

1
2 IN THE SUPERIOR COURT OF THE STATE OF ARIZONA
3 IN AND FOR THE COUNTY OF MARICOPA

4 Case No. CV-07-005399

5 VIDEO DEPOSITION OF DAVID PYATT October 8, 2009
6

7 RAUL ZENDEJAS and ARACELI ZENDEJAS,

8 Plaintiffs,

9 vs.

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

17 Defendants.

18 APPEARANCES:

19 SHRADER & ASSOCIATES

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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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1 P R O C E E D I N G S

2 THE VIDEOGRAPHER: We're on the record at
3 9:34 a.m. Today is October 8, 2009. This begins the
4 videotape deposition of David Pyatt Ph.D. taken by
5 the plaintiff in the matter of Raul Zendejas and
6 Araceli Zendejas vs. Shell Oil Company, et al. We're
7 located at Embassy Suites, 7001 Yampa Street, Board
8 Room 1, in Denver, Colorado. The court reporter is
9 Janet Lee Priestley, and the videographer is Nick
10 Borgia.

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

13 MR. PATTON: Keith Patton for plaintiff.

14 MR. PERRY: Stan Perry for shell.

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

17 DAVID PYATT,
18 being first duly sworn in the above cause, was
19 examined and testified as follows:

20 EXAMINATION

21 BY MR. PATTON:

22 Q Please introduce yourself for the record.

23 A David Pyatt.

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

25 A I have a Ph.D. in toxicology.

1 Q Okay. Dr. Pyatt, you are a toxicology
2 expert, correct?

3 A Yes, sir.

4 Q Were you hired by Shell in this case?

5 A I was hired by. Shell and I think when I
6 first got involved, ConocoPhillips might have been
7 involved, as well.

8 Q Dr. Pyatt, were you hired by Shell to give
9 an opinion in this case?

10 A Yes.

11 Q Have you testified or been retained on
12 behalf of Shell in other benzene exposure cases?

13 A There was other case for sure that I
14 worked with Mr. Perry on. Shell may have been a
15 defendant in some other cases that I've worked on,
16 along with others, but I don't specifically recall
17 that. But I wouldn't say absolutely that they have
18 not been involved.

19 Q Can you explain to us your experience in
20 the area of benzene in working with industry?

21 A Working with industry?

22 Q And by example, sir, today you're working
23 with industry?

24 MR. PERRY: Object to form.

25 Q (By Mr. Patton) Do you agree?

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

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

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

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

18 A Three.

19 Q And are any of those active right now?

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

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

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

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

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

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

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

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

2 Q Okay.

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

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

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

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

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

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

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

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

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

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

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

11 Q Did your -- I'm sorry.

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

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

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

25 Q Children are exposed to benzene above

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

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

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

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

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

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

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

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

12 A Not per se, no.

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

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

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

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

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

10 Q Okay.

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

13 Q I understand.

14 A -- counting diseases.

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

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

21 Q So you didn't find an increased risk?

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

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

5 Q What were the second and third studies you
6 did?

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

16 Q For the API?

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

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

1 A No.

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

7 MR. PERRY: Object to form.

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

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

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

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

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

16 A Yes.

17 Q It dealt with children?

18 A Yes.

19 Q It focused on children?

20 A Correct.

21 Am I providing too much detail. I can --

22 Q It's all right.

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

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

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

6 A Yes.

7 Q Are you involved in that?

8 A No.

9 Q Do you know why you're not involved?

10 MR. PERRY: Object to form.

11 A No.

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

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

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

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

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

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

12 MR. PERRY: Object to form.

13 A Yes, yes.

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

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

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

23 A Yes.

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

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

4 Q Is benzene a poison?

5 A Benzene can be toxic.

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

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

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

25 Q Is there something of a loftier objective

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

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

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

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

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

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

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

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

3 Q Okay.

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

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

15 A Sure.

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

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

24 Q It's probably not even a word?

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

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

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

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

16 Q Right.

17 A -- dose response information.

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

23 A Yes.

24 (Exhibits 1 through 3 marked.)

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

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

10 MR. PERRY: Object to form.

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

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

18 A Well, I'm a toxicologist.

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

25 MR. PERRY: Object to form.

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

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

6 A I'm 51.

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

11 MR. PERRY: Object to form.

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

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

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

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

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

10 MR. PERRY: Object to form.

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

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

15 A Say that again.

16 Q Chemical exposure as a cause of disease or
17 illness.

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

20 A I'm not --

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

23 A Please.

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

1 testified in, correct?

2 A Yes, sir.

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

6 A I have not yet, no.

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

9 A So far, yes.

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

14 A That's right.

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

17 How many of these cases involve benzene?

18 A They all do.

19 Q All 20 of them?

20 A Yes, sir.

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

23 A No; not in those 20, no.

24 Q Okay. Is it leukemia in all these 20?

25 A No, sir.

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

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

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

6 A Right now?

7 Q Sure.

8 A Okay.

9 Q You can write it next --

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

12 Q Let's do it out loud.

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

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

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

1 THE REPORTER: Say that again.

2 MR. PATTON: Worry about it later.

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

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

8 A Right.

9 Q It's still a myeloid disease, right?

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

18 Q Sure.

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

21 THE REPORTER: I can't hear you.

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

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

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

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

11 A Sometimes, not always.

12 Q What do you mean, "not always"?

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

20 Q Mineral spirits, do those contain benzene?

21 A At low levels, yes.

22 Q Does toluene contain benzene?

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

25 Q Crude oil contains benzene, yes?

1 A Crude oil has benzene in it.

2 Q Gasoline contains benzene, correct?

3 A Yes.

4 Q You mentioned earlier part of your work as
5 a toxicologist involves an interest in underlying
6 disease progress. You remember saying that?

7 A Process.

8 Q Underlying disease process. Okay. Would
9 you agree with me that diseases such as the myeloid
10 leukemias, diseases -- I only want to talk about the
11 myeloid leukemias, okay?

12 A Okay.

13 Q Would you agree with me, Dr. Pyatt, that
14 the myeloid leukemias, they do progress through a
15 series of different myeloid disorders before they
16 ultimately arrive at a firm diagnosis? And then once
17 that happens, they might progress to a different
18 myeloid firm diagnosis? Would you agree with that?

19 MR. PERRY: Object to form.

20 Q (By Mr. Patton) I probably didn't phrase
21 that real good, but do you understand what I'm
22 asking?

23 A Kind of.

24 Q Well, let me ask it in a different way.

25 A Okay.

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

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

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

8 A I don't think so, no.

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

11 (Exhibit 4 marked.)

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

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

18 A I think I do, yes.

19 Q What does he have?

20 A I think he has atypical CML.

21 Q As we sit here today?

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

25 Q Okay. Did you review the deposition of

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

3 A Yes.

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

6 A Yes.

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

10 A No.

11 MR. PERRY: Object to form.

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

14 A No.

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

17 A No, sir.

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

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

25 Q Have you ever formally diagnosed anyone

1 with any type of leukemia?

2 A No.

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

7 A No.

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

12 A I recall that.

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

16 A I did see that, yes.

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

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

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

5 Q He's got AML, right?

6 MR. PERRY: Object to form.

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

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

13 A (Deponent complied.)

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

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

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

20 A Okay.

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

24 A Yes.

25 Q Is it your opinion in this case that

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

2 A Yes.

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

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

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

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

20 Q I am.

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

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

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

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

13 A Yes.

14 Q And what did you find?

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

17 Q Hold on. Hold on.

18 MR. PERRY: Let him finish.

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

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

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

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

22 MR. PERRY: Object to form.

23 A Ask that again.

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

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

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

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

15 MR. PERRY: Object to form.

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

18 A No.

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

22 THE DEPONENT: Sure, you bet. Sorry.

23 MR. PERRY: Object to form.

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

25 A No --

1 Q -- the question again, Doctor.

2 A -- because many times AML is diagnosed
3 de novo. Sometimes there is a -- sometimes there is
4 a progression. I'm not arguing that the progression
5 can occur. And frequently people that are diagnosed
6 with myelodysplastic syndrome, based on the form,
7 some percentage will convert to AML. But I think
8 those people will show up in the studies as MDSs.

9 Q Why do you believe that?

10 A Because that's when they would have gone
11 to the doctor, and that's when they would have been
12 pulled into these studies as a myelodysplasia.

13 Q So let's say somebody goes into the study
14 as an MDS. The study's published. When the person
15 goes back to the doctor two months later, they're
16 diagnosed with AML.

17 A That's possible.

18 Q Do the study authors ever go back to the
19 study and redraft and say, Hey, by the way, you know,
20 we just looked at these people who were subjects of
21 the study. They're not MDSs anymore. They're AMLs.
22 Has that ever happened?

23 A I don't think it's needed.

24 Q Okay. What about CML? Would you agree
25 that some patients get CML and then they end up with

1 AML?

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

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

14 MR. PERRY: Object to form.

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

17 A Historically.

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

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

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

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

11 MR. PERRY: Object to form.

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

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

16 A They might have put that in there, yes.

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

19 A That, I don't know.

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

22 MR. PERRY: Object to form.

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

1 Mr. Zendejas actually went through.

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

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

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

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

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

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

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

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

13 A Not at all.

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

16 MR. PERRY: Object to form.

17 A I think that depends on the refinery.

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

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

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

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

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

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

12 Q Who performed that study?

13 A Which study?

14 Q The Pliofilm cohort.

15 A The first one?

16 Q Yes.

17 A I mean, there's like 30.

18 Q Dr. Infante was involved in that, correct?

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

25 Q In any event, the Pliofilm cohort helped

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

3 A That is my understanding, yes.

4 Q It's my understanding, too.

5 And is Pliofilm benzene?

6 A Pliofilm is rubber hydrochloride.

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

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

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

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

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

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

11 MR. PERRY: Object to form.

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

15 Q (By Mr. Patton) Yes, sir.

16 A And the answer is, or course.

17 Q Of course there is individual susceptible?

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

25 Q Because we all respond differently to

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

4 MR. PERRY: Object to form.

5 A I was talking more generally.

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

8 A About benzene specifically?

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

12 MR. PERRY: Object to form.

13 A It depends on the end point.

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

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

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

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

6 MR. PERRY: Object to form.

7 A That would depend on the dose.

8 Q (By Mr. Patton) Does benzene --

9 A It would depend on the exposure duration.

10 MR. PATTON: Objection, nonresponsive.

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

13 A It can.

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

16 A That depends on the dose.

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

20 A In experimental animals, it depends -- you
21 know, I could quibble. It would depend on your
22 definition of a harmful effect. But in experimental
23 animals, you know, the levels were pretty high,
24 300 PPM, five days a week, et cetera, and then you
25 saw pretty severe toxicity in their bone marrow.

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

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

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

11 A Yes.

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

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

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

18 A Correct.

19 Q What is cytopenia?

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

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

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

7 A In that study it was 7.2 PPM --

8 Q Okay.

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

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

16 A Yes.

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

22 MR. PERRY: Object to form.

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

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

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

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

11 MR. PERRY: Object to form.

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

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

20 MR. PERRY: Object to form.

21 A Yes.

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

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

3 MR. PATTON: Benzene.

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

5 MR. PATTON: Benzene, sir.

6 MR. PERRY: Object to form, unfounded.

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

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

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

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

14 THE VIDEOGRAPHER: We're on the record at
15 10:39. This is the beginning of Tape 2.

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

18 A No.

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

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

23 Q Okay. But as far as --

24 A Ethanol alcohol can.

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

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

3 A That's true.

4 Q What is it in the cocktails that causes
5 intoxication?

6 A Ethanol.

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

11 A Both.

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

16 A Yes.

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

20 A No.

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

25 A You'll have to define "intoxication."

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

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

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

17 A Okay.

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

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

22 Q Let's say it's 1.0.

23 A Okay.

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

1 drunk, according to the police?

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

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

12 A That I think is fair, yes.

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

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

20 Q Yes, sir.

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

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

25 A There doesn't appear to be much

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

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

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

14 A Sure.

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

20 A We're talking about inhalation,
21 breathing --

22 Q Sure.

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

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

6 But, I mean, if you said here's
7 100 people, here's 100 molecules of benzene into each
8 one of those people's lungs, how much of that
9 100 molecules of benzene are to get into each of
10 those people's bloodstreams, I've never seen any data
11 to indicate that there would be any difference in
12 that.

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

17 A I've never seen anything contrary to that.

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

20 A Correct.

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

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

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

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

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

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

22 Q I'm not challenging you on that.

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

1 to show that it's different.

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

6 A It's exactly like that.

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

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

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

16 A Yes.

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

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

21 Q Okay.

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

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

1 A Correct.

2 Q Before we talk about the bone marrow, does
3 some of it go anywhere else?

4 A Well, it's in the systemic circulation so
5 it's going to go anywhere the blood's going.

6 Q Okay. So by that time, benzene has
7 metabolized into the blood and into the bone marrow,
8 correct?

9 A By that time, the metabolites have left
10 the liver. Some of those have been conjugated, and
11 they're being excreted. That's part of this process
12 because that's how they get transported to the
13 liver -- to the kidneys and throughout. Some of them
14 are in transit anywhere that the blood is taking
15 them. And then there are specific enzymes in the
16 bone marrow that can take those metabolites and
17 further metabolize them.

18 Q Okay. And so is there a uniform or a --
19 is it the same rate of benzene metabolism among
20 everyone?

21 A No.

22 Q Okay. That differs?

23 A Absolutely.

24 Q Okay. A kid who weighs 20 pounds at
25 infant age compared to a pro football linebacker,

1 they're going to metabolize benzene differently,
2 right?

3 A Probably not; probably not.

4 Q Probably not?

5 A No.

6 Q Not differently?

7 A No.

8 Q Where are we missing each other?

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

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

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

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

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

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

25 A There is an enzyme called NQ01, and NQ01

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

6 Q Okay. So --

7 A As such --

8 Q Go ahead.

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

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

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

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

23 A Sure.

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

1 correct.

2 A Yes.

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

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

13 Q My question isn't --

14 A I'm getting there.

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

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

24 Q For Asian folks, for Chinese people?

25 A Well, it would be the same. They just

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

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

10 A Benzene toxicity.

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

13 A That's correct.

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

16 A NQ01.

17 Q NQ01.

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

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

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

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

5 Q It has some merit?

6 A I think it does, yes.

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

9 A By industry? I can't answer that.

10 Q Ten years --

11 A I can tell you --

12 Q -- ago? 20 years ago?

13 A -- it was first published.

14 THE REPORTER: Excuse me.

15 THE DEPONENT: Sorry.

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

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

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

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

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

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

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

1 detoxify it as quickly, correct?

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

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

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

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

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

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

1 benzene?

2 A I would think so, yes.

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

6 MR. PERRY: Object to form.

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

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

24 A The level of benzene being metabolized?

25 Q Well, let me ask a better question.

1 A I don't know the answer to that.

2 Q If someone is drinking alcoholic
3 beverages, they can take a Breathalyzer and you can
4 evaluate the blood alcohol content of that person,
5 correct?

6 A Yes.

7 Q Okay. Can you examine a person's breath
8 and learn anything about benzene?

9 A You might be able to if you did it really,
10 really quickly. But you blow off benzene really
11 fast. So, you know, if you look in my breath today,
12 you might can say what might be in this room or what
13 I might have been exposed to when I went to the
14 restroom, but you're not going to learn anything
15 about what I was doing yesterday.

16 Q Okay. But if we took a snapshot of your
17 breath yesterday, we could tell if you were being
18 exposed to more benzene, correct.

19 A Perhaps.

20 Q Okay. So like today you're sitting in a
21 hotel conference room. If we were to detect your
22 breath, you certainly wouldn't -- would you -- or
23 would you expect to find any appreciable amount of
24 benzene?

25 MR. PERRY: Object to form.

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

3 Q (By Mr. Patton) Okay.

4 A You certainly could measure it.

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

10 MR. PERRY: Object to form.

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

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

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

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

24 A You asked two questions.

25 Q Can you answer them both?

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

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

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

1 you know, whatever -- whatever the situation is.

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

14 Q How did we --

15 A Sorry -- the scientific community.

16 Q I'm with you on that.

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

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

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

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

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

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

13 MR. PERRY: Object to form.

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

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

1 an acceptable amount of exposure is --

2 A That's --

3 Q -- in the blood?

4 A That's true.

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

11 A Yes.

12 Q With that end goal?

13 A We're doing it.

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

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

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

1 the purpose of the research?

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

13 Q Okay.

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

20 Q Is industry funding that?

21 A Yes.

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

1 benzene at the end of that research some?

2 MR. PERRY: Object to form.

3 A Would I -- would they support it?

4 Q (By Mr. Patton) Correct.

5 A Well --

6 MR. PERRY: Object to form.

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

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

11 A Okay.

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

15 A Okay.

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

22 A Yes.

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

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

3 A Uh-huh.

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

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

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

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

24 MR. PERRY: Object to form.

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

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

4 MR. PERRY: Object to form.

5 A That's my general understanding, yes.

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

8 A Yes.

9 Q And industry opposed those stricter
10 guidelines, correct?

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

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

18 A I did not.

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

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

25 (Exhibit 5 marked.)

1 A No.

2 Q (By Mr. Patton) Can you take a minute to
3 read through it?

4 A The whole thing?

5 Q Well, if you want to read the whole thing,
6 that's fine. But I want to point your attention to a
7 couple portions.

8 A Yeah. If you're going to quiz me on it,
9 I'd like to read it.

10 Q Okay.

11 A (Deponent examined exhibit.)

12 Q Actually, Doctor, I don't -- let me -- I'm
13 not going to ask you a question. I want to give you
14 a chance to read it.

15 A Okay.

16 Q But what I'd like to do is if I can give
17 you that document and another document, and then
18 we'll take -- and then I'll let you read them and
19 then ask you some questions after that. Can we do
20 that?

21 A Sure.

22 Q Okay. And I will show you what I will
23 mark as Exhibit 6, and this is an article called
24 "Benzene: an historical perspective on the American
25 and European occupational setting." This is a study

1 by Dr. Infante. Are you familiar with this?

2 A No.

3 Q Have you ever seen that article before?

4 A No.

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

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

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

13 A Okay.

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

17 A I consider myself an expert on benzene
18 toxicology --

19 Q Okay.

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

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

25 A I'll try.

1 Q Okay. Let's go off --

2 A Each one of the paragraphs?

3 Q Yes, sir.

4 A Okay.

5 MR. PATTON: Let's go off the record.

6 THE VIDEOGRAPHER: We're off the record at

7 11:11.

8 (Recess from 11:11 a.m. to 11:48 a.m.)

9 THE VIDEOGRAPHER: We're back on the
10 record at 11:48.

11 Q (By Mr. Patton) Dr. Pyatt, if you could
12 take a look at Exhibit 5, turn to Page 6. Did you
13 have a chance to read this document during the break?

14 A Yes. I read both of them.

15 Q Would you look at Page 6, please. It says
16 6 in the lower left-hand corner right there.

17 A Okay.

18 Q Do you see near the top in the left-hand
19 corner --

20 A What am I looking for?

21 Q The paragraph beginning "Certainly." Do
22 you see where it says, "Certainly we are not
23 considering maintaining exposures that are clearly
24 unsafe. Such risks are unacceptable and must be
25 eliminated by reducing exposure drastically or, if

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

3 A I see that paragraph, yes.

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

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

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

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

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

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

20 Q You think Shell would be for increased
21 safety --

22 A Correct.

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

25 MR. PERRY: Object to form.

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

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

17 MR. PERRY: Object to form.

18 A No.

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

21 A No.

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

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

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

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

17 A I can't answer that yes or no.

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

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

1 workers exposed to gasoline.

2 Q What do you mean, "meaningful increase"?

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

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

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

21 MR. PERRY: Objection to form.

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

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

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

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

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

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

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

9 A I've heard all those words, sure.

10 Q Okay. Have you heard the phrase?

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

13 Q Okay. Do you know what that means?

14 MR. PERRY: Object to form.

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

18 MR. PERRY: Object to form.

19 A No.

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

23 A I have had no reason to do that.

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

1 million years cumulative dose exposure in the
2 workplace?

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

5 MR. PATTON: Objection, nonresponsive.

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

8 A You didn't. Sorry.

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

11 A Okay.

12 Q Sir, do you believe there's an increased
13 risk for AML in individuals exposed to greater than
14 20 part per million of benzene exposure?

15 A The way you worded it, yes. At something
16 greater than 20 PPM years, there will be an increased
17 risk. The critical question is at what level above
18 20 are you going to start seeing that increased risk.
19 So I don't believe that 21 will get you there, but
20 somewhere above 20, yes, you're going to have an
21 increased risk of developing AML.

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

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

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

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

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

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

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

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

1 Q Besides the report from Petty?

2 A Right.

3 Q As we sit here today, do you have any
4 opinions on the range of exposure to benzene commonly
5 experienced by gasoline distribution workers?

6 A That is outside of my area of expertise.
7 But I will tell you that irrespective of what those
8 end up being, the literature is pretty clear that
9 those workers do not have an increased risk of
10 leukemia. So whatever it is, it's going to be
11 something less than a threshold amount where you're
12 going to start seeing an increased risk of leukemia,
13 AML, in the literature.

14 Q The report by Steve Petty indicates that
15 Raul Zendejas was exposed to an estimated amount of
16 25 PPM year cumulative dose to benzene. Is that your
17 understanding of his report?

18 A That's my understanding of his report.

19 Q Do you have any reason to disagree with
20 his report?

21 A Yes.

22 Q What are those -- what are your opinions
23 in that regard?

24 A Well, in order for that to have occurred,
25 since most of that exposure that I could tell,

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

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

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

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

8 Then he mistook -- I'm doing this from
9 memory. But he read something that Mr. Horton said
10 that he had done in his career, and he extrapolated
11 and said that Mr. Zendejas had done that same thing,
12 spilled gasoline on himself a hundred times. That's
13 incorrect. And at least in one of the calculations
14 that he had done, he did not account for the fact
15 that Mr. Zendejas was not working with gasoline
16 100 percent of the time, but only -- I think by his
17 own estimation, only 20 percent of the time, and the
18 rest of it was diesel fuel.

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

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

1 A Yes.

2 Q Do you have any opinions on what would be
3 a fair estimate of Raul's exposure?

4 A Something much less than that. I mean,
5 even if you just take the factors that I just told
6 you, you know, you divide that by 5; okay, 5 PPM
7 years, even that's really high. That's a lot. Over
8 an eight-hour time weighted average, it takes a lot
9 of benzene to get those numbers up. But even that I
10 think is probably too high.

11 Q Have you seen any data from Shell
12 estimating the amount of cumulative dose of benzene
13 Raul may have experienced in his work?

14 A From Shell?

15 Q Yes, sir.

16 A I have not, no.

17 Q Have you seen any evidence or any
18 documents from Shell of any air monitoring performed
19 at facilities similar to the McNeece Brothers
20 facility?

21 A That I couldn't tell you. I haven't seen
22 it, but I think it exists.

23 Q Have any Shell experts provided you with
24 any numbers or estimates of how much benzene Raul was
25 exposed to in his work?

1 A Not at this point. I haven't specifically
2 asked for that.

3 Q Are you an industrial hygienist?

4 A No, sir.

5 Q Have you ever performed an exposure
6 analysis where you've estimated how much benzene
7 someone is exposed to while working with gasoline?

8 A Yes. Actually, I have.

9 Q Tell us about that.

10 A Well, I mean, I was kind of on the
11 sideline, but it was looking at what people using
12 chainsaws -- tree trimmers using chainsaws with
13 gasoline -- gasoline-driven chainsaws, what their
14 personal exposures to benzene would be from using a
15 gasoline-driven chainsaw. So we estimated it,
16 they -- we calibrated pumps. We looked at the data.
17 So yes, at least once, but I'm not an industrial
18 hygienist. I don't -- I'm not pretending to be one.

19 Q Have you ever -- are you familiar with
20 Dr. Neitzel?

21 A No.

22 Q Have you ever worked with him before?

23 A No. I'm not familiar with him.

24 Q Have you ever seen any of his work for
25 this case?

1 A No.

2 Q Do you have any understanding of what he
3 is going to -- what the purpose of his role in this
4 case is?

5 A Mr. Perry said something to me about it
6 yesterday, but I honestly don't remember, so no.

7 Q Dr. Ijazz Jamall, do you know him?

8 A I do not.

9 Q Have you ever worked with him before?

10 A No.

11 Q Have you seen any of his work in this
12 case?

13 A No.

14 Q Do you have an understanding of what he --
15 the subject of his testimony will be?

16 A Yes.

17 Q What is that?

18 A My understanding is that he will talk --
19 kind of follow the same lines that he wrote in his
20 paper about why gasoline is not just a vehicle for
21 distributing benzene to people but it is its own
22 unique chemical, and it has its own unique
23 toxicology. And I think he's going to, on a more
24 quantitative basis, evaluate Mr. Petty's report.

25 Q Have you seen any more specific

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

2 A I just understand that he's doing it.

3 Q Did you ask him for that information?

4 A I've never spoken with him.

5 Q What if Raul was exposed to 5 PPM years?

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

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

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

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

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

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

5 MR. PERRY: Object.

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

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

13 A No.

14 MR. PERRY: Object to form.

15 Q (By Mr. Patton) Why not?

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

1 groups think gasoline is carcinogenic.

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

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

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

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

25 THE DEPONENT: I thought you were going to

1 object.

2 MR. PERRY: Objection.

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

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

7 Q Well, he had 10 seconds that he could have
8 --

9 A -- that we have to --

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

12 THE DEPONENT: Sorry.

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

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

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

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

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

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

19 A He only did it for two years. It was in
20 2004, 2005, 2006. So we're not talking about
21 historical exposures back in the '40s and '50s. I
22 don't know what the numbers are.

23 Q Sir, the man worked from 2000 to 2006.

24 A Well, he was a truck driver from '04 to
25 '06.

1 Q Did you -- did you read his deposition?

2 A I did.

3 Q Did you read the other depositions in the
4 case?

5 A Yes.

6 Q Did you see where he testified that he
7 loaded fuel before he was a driver?

8 A That's true.

9 Q Would you agree with me that he's exposed
10 to benzene while loading fuel?

11 MR. PERRY: Object to form. It depends on
12 the fuel.

13 MR. PATTON: On gasoline.

14 A Well, I mean, he's going to be -- there's
15 going to be benzene exposure in any of these things,
16 just like there's benzene exposure from drinking that
17 Diet Coke, so that's a given. But yes, you're right,
18 he did testify that during that four-year period --
19 that's fine. Six years.

20 Q (By Mr. Patton) Then why did you say two
21 years earlier?

22 A Well, because I was thinking about being a
23 truck driver and loading gasoline in those big tanker
24 trucks. That's where -- that's where I thought your
25 question was going.

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

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

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

10 A That, I couldn't answer.

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

15 A Well, the only --

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

20 A Were you done?

21 Q Yes.

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

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

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

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

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

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

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

19 MR. PERRY: Object to form.

20 A No.

21 Q (By Mr. Patton) Why not?

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

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

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

18 A I guess that's how the saying goes.

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

24 MR. PERRY: Object to form.

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

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

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

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

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

1 as a result; possible?

2 MR. PERRY: Objection.

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

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

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

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

2 MR. PATTON: Objection, nonresponsive.

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

5 A Yes.

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

9 A It depends on the form of leukemia.

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

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

16 Q Let's get the Hayes study out.

17 A Sure. This is the whole NCI series.

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

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

1 '80s. It was a mess. They came in, tried to make
2 sense of this mess, but it was not an easy task. So
3 before I list any criticisms, it's not of these
4 investigators. It's that this was a really difficult
5 task.

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

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

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

22 A No.

23 Q Why not?

24 A I don't think I have it.

25 Q Have you read any of his work in other

1 cases?

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

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

7 A Say that again.

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

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

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

21 A No.

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

25 MR. PERRY: Object to form.

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

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

6 A No.

7 Q You disagree with that?

8 A Yes.

9 Q Why do you disagree with that?

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

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

22 A Yes.

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

25 MR. PERRY: Object to form.

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

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

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

11 Q What do you disagree with?

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

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

23 A I am not.

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

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

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

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

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

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

17 A Uh-huh. I have that.

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

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

22 Q But Raul was exposed to gasoline, correct?

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

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

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

10 A Say that again.

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

16 A Yes.

17 Q Can you give us some examples?

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

25 Q That's pretty simple.

1 A Well, you asked for an example.

2 Q No. I --

3 A I'm giving you an example.

4 Q I'm not --

5 A I think --

6 THE REPORTER: Excuse me.

7 A I think the guy's hydrochloric acid
8 exposure caused his loss of taste.

9 Q (By Mr. Patton) Okay. Give me some other
10 examples where you've concluded that a man's or
11 woman's exposure to Chemical or Substance X caused
12 Disease Y.

13 A I'm trying to think of when I've been
14 asked. That's the only time that I can think that
15 anyone has specifically asked. Now, there have been
16 some cases, examples, where people have called me and
17 said, Will you take a look at this, and I say yes. I
18 take a look at it, and they send me the information.
19 And they call me back, and I say, I don't know.
20 It -- maybe it did, maybe it didn't. I don't -- I
21 cannot defend that it did not. And then so -- to my
22 way of thinking I said, yeah, it could be. It could
23 be. So that's happened several times in my career,
24 but it did not happen in this case.

25 Q So if I understand your testimony,

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

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

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

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

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

12 Q Do you feel good about your job?

13 A Yes.

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

17 A Today?

18 Q Yes, sir.

19 A Working today, '09, no.

20 Q You don't have any opinions?

21 A I don't know.

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

25 MR. PERRY: Object to form.

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

9 MR. PATTON: Objection, nonresponsive.

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

14 A It would be pretty speculative on my part.

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

19 A As associated with a benzene exposure?

20 Q Yes, sir.

21 A They would really be essentially the same
22 question.

23 Q And you don't know?

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

1 Q Do you have an opinion as to what amount
2 of exposure to benzene is necessary for it to be a
3 causative factor in someone's AML?

4 A Yes.

5 Q Tell us that.

6 A I think the quantitative epidemiology
7 supports 40 part per million years at the low end.

8 Q Okay. Must those people exposed to 40 PPM
9 years be exposed to pure benzene?

10 A No.

11 Q They can be exposed to benzene as a
12 constituent in other chemicals?

13 A Yes.

14 Q But not gasoline, right?

15 A Well --

16 Q Is that your opinion?

17 A I don't know that you could achieve those
18 with gasoline, quite honestly, without the other
19 chemicals in gasoline being acutely toxic and someone
20 passing out or dying from it. But gasoline has its
21 own set of epidemiology. So while gasoline has
22 benzene in it, I don't think anyone is ever going to
23 argue that it doesn't. It seems counterproductive to
24 ignore this binder -- wherever it went -- this binder
25 of gasoline studies and say, Well, we're going to

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

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

7 MR. PERRY: Object to form.

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

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

16 A Say that again.

17 Q If someone were --

18 A Go ahead.

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

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

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

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

18 A That wouldn't matter.

19 Q Why not?

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

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

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

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

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

17 MR. PERRY: Object to form.

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

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

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

1 through the literature to see. I mean, there's --

2 Otto Wong looked at 18,000 distribution workers.

3 Lesley Rushton looked at 20,000 distribution workers.

4 Rob Schnatter looked at distribution workers.

5 Sorahan looked at distribution workers. I've got to

6 believe that a lot of those people that they pulled

7 into those studies would fit into that category.

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

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

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

15 MR. PERRY: Object to form.

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

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

20 A Okay.

21 Q Let's say we have two gasoline
22 distribution terminals 10 miles away from each other,
23 okay, and at one of the terminals the men are loading
24 in -- they're loading with vapor recovery, okay.

25 That's to mean they are exposed to fewer gasoline

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

9 A Okay.

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

12 MR. PERRY: Objection.

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

15 MR. PERRY: Objection to form.

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

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

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

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

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

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

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

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

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

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

1 Go ahead and do it again.

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

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

9 A We've done this three times now.

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

13 A Well, no.

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

22 MR. PATTON: Objection, nonresponsive.

23 MR. PERRY: Objection, nonresponsive.

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

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

1 once before, and what I said --

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

4 A Okay.

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

7 A Take as long as you need.

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

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

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

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

16 Q So if someone came to you with solid
17 evidence that a worker was exposed to benzene above
18 40 PPM years, would you then be willing to give the
19 opinion that that benzene exposure was a causative
20 factor in his AML? Would you be willing to do that?

21 A Perhaps; other considerations, but
22 perhaps.

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

1 or no?

2 A Yes. That is my opinion --

3 Q Okay.

4 A -- as based on the scientific literature.

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

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

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

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

4 Q Does the literature say anything about
5 gasoline causing AML?

6 A Oh, sure.

7 Q What does it say?

8 A It says it doesn't happen.

9 MR. PATTON: Okay. Off the record.

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

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

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1 AFTERNOON SESSION 1:40 p.m.

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

4 EXAMINATION (continued)

5 BY MR. PATTON:

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

11 MR. PERRY: Object to form.

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

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

20 MR. PERRY: Object to form.

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

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

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

7 A I don't know the answer to that.

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

12 A No.

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

16 A No.

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

19 A No.

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

23 A I have no opinion about that.

24 Q Do you think they should be warned about
25 anything?

1 MR. PERRY: Object to form.

2 A Who?

3 Q (By Mr. Patton) Gasoline distribution
4 workers.

5 A Who should warn them? I mean --

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

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

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

18 A In humans --

19 Q Yes.

20 A -- or in experimental animals?

21 Q Humans.

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

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

4 Q How much is a lot?

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

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

10 MR. PERRY: Object to form.

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

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

25 A That's correct.

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

5 A No.

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

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

14 Q And he works for Shell?

15 A Uh-huh.

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

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

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

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

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

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

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

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

22 MR. PERRY: Object to form.

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

1 Q (By Mr. Patton) It's all right.

2 Do you require scientific certainty in
3 terms of causality for every specific
4 lymphohematopoietic disease related to exposure by
5 benzene by exposure level?

6 MR. PERRY: Object to form.

7 A Are you asking whether -- what my criteria
8 would be before I could say that benzene can cause a
9 disease?

10 Q (By Mr. Patton) I'm saying would you
11 require scientific certainty in terms of causality
12 for every subtype of a leukemia?

13 A Of leukemia?

14 Q Of a given -- let me start again.

15 A Okay.

16 Q For you to testify that benzene caused
17 someone's leukemia -- and again, you -- besides the
18 dental example you've never testified or given the
19 opinion that Chemical X caused Disease Y, correct?

20 MR. PERRY: Object to form.

21 A Well, like I said, there have been three
22 or four times when I've had that opinion, and then
23 they either settled the case or went somewhere else.

24 Q (By Mr. Patton) You didn't say that
25 earlier, sir.

1 MR. PERRY: Yes, he did.

2 A Yes, I did.

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

8 A No.

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

12 A Well --

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

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

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

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

4 Q What were the chemicals in question?

5 A Benzene.

6 Q What kind of exposure was it? Pure
7 benzene?

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

10 Q What were the diseases?

11 A The diseases were AML.

12 Q What kind of AML?

13 A That, I don't recall.

14 Q Wouldn't that matter to you?

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

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

1 A No.

2 Q Do you have any opinions as to what Raul
3 should have done differently?

4 A No.

5 Q Do you have any criticisms of the way Raul
6 worked with gasoline?

7 A Well, I mean, just kind of general. If
8 what Stephen Petty said was true and he really did
9 spill gasoline on himself 100 times while he's
10 loading up tanks, I might say you might want to be a
11 little more careful and pay attention to what you're
12 doing.

13 Q But from a leukemia standpoint, if
14 gasoline can't cause leukemia anyway --

15 A No, I agree.

16 Q -- what does it matter if he spills any on
17 himself?

18 A I agree.

19 Q Okay. So is it your opinion that it
20 doesn't matter how much Raul -- or how he was working
21 with the gasoline or even if he spilled it on
22 himself, because it still wouldn't cause his form of
23 leukemia? Is that your opinion?

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

1 Q And therefore that is your opinion?

2 A Based on the literature, correct.

3 Q Why do you always qualify it, "based on
4 the literature"?

5 A Because I want the jury to understand that
6 my opinion is solidly based in the literature. This
7 is not an authority based, look at me, I've got all
8 these publications, I know this much about benzene.
9 My opinions in this case are based on these binders
10 of studies.

11 (Exhibit 7 marked.)

12 Q (By Mr. Patton) Let's talk about some of
13 your studies. I've marked as Exhibit 7 a study about
14 occupational hobby or lifestyle exposures associated
15 with Philadelphia chromosome positive CML. Is that
16 what Mr. Zendejas had?

17 A No.

18 Q Okay.

19 A But it was a lot closer to him than AML.

20 (Exhibit 8 marked.)

21 Q (By Mr. Patton) Exhibit 8, "Occupation
22 and Leukemia Mortality Among Men in 16 States." Are
23 you familiar with this study, Loomis?

24 A I can't tell from you holding it way over
25 there. I mean, it was one of my papers that I

1 referenced in my report so I'm --

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

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

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

11 A Same thing.

12 Q What is it?

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

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

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

1 David Savit?

2 MR. PATTON: Yes.

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

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

13 (Exhibit 9 marked.)

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

16 A Yes.

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

19 A Correct.

20 Q Is Mr. Zendejas a petrol worker?

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

25 Q Would you agree with me that someone who

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

4 A I don't have an opinion either way.

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

8 A That's correct.

9 Q Statistically significant, correct?

10 A Yes.

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

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

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

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

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

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

8 Q Yes.

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

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

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

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

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

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

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

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

24 Q What other chemicals impact on benzene's
25 metabolism?

1 A I think all of the aromatic hydrocarbons
2 do --

3 Q Okay.

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

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

11 MR. PERRY: Object.

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

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

24 A That, I don't know.

25 Q If they did, it wouldn't make sense

1 because of competitive inhibition, right?

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

9 (Exhibit 10 marked.)

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

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

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

16 A Okay.

17 Q "The odds ratio among those ever exposed
18 to benzene was 1.7, 95 percent confidence interval,
19 1.1 to 2.6." So that is statistically significant,
20 correct?

21 A That's correct.

22 Q For total leukemia?

23 A That, I don't remember.

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

25 A Okay.

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

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

6 Q It shows the ever exposed category had a
7 significantly increased risk for CML. Odds ratio
8 2.5, a 95 percent confidence interval, 1.3 to 4.9.
9 That's statistically significant, correct? Here you
10 go.

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

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

15 MR. PERRY: Is it correct or incorrect?

16 Q (By Mr. Patton) Is it correct?

17 A Let's see. The ever exposed category had
18 a significantly increased risk for CML. Odds ratio
19 2.5, 95 percent confidence interval, 1.3 to 4.8. So
20 yes, that is -- that one finding is statistically
21 significantly elevated.

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

25 A No.

1 Q No? Why not?

2 A Because when you look where they've
3 divided up the all leukemias, the less than 15 year
4 category, which Mr. Raul would -- I'm sorry --
5 Mr. Zendejas would fit into, that is not
6 statistically significant or even elevated and for
7 CML or all leukemias. And then like we talked about
8 earlier, the benzene category -- sorry -- the
9 gasoline category, which is the most pertinent to
10 this case and Mr. Zendejas, does not support an
11 increased risk of leukemia.

12 Q This is kind of a futile exercise, isn't
13 it? I mean, just as you've done in the other
14 20 cases that you've testified for on behalf of
15 industry, you and I could go through all these
16 studies, and you could just carve it out as you see
17 fit? And that's what you're doing, correct?

18 MR. PERRY: Object to form.

19 A I don't think I'm carving it out at all.
20 I mean, like I said, I will show the jury and they
21 will see the word "gasoline" and the risks associated
22 with gasoline exposure. I'm not carving it out.
23 That's what this study supports.

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

1 elucidated?

2 A Say that again.

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

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

10 (Exhibit 11 marked.)

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

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

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

24 (Exhibit 12 marked.)

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

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

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

17 (Exhibit 13 marked.)

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

1 A Yes. And Lesley is a woman.

2 Q What does it have to do with your
3 opinions, that study, Exhibit 13?

4 A Well, this is a -- this is a very
5 important study. This was looking at a large number
6 of distribution workers, which I think would be
7 relevant to Mr. Zendejas. They looked at various
8 forms of leukemia. They looked at various forms of
9 leukemia associated with cumulative exposures, peaks,
10 intensities, a variety of exposure metrics associated
11 with those workers. And there is a chart
12 specifically for chronic myeloid leukemia along --
13 associated with various benzene exposure metrics.

14 Q What kind of benzene exposure were these
15 workers experiencing via gasoline operations?

16 A You mean in terms of quantification?

17 Q Yes, sir.

18 A I don't -- well, yeah. I mean, there was
19 less than 4.5. There was a 4.5 to something. There
20 was a greater than 45 PPM years. I don't know
21 whether those were the distribution workers or not.
22 But at no point in that dose response was there an
23 increased risk of CML, which I think is directly
24 germane to what we're doing here.

25 Q So even when it shows like all leukemias,

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

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

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

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

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

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

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

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

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

11 (Exhibit 14 marked.)

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

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

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

25 A Yes.

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

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

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

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

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

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

22 A No.

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

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

4 MR. PERRY: Objection, form and vague.

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

10 THE VIDEOGRAPHER: One minute, Counsel.

11 MR. PATTON: Off the record.

12 THE VIDEOGRAPHER: We're off the record at
13 2:10. This is the end of Tape 3.

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

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

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

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

24 Q Schnatter worked for Exxon, right?

25 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

1 benefit industry when the studies specific to
2 gasoline are inconclusive?

3 MR. PERRY: Object to form.

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

8 MR. PERRY: Object to form.

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

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

13 A Absolutely.

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

17 A I think I had everything I needed.

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

21 A No.

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

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

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

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

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

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

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

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

18 MR. PERRY: Object to form.

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

21 MR. PERRY: Object.

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

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

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

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

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

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

18 (Exhibit 17 marked.)

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

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

1 you'll have to show it to me.

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

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

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

11 A My understanding is no, he did not.

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

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

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

1 chemically related, so to speak?

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

5 Q I'm talking about benzene.

6 A Oh.

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

9 A You don't see --

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

11 A I'm sorry about that.

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

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

25 Q So if someone had a myeloid leukemia and

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

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

7 Q What if it's MDS?

8 A Same.

9 Q The same.

10 A You want this one back?

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

12 (Exhibit 18 marked.)

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

17 A Okay.

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

23 A Okay.

24 Q But I don't want to get leukemia.

25 A Okay.

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

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

6 Q I can just go work with benzene all day?

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

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

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

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

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

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

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

11 MR. PERRY: Object to form.

12 A I disagree with that.

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

14 A Yes.

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

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

19 Q What about for all leukemias?

20 A I don't remember the numbers for all
21 leukemias. For AML, then the risks are statistically
22 significant at something greater than 8 but less than
23 57. So to me, that is consistent with many of the
24 other studies that show an increase somewhere around
25 40 to 50 PPM years.

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

4 (Exhibit 19 marked.)

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

7 Q (By Mr. Patton) Maybe.

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

10 Q I did, too.

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

13 A I can try.

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

16 A Okay.

17 Q -- of Exhibit 19. "Quantitative
18 assessment of exposure is difficult, posing
19 challenges to epidemiological investigation of
20 occupation/cancer relationships." Do you agree with
21 that?

22 A Yes.

23 Q "Whereas individuals usually can describe
24 their smoking histories and eating habits reasonably
25 well, they're often relatively ignorant about their

1 work exposures." Do you agree with that?

2 A Not completely. I mean, I think -- well,
3 they could describe their smoking, but I think
4 frequently they don't. But yes, it's hard to
5 measure -- the occupational exposure part of an epi
6 study is frequently the weakest part.

7 Q Right. I mean, you ask a guy what he ate
8 for lunch or how much he smokes, he can tell you
9 about that?

10 A Correct.

11 Q You ask him how many PPM he's exposed to
12 of benzene when he's working with whatever solvent, I
13 mean, you wouldn't expect a guy to know that, would
14 you?

15 A He wouldn't know, I agree.

16 Q "Moreover, because of long latency periods
17 between exposure and cancer diagnosis and because
18 working conditions change over time, data on current
19 workplace exposures may not reflect those experienced
20 by past workers," correct?

21 A Yes.

22 Q You agree with that?

23 A Yes.

24 Q And you would also agree that workplace
25 exposures in past data like some of the numbers we

1 might find in these studies you're relying on, those
2 might not accurately reflect exposure data for a
3 given situation like Yuma, Arizona, a small bulk
4 terminal, top-loading, no vapor recovery; would you
5 agree with that?

6 A Well, in terms of your specific scenario
7 with that case, I mean, all I could say is that even
8 if you accepted Stephen Petty's assessment, then yes,
9 that cumulative exposure is absolutely encompassed in
10 these studies. That is not something that's so far
11 out there that we don't have a study that would tell
12 us about the risk associated with those exposures.

13 Q So you would agree with me that the
14 studies -- some of the studies do show exposures at
15 the level that Petty estimates? However, you're
16 critical of Petty for other reasons?

17 A I would say that this literature does have
18 people that are exposed at that level but not from
19 working five years and not from being -- well, I'm
20 not sure about the tank truck drivers. But the ones
21 I'm thinking about are the Pliofilm workers, the
22 rubber workers, the shoe workers. These people have
23 really high benzene exposures.

24 Q "Epidemiologists" -- and I'm still reading
25 from Exhibit 19. "Epidemiologists often have had to

1 rely on proxy measures of exposure and studies of
2 occupational cancer." What does that mean, "proxy
3 measures"?

4 A Like duration.

5 Q Surveys?

6 A No. Like instead of actually having
7 quantitative air monitoring data that might be
8 lacking or problematic, they would just say, Okay,
9 well, this guy worked there for five years, these
10 guys worked there for ten years, all those people
11 over there worked there for 20 years, so let's see
12 whether or not there's changes in their risk. So in
13 that case, duration would kind of be a proxy for
14 exposure.

15 Q Reading on in Exhibit 19, "For example,
16 the epidemiological studies which showed that
17 physicians who specialized in radiology prior to 1950
18 were at an increased risk of leukemia because of high
19 radiation exposure relied primarily on knowledge of
20 typical radiation protection practices in different
21 time periods, rather than on specifics about the
22 exposure of particular individuals." Does that make
23 sense to you?

24 A No. I'd have to read it again and look at
25 it in the context of that.

1 Q "Difficulty in obtaining accurate exposure
2 data remains a problem today, hampering recent
3 efforts such as the determination of whether
4 occupational exposure to pesticides and
5 electromagnetics fields influence cancer risk." Do
6 you agree that difficulty in obtaining accurate
7 exposure data remains a problem today?

8 A It depends on what it is you're looking
9 for. Certainly something like electromagnetic
10 fields, that's really problematic; benzene, less so.
11 But in an epidemiological study, the exposure
12 assessment is always the weakest part.

13 Q Because the exposure assessment is always
14 the weakest part in these epi studies, doesn't it
15 make a little more sense to look at the group of
16 workers a little more specifically with a little more
17 care, so to speak, to draw conclusions?

18 A I don't understand. I think they have
19 done that.

20 Q Well, I mean, I think that's what
21 Dr. Carroll did and Dr. Gore did for Raul. I mean,
22 they look at -- looked at his specific circumstances.
23 It's in the medical records that the guy's exposed to
24 gasoline and hydrocarbons. They considered the
25 unlikelihood that he would get his disease at his

1 age, and they concluded that benzene caused it. Why
2 isn't that fair?

3 MR. PERRY: Object to form.

4 Q (By Mr. Patton) I mean, you've got all
5 these problems with these epi studies, but epi
6 studies are what you're relying on to disprove
7 causation. Why isn't it fair to do what Dr. Gore and
8 Dr. Carroll did?

9 A It's not just me. Epi studies are what
10 EPA, IARC, OSHA, ATSDR -- I mean, this is the gold
11 standard for what we're doing. These are human
12 studies with toxicity. The difference is whether
13 you're going to have an authority based kind of
14 opinion or whether you're going to have a science
15 based opinion. And I think that will be evident if
16 this goes to trial when they start asking, Well, what
17 is the scientific basis in support of your opinion.
18 And that's going to be lacking.

19 Q You believe there is a -- that the
20 scientific basis supporting those experts' opinion is
21 lacking?

22 A Yes.

23 Q Isn't it easier, though, to use the
24 epidemiology to carve out a certain disease or a
25 certain specific circumstance to say it did not cause

1 it? Isn't that easier to do than collect the
2 literature to say it did cause it?

3 MR. PERRY: Object to form.

4 A I don't -- I don't how to answer that
5 question. I don't think that I have carved out
6 anything. I have brought in the literature that is
7 related to gasoline that is related to CML. That's
8 what he was exposed to. That's what everyone said.
9 That's the disease that he had. That's agreed upon
10 with everyone. I don't -- I don't see how I've
11 carved out something specific out of the literature.

12 Q (By Mr. Patton) He's got AML. I mean, if
13 he died tomorrow, it's AML.

14 MR. PERRY: Object to form.

15 A We've gone through that. I mean, your own
16 expert said that he would call it blast crisis.

17 Q (By Mr. Patton) Dr. Carroll already
18 called it AML.

19 A Dr. Gore called it blast crisis, and
20 that's what -- if you pick up any hematology textbook
21 and you're talking about a person who has his disease
22 following atypical or typical CML, they will call it
23 blast crisis.

24 Q Your job is easier because you can quibble
25 with these -- with calling it atypical CML?

1 MR. PERRY: Object to form.

2 A I'm not sure about that. I don't think my
3 job is easy, although -- I take that back. In this
4 case, it was pretty straightforward.

5 Q (By Mr. Patton) Was it easy for you to
6 conclude that Raul's exposure to benzene via gasoline
7 did not cause his form of leukemia?

8 A I wouldn't say it's easy, but I think the
9 literature is less schizophrenic with regard to
10 gasoline and CML than it is a lot of other diseases.
11 I think that literature is pretty clear, pretty
12 clearly negative.

13 (Exhibit 20 marked.)

14 Q (By Mr. Patton) Exhibit 20, this is one
15 of the medical records you have. I circled the
16 portion about cytogenetics. Does that indicate he
17 had a translocation?

18 A In one cell, which in a cytogenetic point
19 of view is insufficient. They have to see it in at
20 least three cells, or they don't count it.

21 Q Are you a cytogeneticist or a pathologist?

22 A I took a lot of pathology. Pathology and
23 toxicology in my institution were hand in hand. And
24 I wasn't intimately involved with the studies, but we
25 published -- out of the lab that I worked in, we

1 published several cytogenetic papers on benzene
2 metabolites and various lymphocytes. I know the
3 person who did the cytogenetics. I mean, I'm not a
4 cytogeneticist, but I'm familiar with the techniques.

5 Q The term "atypical CML" did not exist
6 before 2000, correct?

7 A I think they called it Philadelphia
8 negative -- Philadelphia chromosome negative. And I
9 don't remember when they stopped doing that.

10 Q Because the definition atypical CML was
11 not even used until around 2000 or so, you wouldn't
12 expect that term to be reflected in the literature,
13 would you?

14 A I think that's right, yes.

15 (Exhibits 21 and 22 marked.)

16 Q (By Mr. Patton) Exhibit 21 is another
17 medical record.

18 Exhibit 22, upon diagnosis Dr. Fayssoux
19 and Dr. Abdella, they mentioned occupation and
20 habits: "He works as a delivery man or a truck
21 driver for diesel fuel and gasoline." Why should
22 they even bother to mention that?

23 A You'll have to ask them. I mean, I think
24 as Dr. Nadelson described in his deposition, that is
25 something that they typically do, take an at least

1 brief occupational history of people that have
2 leukemia.

3 (Exhibit 23 marked.)

4 Q (By Mr. Patton) Exhibit 23, Dr. Fayssoux
5 originally called Raul's disease a myeloid
6 proliferative disorder, MDS, right?

7 MR. PERRY: Objection, form.

8 Q (By Mr. Patton) MPD?

9 A A myelo -- a myeloproliferative disorder
10 is CML. CML is a myeloproliferative disorder.

11 Q It's a myeloid leukemia, a myeloid
12 illness, right?

13 A Which one?

14 Q MPD or CML or MDS, all of them?

15 A They are -- no. Okay. MDS --

16 Q Let me ask a better question.

17 A Okay.

18 Q Is MDS a bone marrow disease?

19 A Yes.

20 Q Is PMD a bone marrow disease?

21 A Yes.

22 Q Is CML a bone marrow disease?

23 A Yes.

24 Q Is atypical CML a bone marrow disease?

25 A Correct.

1 Q Is AML a bone marrow disease?

2 A Yes.

3 Q Is CMML a bone marrow disease?

4 A Yes. It's a form of MDS, or used to be.

5 THE DEPONENT: Is this the exhibit pile
6 here (indicating)?

7 MR. PERRY: Yes, sir.

8 THE DEPONENT: You want my report in that
9 pile?

10 MR. PERRY: Yeah. I've just been kind of
11 gathering them as we go.

12 THE DEPONENT: These are the ones that he
13 asked me to read.

14 Q (By Mr. Patton) Let's go through your
15 report.

16 A I need it back. Sorry.

17 Q First of all, do you know when benzene was
18 first associated with any form of bone marrow
19 disease? Do you know approximately what year?

20 A I can't tell you when it was first
21 associated. I can tell you when the first
22 publication came out.

23 Q When was that?

24 A 1897, I think it was.

25 Q And they drew the conclusion that benzene

1 is toxic to the bone marrow, correct?

2 A Yes.

3 Q And they drew that conclusion before we
4 have modern science today and before we started
5 classifying and reclassifying, for treatment and
6 prognosis purposes, the different forms of bone
7 marrow disease or myeloid diseases, correct?

8 MR. PERRY: Object to form.

9 A I don't understand that question. It was
10 aplastic anemia. It was a fairly specific diagnosis.

11 Q (By Mr. Patton) Do you know when benzene
12 was first connected with any type of myeloid leukemia
13 rather than aplastic anemia? Do you know what
14 decade?

15 A The first publication of benzene leukemia
16 was in 1828 -- sorry -- 1928, but it was actually a
17 lymphoid leukemia. So based on what we know now,
18 they were probably -- they were incorrect, and that
19 wasn't a benzene-related malignancy. But
20 nonetheless, that's when the first paper came out,
21 Delores and something.

22 Q "In summary, there is" -- this is Page 10
23 of your report.

24 A Okay.

25 Q "In summary, there is no consistent

1 evidence in the existing body of scientific
2 literature that occupational exposure to gasoline
3 results in an elevated risk of developing CML, MDS,
4 or any other human cancer." Do you still agree with
5 that?

6 A Absolutely.

7 Q I mean, we went through some of these
8 studies, and I pointed out to you a handful of them
9 that did show evidence of an increased or elevated
10 risk of developing leukemias. Didn't we do that?

11 A No, no. That's why no consistent
12 evidence -- and that's why Bradford Hill in his 1965
13 paper said, Here's what you've got to do in order to
14 establish causation. That was the whole idea.
15 Because we know that you're going to have, just by
16 random chance, a positive finding here or a positive
17 finding there. When you look collectively at that
18 overall literature, it does not support an
19 association.

20 Q Exhibit 5 or Exhibit 6 -- I'm not sure
21 which one it is.

22 A Okay. Well, I'll have to get those back.
23 I'm sorry.

24 Q It's Infante's paper.

25 A Okay. It's in here. I think it's on the

1 top, maybe just right under those medical reports.

2 All right. It's in there.

3 Q Exhibit 6.

4 A Okay.

5 Q Turn to Page 43.

6 A Okay.

7 Q See the upper right -- the right-hand
8 column? Or can you just start at the lower left-hand
9 corner. Can you read aloud for us, "In the 1970s."

10 A I'll try. "In the 1970s benzene
11 manufacturers and users began a new approach for
12 conveying knowledge about the toxicity of benzene to
13 the public, in general, and to workers and plant
14 managers specifically which contributed to a
15 continuation of overexposure to benzene. This is the
16 period when manufacturers began to hire consultants
17 to downplay the importance of scientific observations
18 related to the toxicity of benzene and to introduce
19 unresolvable arguments about dose response analysis,
20 which had an impact in delaying much-needed
21 government regulations that sought to reduce benzene
22 exposure in the workplace."

23 Q Let's stop right there.

24 MR. PERRY: Wait. What page is that?

25 THE DEPONENT: 43.

1 MR. PERRY: 43 of Exhibit 6.

2 Q (By Mr. Patton) Dr. Pyatt, manufacturers
3 like Shell have hired you as consultants, correct?

4 A I am a consultant, yes.

5 Q Do you believe it is fair to say that you
6 are downplaying the importance of the scientific
7 observations related to the toxicity of benzene? Do
8 you believe you're doing that?

9 A No, absolutely not. I think that's what
10 your experts are doing.

11 Q Do you believe you're introducing
12 unresolvable arguments about dose response analysis?

13 A No.

14 Q Do you believe to better understand the
15 relationship between -- well, let me look at it a
16 different way.

17 You have essentially three things going on
18 for a worker like Raul in a case like this. You have
19 benzene exposure, gasoline exposure, and him as a
20 gasoline distribution worker, okay? Wouldn't it be
21 helpful to study gasoline distribution workers who
22 worked in similar environments to him to better
23 understand the significance of benzene exposure to
24 that class of worker?

25 A We have; Schnatter, Rushton, Wong,

1 Sorahan, other refinery studies that have included
2 distribution workers.

3 Q Sir, I'll represent --

4 A We have.

5 Q I'll represent to you that none of the
6 studies that we went through talked about top-loading
7 without vapor recovery. Do you have any reason to
8 disagree with me?

9 A I don't know whether they specifically
10 addressed that or not. But when they're looking at
11 18,000 distribution workers, I would assume that some
12 of them are doing exactly what Mr. Zendejas did.

13 MR. PATTON: Off the record.

14 THE VIDEOGRAPHER: We're off the record at
15 2:46.

16 (Recess from 2:46 p.m. to 2:47 p.m.)

17 THE VIDEOGRAPHER: We're back on the
18 record at 2:47.

19 Q (By Mr. Patton) Dr. Pyatt, do you believe
20 that industry is responsible to anyone or any class
21 of workers who have suffered leukemia as a result of
22 chemical exposure?

23 MR. PERRY: Object to form.

24 A What do you mean, "responsible"? I don't
25 think --

1 Q (By Mr. Patton) Your answer says enough,
2 sir.

3 A Okay.

4 Q Do you believe there's a difference in
5 something like gasoline being called a carcinogen or
6 not and someone being exposed in a manner and at a
7 rate that exposes them to dangerous levels and an
8 increased risk?

9 MR. PERRY: Object to form.

10 A I'm not quite sure how to answer that.
11 But I would go back to what we've talked about
12 before, and that is even if you accept Stephen
13 Petty's quantitative exposure assessment, which
14 should take care of all of these other things that
15 you're addressing -- heat and Yuma and this, that,
16 and the other, top-loading -- it's not enough. It
17 just is not sufficient exposure.

18 Q (By Mr. Patton) Do you believe benzene
19 exposure played any role, even a 1 percent role, in
20 causing Raul's leukemia?

21 A I don't know how to answer that question.

22 Q Why not? Why can't you answer it?

23 A I just can't. I just don't know how to
24 say, well, it's a 6.5 percent probability or it's an
25 8 percent probability or it's 1 or 80. I just don't

1 know how to answer that. What I can tell you is the
2 scientific literature clearly does not support an
3 increased risk of CML from gasoline exposure. To me,
4 that's what this case is about, CML and gasoline.
5 And it does not establish a causal association.

6 Q Are there any other opinions that you
7 intend to offer in this case that are not described
8 in your report or that we haven't talked about?

9 A I don't -- I can't imagine what those
10 would be, no.

11 Q Have you been asked to testify at trial in
12 this case?

13 A Yes.

14 Q Do you know when that is?

15 A Yes, vaguely.

16 Q What's that?

17 A Yes.

18 Q Is there any additional information you'd
19 like to see in this case before you testify live in
20 front of a judge and a jury?

21 A The only thing that I would like to see
22 is -- and mainly just curiosity -- what Dr. Jamall
23 ends up saying about Stephen Petty's report and if he
24 does a cumulative exposure estimate.

25 Q Putting benzene aside, aren't there other

1 chemicals out there and other substances out there
2 for which there are no safe levels of exposure?

3 MR. PERRY: Object to form.

4 A I don't think that's true for benzene.
5 Well, that's what you said, putting benzene aside. I
6 mean, I do believe there are safe exposure levels to
7 benzene. I don't know of a chemical -- I mean,
8 methylmercury is really, really toxic, and it's
9 really, really toxic at really low levels. I don't
10 know whether you could actually be exposed to
11 measurable amounts of methylmercury and not have some
12 kind of toxicity, but I'm not positive.

13 Q (By Mr. Patton) What if someone had AML
14 with minus 5 minus, minus 7 chromosomes, and they
15 worked with gasoline for six years? Would you
16 believe that gasoline could have possibly played a
17 role in that person's disease?

18 A That's not what the literature would
19 support.

20 Q It's got to be pure benzene?

21 A It's got to be benzene at a high enough
22 level to put them into an increased risk based on the
23 quantitative epi. The other things, the minus 5 and
24 the 7, that's what we would expect the disease to
25 look like if it were, in fact, caused by benzene,

1 perhaps. You can't take those features and say,
2 Okay, because we've got these features, we don't care
3 about exposure anymore, it must have been caused by
4 benzene.

5 They're not that specific. People get 5
6 and 7, monosomy 7 and interstitial deletions in 5Q31.
7 They get that all the time without any exposure. So
8 that is just a description of what a person would
9 look like -- potentially look like if they had a
10 benzene induced leukemia.

11 Q Some of these studies in the exhibits,
12 they talk about the relationship between benzene and
13 specific types of leukemias or all leukemias. Some
14 of these studies don't even do a dose analysis, do
15 they?

16 A That's true. And from a public health
17 point of view, those studies are not very useful.

18 Q So all the studies that we've marked as
19 exhibits that don't look at benzene exposure in a
20 quantitative or an exposure or dose type viewpoint,
21 those just do not have much value to the public
22 health perspective; is that your testimony?

23 A No. I mean, if you -- you can look at --
24 say, you can do an epi study, and you can say, never,
25 ever, okay? And one of those that we talked about

1 did, never, ever; exposed to gasoline, yes; exposed
2 to gasoline, no. All right. So what if the answer
3 is yes; people who say, yes, I was exposed to
4 gasoline, I have an increased risk of lung cancer.

5 Okay, well, a public health official who
6 is tasked with setting an exposure level for gasoline
7 to protect workers from getting lung cancer, a yes/no
8 answer doesn't help them. A high, low, medium
9 doesn't help them unless someone can go in and say,
10 Okay, when they said "low," here's what the exposures
11 were. When they said "medium," here's what they
12 were.

13 So the more quantitative information you
14 have about dose, the more useful it is from a risk
15 assessment public health causation basis.

16 Q On those three cases that you said you've
17 told industry, Hey, I can't testify for you, did you
18 tell them that you thought benzene, 51 percent
19 chance, did play a role?

20 A What I told them was, I cannot rule it
21 out.

22 Q But if you -- could you not rule it out to
23 51 percent?

24 A They didn't ask, and I just -- I can't do
25 that. I mean, I don't know how to do this 8, 75.

1 You know, I just said I can't rule it out. I can't
2 say definitively that there is evidence to support
3 this person's disease wasn't caused by their
4 occupation.

5 Q Putting aside the person who had the
6 dental work and the hydrochloric acid, can you cite
7 for me another example where you've given the opinion
8 in literature or elsewhere in your work that exposure
9 to Chemical or Substance X caused Disease or Health
10 Effect Y?

11 A No, other than the ones that we've talked
12 about.

13 Q Okay. Thank you for your time.

14 A You're welcome.

15 EXAMINATION

16 BY MR. PERRY:

17 Q Can you please state your name.

18 A David Pyatt.

19 Q And what is your occupation?

20 Is it Mr. Pyatt or Dr. Pyatt?

21 A My family calls me David or Dad, but
22 students usually call me Doctor.

23 Q And do you have a Ph.D?

24 A I do, in toxicology.

25 Q And what is your current occupation?

1 A I am a toxicologist.

2 Q With which company?

3 A I started my own company in 2005 called
4 Summit Toxicology, but I have been with the
5 university since 1996 teaching, sitting on
6 committees, doing research, that kind of thing.

7 Q And it's October 8, 2009, and we're
8 outside of Denver, Colorado, correct?

9 A That's right.

10 Q And when you say "the university," which
11 university are you referring to?

12 A I'm talking about the University of
13 Colorado. We've only got one really big one here in
14 Colorado so that's probably why we say that.

15 Q And really, today we've been -- we started
16 your deposition this morning around 9:30 mountain
17 time. And there's been lots of questions about lots
18 of literature, and I want us to kind of focus on the
19 ultimate issue. And this is the question, Dr. Pyatt,
20 I'm asking you as a professional toxicologist with
21 university appointments, with membership in
22 professional societies dealing with toxicology, and
23 the question is this: Is it generally accepted in
24 the scientific community that gasoline exposure
25 causes leukemia?

1 MR. PATTON: Objection, form.

2 A No.

3 Q (By Mr. Perry) And why do you say that?

4 A Well, because if you put quotations around
5 generally accepted, how would you know whether
6 something is generally accepted? You know, did you
7 go out and do a survey of all the toxicologists?
8 Well, in fact, in this case, that is what has been
9 done. And IARC, ATSDR, The Agency For Toxic
10 Substances and Disease Registry, a bunch of
11 toxicologists, physicians, epidemiologists, they
12 don't believe gasoline is carcinogenic.

13 IARC, the International Agency for the
14 Research on Cancer, preeminent physicians,
15 toxicologists, epidemiologists that evaluate any
16 chemical that has the potential to be a human
17 carcinogen, they don't believe gasoline is a human
18 carcinogen, let alone causing leukemia. OSHA, United
19 States Environmental Protection Agency, the American
20 Conference of Government Industrial Hygienists -- I
21 mean, collectively that is a lot of really talented,
22 really smart epidemiologists and toxicologists that
23 collectively say gasoline is not a carcinogen. So I
24 believe saying it is not generally accepted is
25 totally supportable.

1 Q Is it generally accepted in the scientific
2 community that gasoline exposure causes CML or
3 atypical CML?

4 A Absolutely not. If you can't even say
5 that it causes cancer as a collective group of 200 or
6 so miscellaneous diseases, the answer to that is no.
7 There is -- no.

8 Q Is it generally accepted in the scientific
9 literature that benzene exposure, regardless of the
10 level of exposure, causes CML, including 1atypical
11 CML?

12 A That has not been shown in the scientific
13 literature. We know that high dose exposure to
14 benzene can cause acute myelogenous leukemia; some
15 types, certain situations, but it can happen. That
16 is the only form that has been clearly established.
17 And CML, there is -- there is no association with
18 high dose -- even high dose exposure to benzene in
19 CML.

20 MR. PERRY: No further questions.

21 EXAMINATION

22 BY MR. PATTON:

23 Q Dr. Pyatt, if it is so clearly established
24 in the relevant scientific community that exposure to
25 gasoline isn't going to cause any leukemia, why did

1 all these people and all these journals and all these
2 resources study benzene exposure in the context of
3 gasoline? And why did some of them find an increased
4 risk of certain types of leukemia among gasoline
5 distribution workers?

6 A Which one would you like for me to answer?

7 Q Both.

8 A Okay. Can you start again with the first
9 one? Why they do the studies with gasoline?

10 Q Let me back up.

11 A Okay.

12 Q If it is so widely accepted, as Mr. Perry
13 just asked you, in the scientific community that
14 gasoline exposure just doesn't cause leukemia, why is
15 it still studied in the context of gasoline
16 distribution workers? And more importantly, if it is
17 so widely accepted, how do you reconcile that with
18 the fact that some of these studies do show an
19 increased risk of certain types of leukemia among
20 gasoline distribution workers?

21 A Fine. The reason why I say it is widely
22 accepted in these scientific agencies -- I mean, a
23 lot of the data that's in this pile and a lot of the
24 data that's not in this pile that's in this binder,
25 that is why they know what they know. So this

1 literature -- I mean ATSDR, it was 2005 when they
2 came out. IARC's going to reevaluate everything this
3 year. I don't know if gasoline's on that list or
4 not. I actually don't think that it is. They feel
5 like they've -- they know enough about gasoline from
6 the last time they did it. The ACGIH, when they
7 write their TLVs, which they do every year, they
8 update it with the literature. So all of that
9 science has gone into this general acceptance.

10 Now, your question -- which now this is
11 probably the third time -- you can always find
12 spotty, you know, quirky findings here and there.
13 That Jakobsson was a real -- that is a positive
14 study. Jakobsson is a positive study, but it's
15 really small. And there was like three or four
16 leukemias that maybe were associated with petroleum
17 workers. When you look at it collectively, the
18 overall literature, it doesn't -- it's not
19 consistent. It doesn't hold up. And that's what you
20 have to do to say gasoline is or gasoline isn't a
21 carcinogen.

22 Q What gives you the authority, you
23 personally as Dr. Pyatt, to say that it's just not
24 widely accepted? I mean, where do you get that power
25 from?

1 A It's not my power. He asked the question.
2 And what I said was, ATSDR said no. There's no power
3 with me. I'm just telling you what they said. If
4 you don't believe it, you know, I can pull up their
5 Web site and show you what they say about gasoline.

6 Q We're misunderstanding each other here.
7 Do you believe it is widely accepted in the relevant
8 scientific community that gasoline exposure and
9 exposure to benzene as a gasoline distribution worker
10 does not create an increased risk of leukemia? Do
11 you believe that? Do you believe it's widely
12 accepted as to those -- what I just stated?

13 A That was kind of a double negative. I
14 believe it is widely accepted in the scientific
15 community, as evidenced by what we were just
16 discussing, that gasoline is not a carcinogen.

17 Q Okay. I heard that part already.

18 A Okay.

19 Q Do you believe it is widely accepted in
20 the scientific community that exposure to benzene
21 from gasoline as a gasoline distribution worker does
22 not cause or cannot cause an increased risk of
23 leukemia?

24 A I don't see that as being any different.

25 Q Okay. What gives you the power -- what

1 makes you the guru to say that it's widely accepted
2 as it relates to benzene via gasoline, specific to
3 gasoline distribution workers?

4 A There's no guruness here. I'm not being a
5 guru. You are emphasizing the benzene in the
6 gasoline, as if that is something unique to
7 Mr. Zendejas and it is not commonly found in every
8 other form of gasoline. Benzene is in gasoline in
9 every one of these studies. Benzene is in gasoline
10 that ATSDR evaluated. OSHA, US EPA, ACGIH -- all of
11 those have gasoline that has benzene in it.

12 Q Do you believe there's --

13 A Wait a minute. I'm not done yet.

14 Q Okay. I'm sorry.

15 A So collectively, all of those studies have
16 said that gasoline with benzene in it is not
17 carcinogenic. If it's not carcinogenic, it's not
18 causing leukemia. It's not causing atypical CML.
19 It's not causing myelodysplasia. It's not causing
20 whatever you want to say.

21 Q Do you believe there is an increased risk
22 of leukemia by gasoline distribution workers?

23 A By gasoline distribution workers?

24 Q For gasoline distribution workers.

25 A No.

1 Q Let me say it in a different way. Do you
2 believe gasoline distribution workers have an
3 increased risk of leukemia?

4 A If you're talking about collective
5 leukemias, no. We've gone through that several
6 times, and we've also discussed what do you mean by
7 an increased risk.

8 Q What about AML? Do gasoline distribution
9 workers have an increased of AML?

10 A That would not be supportable by the
11 scientific literature.

12 Q That's your opinion?

13 A That's the scientific literature's
14 opinion.

15 Q But you're the one making the conclusion
16 that it's not supportable?

17 A That's true.

18 MR. PATTON: Okay. Thank you.

19 THE DEPONENT: Are you done, too?

20 THE VIDEOGRAPHER: We're off the record at
21 3:03. This concludes today's deposition of Dr. David
22 Pyatt.

23 (The deposition concluded at
24 3:03 p.m., October 8, 2009.)

25

1 I, DAVID PYATT, do hereby certify that I
2 have read the foregoing transcript and that the same
3 and accompanying amendment sheets, if any, constitute
4 a true and complete record of my testimony.

5
6
7
8 _____
Signature of Deponent

() No Amendments

9 () Amendments Attached

10 Subscribed and sworn to before me this
11 _____ day of _____, 2009.

12
13 Notary Public: _____

14 Address: _____

15 _____
16 My commission expires _____

17 Seal:
18
19

20 JLP
21
22
23
24
25

1 STATE OF COLORADO)

2) ss. REPORTER'S CERTIFICATE

3 COUNTY OF DENVER)

4 I, Janet Lee Priestley, do hereby certify
5 that I am a Registered Professional Reporter and
6 Notary Public within the State of Colorado; that
7 previous to the commencement of the examination, the
8 deponent was duly sworn to testify to the truth.

9 I further certify that this deposition was
10 taken in shorthand by me at the time and place herein
11 set forth, that it was thereafter reduced to
12 typewritten form, and that the foregoing constitutes
13 a true and correct transcript.

14 I further certify that I am not related to,
15 employed by, nor of counsel for any of the parties or
16 attorneys herein, nor otherwise interested in the
17 result of the within action.

18 In witness whereof, I have affixed my
19 signature and seal this 12th day of October, 2009.

20 My commission expires October 29, 2009.

21
22 _____
Janet Lee Priestley

23 216 - 16th Street, Suite 650

24 Denver, Colorado 80202
25

- 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
6	_____	_____	_____
7	_____	_____	_____
8	_____	_____	_____
9	_____	_____	_____
10	_____	_____	_____
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19	_____	_____	_____

Signature of Deponent: _____

Subscribed and sworn to before me this ____ day of
_____, 2009.

Notary's signature _____

(seal) Notary's address _____

My commission expires _____.