bone marrow disease?
A Yes.
Q Is CMML a bone marrow disease?
A Yes. It's a form of MDS, or used to be.
THE DEPONENT: Is this the exhibit pile here (indicating)?
MR. PERRY: Yes, sir.
THE DEPONENT: You want my report in that pile?
MR. PERRY: Yeah. I've just been kind of gathering them as we go.
THE DEPONENT: These are the ones that he asked me to read.
Q (By Mr. Patton) Let's go through your report.
A I need it back. Sorry.
Q First of all, do you know when benzene was first associated with any form of bone marrow disease? Do you know approximately what year?
A I can't tell you when it was first associated. I can tell you when the first publication came out.
Q When was that?
A 1897, I think it was.
Q And they drew the conclusion that benzene

is toxic to the bone marrow, correct?

A Yes.

Q And they drew that conclusion before we have modern science today and before we started classifying and reclassifying, for treatment and prognosis purposes, the different forms of bone marrow disease or myeloid diseases, correct?

MR. PERRY: Object to form.

A I don't understand that question. It was aplastic anemia. It was a fairly specific diagnosis.

Q (By Mr. Patton) Do you know when benzene was first connected with any type of myeloid leukemia rather than aplastic anemia? Do you know what decade?

A The first publication of benzene leukemia was in 1828 -- sorry -- 1928, but it was actually a lymphoid leukemia. So based on what we know now, they were probably -- they were incorrect, and that wasn't a benzene-related malignancy. But nonetheless, that's when the first paper came out, Delores and something.

Q "In summary, there is" -- this is Page 10 of your report.

A Okay.

Q "In summary, there is no consistent

evidence in the existing body of scientific literature that occupational exposure to gasoline results in an elevated risk of developing CML, MDS, or any other human cancer." Do you still agree with that?

A Absolutely.

Q I mean, we went through some of these studies, and I pointed out to you a handful of them that did show evidence of an increased or elevated risk of developing leukemias. Didn't we do that?

A No, no. That's why no consistent evidence -- and that's why Bradford Hill in his 1965 paper said, Here's what you've got to do in order to establish causation. That was the whole idea. Because we know that you're going to have, just by random chance, a positive finding here or a positive finding there. When you look collectively at that overall literature, it does not support an association.

Q Exhibit 5 or Exhibit 6 -- I'm not sure which one it is.

A Okay. Well, I'll have to get those back. I'm sorry.

Q It's Infante's paper.

A Okay. It's in here. I think it's on the

top, maybe just right under those medical reports. All right. It's in there.

Q Exhibit 6.

A Okay.

Q Turn to Page 43.

A Okay.

Q See the upper right -- the right-hand column? Or can you just start at the lower left-hand corner. Can you read aloud for us, "In the 1970s."

A I'll try. "In the 1970s benzene manufacturers and users began a new approach for conveying knowledge about the toxicity of benzene to the public, in general, and to workers and plant managers specifically which contributed to a continuation of overexposure to benzene. This is the period when manufacturers began to hire consultants to downplay the importance of scientific observations related to the toxicity of benzene and to introduce unresolvable arguments about dose response analysis, which had an impact in delaying much-needed government regulations that sought to reduce benzene exposure in the workplace."

Q Let's stop right there.

MR. PERRY: Wait. What page is that?

THE DEPONENT: 43.

MR. PERRY: 43 of Exhibit 6.

Q (By Mr. Patton) Dr. Pyatt, manufacturers like Shell have hired you as consultants, correct?

A I am a consultant, yes.

Q Do you believe it is fair to say that you are downplaying the importance of the scientific observations related to the toxicity of benzene? Do you believe you're doing that?

A No, absolutely not. I think that's what your experts are doing.

Q Do you believe you're introducing unresolvable arguments about dose response analysis?

A No.

Q Do you believe to better understand the relationship between -- well, let me look at it a different way.

You have essentially three things going on for a worker like Raul in a case like this. You have benzene exposure, gasoline exposure, and him as a gasoline distribution worker, okay? Wouldn't it be helpful to study gasoline distribution workers who worked in similar environments to him to better understand the significance of benzene exposure to that class of worker?

A We have; Schnatter, Rushton, Wong,

Sorahan, other refinery studies that have included distribution workers.

Q Sir, I'll represent --

A We have.

Q I'll represent to you that none of the studies that we went through talked about top-loading without vapor recovery. Do you have any reason to disagree with me?

A I don't know whether they specifically addressed that or not. But when they're looking at 18,000 distribution workers, I would assume that some of them are doing exactly what Mr. Zendejas did.

MR. PATTON: Off the record.

THE VIDEOGRAPHER: We're off the record at 2:46.

(Recess from 2:46 p.m. to 2:47 p.m.)

THE VIDEOGRAPHER: We're back on the record at 2:47.

Q (By Mr. Patton) Dr. Pyatt, do you believe that industry is responsible to anyone or any class of workers who have suffered leukemia as a result of chemical exposure?

MR. PERRY: Object to form.

A What do you mean, "responsible"? I don't think --

Q (By Mr. Patton) Your answer says enough, sir.

A Okay.

Q Do you believe there's a difference in something like gasoline being called a carcinogen or not and someone being exposed in a manner and at a rate that exposes them to dangerous levels and an increased risk?

MR. PERRY: Object to form.

A I'm not quite sure how to answer that. But I would go back to what we've talked about before, and that is even if you accept Stephen Petty's quantitative exposure assessment, which should take care of all of these other things that you're addressing -- heat and Yuma and this, that, and the other, top-loading -- it's not enough. It just is not sufficient exposure.

Q (By Mr. Patton) Do you believe benzene exposure played any role, even a 1 percent role, in causing Raul's leukemia?

A I don't know how to answer that question.

Q Why not? Why can't you answer it?

A I just can't. I just don't know how to say, well, it's a 6.5 percent probability or it's an 8 percent probability or it's 1 or 80. I just don't

know how to answer that. What I can tell you is the scientific literature clearly does not support an increased risk of CML from gasoline exposure. To me, that's what this case is about, CML and gasoline. And it does not establish a causal association.

Q Are there any other opinions that you intend to offer in this case that are not described in your report or that we haven't talked about?

A I don't -- I can't imagine what those would be, no.

Q Have you been asked to testify at trial in this case?

A Yes.

Q Do you know when that is?

A Yes, vaguely.

Q What's that?

A Yes.

Q Is there any additional information you'd like to see in this case before you testify live in front of a judge and a jury?

A The only thing that I would like to see is -- and mainly just curiosity -- what Dr. Jamall ends up saying about Stephen Petty's report and if he does a cumulative exposure estimate.

Q Putting benzene aside, aren't there other

chemicals out there and other substances out there for which there are no safe levels of exposure?

MR. PERRY: Object to form.

A I don't think that's true for benzene. Well, that's what you said, putting benzene aside. I mean, I do believe there are safe exposure levels to benzene. I don't know of a chemical -- I mean, methylmercury is really, really toxic, and it's really, really toxic at really low levels. I don't know whether you could actually be exposed to measurable amounts of methylmercury and not have some kind of toxicity, but I'm not positive.

Q (By Mr. Patton) What if someone had AML with minus 5 minus, minus 7 chromosomes, and they worked with gasoline for six years? Would you believe that gasoline could have possibly played a role in that person's disease?

A That's not what the literature would support.

Q It's got to be pure benzene?

A It's got to be benzene at a high enough level to put them into an increased risk based on the quantitative epi. The other things, the minus 5 and the 7, that's what we would expect the disease to look like if it were, in fact, caused by benzene,

perhaps. You can't take those features and say, Okay, because we've got these features, we don't care about exposure anymore, it must have been caused by benzene.

They're not that specific. People get 5 and 7, monosomy 7 and interstitial deletions in 5Q31. They get that all the time without any exposure. So that is just a description of what a person would look like -- potentially look like if they had a benzene induced leukemia.

Q Some of these studies in the exhibits, they talk about the relationship between benzene and specific types of leukemias or all leukemias. Some of these studies don't even do a dose analysis, do they?

A That's true. And from a public health point of view, those studies are not very useful.

Q So all the studies that we've marked as exhibits that don't look at benzene exposure in a quantitative or an exposure or dose type viewpoint, those just do not have much value to the public health perspective; is that your testimony?

A No. I mean, if you -- you can look at -- say, you can do an epi study, and you can say, never, ever, okay? And one of those that we talked about

did, never, ever; exposed to gasoline, yes; exposed to gasoline, no. All right. So what if the answer is yes; people who say, yes, I was exposed to gasoline, I have an increased risk of lung cancer.

Okay, well, a public health official who is tasked with setting an exposure level for gasoline to protect workers from getting lung cancer, a yes/no answer doesn't help them. A high, low, medium doesn't help them unless someone can go in and say, Okay, when they said "low," here's what the exposures were. When they said "medium," here's what they were.

So the more quantitative information you have about dose, the more useful it is from a risk assessment public health causation basis.

Q On those three cases that you said you've told industry, Hey, I can't testify for you, did you tell them that you thought benzene, 51 percent chance, did play a role?

A What I told them was, I cannot rule it out.

Q But if you -- could you not rule it out to 51 percent?

A They didn't ask, and I just -- I can't do that. I mean, I don't know how to do this 8, 75.

You know, I just said I can't rule it out. I can't say definitively that there is evidence to support this person's disease wasn't caused by their occupation.

Q Putting aside the person who had the dental work and the hydrochloric acid, can you cite for me another example where you've given the opinion in literature or elsewhere in your work that exposure to Chemical or Substance X caused Disease or Health Effect Y?

A No, other than the ones that we've talked about.

Q Okay. Thank you for your time.

A You're welcome.

EXAMINATION

BY MR. PERRY:

Q Can you please state your name.

A David Pyatt.

Q And what is your occupation?

Is it Mr. Pyatt or Dr. Pyatt?

A My family calls me David or Dad, but students usually call me Doctor.

Q And do you have a Ph.D?

A I do, in toxicology.

Q And what is your current occupation?

A I am a toxicologist.
Q With which company?
A I started my own company in 2005 called Summit Toxicology, but I have been with the university since 1996 teaching, sitting on committees, doing research, that kind of thing.
Q And it's October 8, 2009, and we're outside of Denver, Colorado, correct?
A That's right.
Q And when you say "the university," which university are you referring to?
A I'm talking about the University of Colorado. We've only got one really big one here in Colorado so that's probably why we say that.
Q And really, today we've been -- we started your deposition this morning around 9:30 mountain time. And there's been lots of questions about lots of literature, and I want us to kind of focus on the ultimate issue. And this is the question, Dr. Pyatt, I'm asking you as a professional toxicologist with university appointments, with membership in professional societies dealing with toxicology, and the question is this: Is it generally accepted in the scientific community that gasoline exposure causes leukemia?

MR. PATTON: Objection, form.

A No.

Q (By Mr. Perry) And why do you say that?

A Well, because if you put quotations around generally accepted, how would you know whether something is generally accepted? You know, did you go out and do a survey of all the toxicologists? Well, in fact, in this case, that is what has been done. And IARC, ATSDR, The Agency For Toxic Substances and Disease Registry, a bunch of toxicologists, physicians, epidemiologists, they don't believe gasoline is carcinogenic.

IARC, the International Agency for the Research on Cancer, preeminent physicians, toxicologists, epidemiologists that evaluate any chemical that has the potential to be a human carcinogen, they don't believe gasoline is a human carcinogen, let alone causing leukemia. OSHA, United States Environmental Protection Agency, the American Conference of Government Industrial Hygienists -- I mean, collectively that is a lot of really talented, really smart epidemiologists and toxicologists that collectively say gasoline is not a carcinogen. So I believe saying it is not generally accepted is totally supportable.

Q Is it generally accepted in the scientific community that gasoline exposure causes CML or atypical CML?

A Absolutely not. If you can't even say that it causes cancer as a collective group of 200 or so miscellaneous diseases, the answer to that is no. There is -- no.

Q Is it generally accepted in the scientific literature that benzene exposure, regardless of the level of exposure, causes CML, including 1atypical CML?

A That has not been shown in the scientific literature. We know that high dose exposure to benzene can cause acute myelogenous leukemia; some types, certain situations, but it can happen. That is the only form that has been clearly established. And CML, there is -- there is no association with high dose -- even high dose exposure to benzene in CML.

MR. PERRY: No further questions.

EXAMINATION

BY MR. PATTON:

Q Dr. Pyatt, if it is so clearly established in the relevant scientific community that exposure to gasoline isn't going to cause any leukemia, why did

all these people and all these journals and all these resources study benzene exposure in the context of gasoline? And why did some of them find an increased risk of certain types of leukemia among gasoline distribution workers?

A Which one would you like for me to answer?

Q Both.

A Okay. Can you start again with the first one? Why they do the studies with gasoline?

Q Let me back up.

A Okay.

Q If it is so widely accepted, as Mr. Perry just asked you, in the scientific community that gasoline exposure just doesn't cause leukemia, why is it still studied in the context of gasoline distribution workers? And more importantly, if it is so widely accepted, how do you reconcile that with the fact that some of these studies do show an increased risk of certain types of leukemia among gasoline distribution workers?

A Fine. The reason why I say it is widely accepted in these scientific agencies -- I mean, a lot of the data that's in this pile and a lot of the data that's not in this pile that's in this binder, that is why they know what they know. So this

literature -- I mean ATSDR, it was 2005 when they came out. IARC's going to reevaluate everything this year. I don't know if gasoline's on that list or not. I actually don't think that it is. They feel like they've -- they know enough about gasoline from the last time they did it. The ACGIH, when they write their TLVs, which they do every year, they update it with the literature. So all of that science has gone into this general acceptance.

Now, your question -- which now this is probably the third time -- you can always find spotty, you know, quirky findings here and there. That Jakobsson was a real -- that is a positive study. Jakobsson is a positive study, but it's really small. And there was like three or four leukemias that maybe were associated with petroleum workers. When you look at it collectively, the overall literature, it doesn't -- it's not consistent. It doesn't hold up. And that's what you have to do to say gasoline is or gasoline isn't a carcinogen.

Q What gives you the authority, you personally as Dr. Pyatt, to say that it's just not widely accepted? I mean, where do you get that power from?

A It's not my power. He asked the question. And what I said was, ATSDR said no. There's no power with me. I'm just telling you what they said. If you don't believe it, you know, I can pull up their Web site and show you what they say about gasoline.

Q We're misunderstanding each other here. Do you believe it is widely accepted in the relevant scientific community that gasoline exposure and exposure to benzene as a gasoline distribution worker does not create an increased risk of leukemia? Do you believe that? Do you believe it's widely accepted as to those -- what I just stated?

A That was kind of a double negative. I believe it is widely accepted in the scientific community, as evidenced by what we were just discussing, that gasoline is not a carcinogen.

Q Okay. I heard that part already.

A Okay.

Q Do you believe it is widely accepted in the scientific community that exposure to benzene from gasoline as a gasoline distribution worker does not cause or cannot cause an increased risk of leukemia?

A I don't see that as being any different.

Q Okay. What gives you the power -- what

makes you the guru to say that it's widely accepted as it relates to benzene via gasoline, specific to gasoline distribution workers?

A There's no guruness here. I'm not being a guru. You are emphasizing the benzene in the gasoline, as if that is something unique to Mr. Zendejas and it is not commonly found in every other form of gasoline. Benzene is in gasoline in every one of these studies. Benzene is in gasoline that ATSDR evaluated. OSHA, US EPA, ACGIH -- all of those have gasoline that has benzene in it.

Q Do you believe there's --

A Wait a minute. I'm not done yet.

Q Okay. I'm sorry.

A So collectively, all of those studies have said that gasoline with benzene in it is not carcinogenic. If it's not carcinogenic, it's not causing leukemia. It's not causing atypical CML. It's not causing myelodysplasia. It's not causing whatever you want to say.

Q Do you believe there is an increased risk of leukemia by gasoline distribution workers?

A By gasoline distribution workers?

Q For gasoline distribution workers.

A No.

Q Let me say it in a different way. Do you believe gasoline distribution workers have an increased risk of leukemia?

A If you're talking about collective leukemias, no. We've gone through that several times, and we've also discussed what do you mean by an increased risk.

Q What about AML? Do gasoline distribution workers have an increased of AML?

A That would not be supportable by the scientific literature.

Q That's your opinion?

A That's the scientific literature's opinion.

Q But you're the one making the conclusion that it's not supportable?

A That's true.

MR. PATTON: Okay. Thank you.

THE DEPONENT: Are you done, too?

THE VIDEOGRAPHER: We're off the record at 3:03. This concludes today's deposition of Dr. David Pyatt.

(The deposition concluded at 3:03 p.m., October 8, 2009.)

I, DAVID PYATT, do hereby certify that I have read the foregoing transcript and that the same and accompanying amendment sheets, if any, constitute a true and complete record of my testimony.

_____

Signature of Deponent

( ) No Amendments

( ) Amendments Attached

Subscribed and sworn to before me this _____ day of _____, 2009.

Notary Public: _____

Address: _____

_____

My commission expires _____

Seal:

JLP

STATE OF COLORADO)
) ss. REPORTER'S CERTIFICATE
COUNTY OF DENVER)

I, Janet Lee Priestley, do hereby certify that I am a Registered Professional Reporter and Notary Public within the State of Colorado; that previous to the commencement of the examination, the deponent was duly sworn to testify to the truth.

I further certify that this deposition was taken in shorthand by me at the time and place herein set forth, that it was thereafter reduced to typewritten form, and that the foregoing constitutes a true and correct transcript.

I further certify that I am not related to, employed by, nor of counsel for any of the parties or attorneys herein, nor otherwise interested in the result of the within action.

In witness whereof, I have affixed my signature and seal this 12th day of October, 2009.

My commission expires October 29, 2009.

_____

Janet Lee Priestley
216 - 16th Street, Suite 650
Denver, Colorado 80202

- AMENDMENT SHEET -

Video Deposition of DAVID PYATT

October 8, 2009

Zendejas vs. Shell

Case No. CV-07-005399

The deponent wishes to make the following changes in the testimony as originally given:

Page	Line	Should Read	Reason
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Signature of Deponent: _____

Subscribed and sworn to before me this ____ day of _____, 2009.

Notary's signature _____

(seal) Notary's address _____

_____

My commission expires _____.