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IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF TEXAS
MARSHALL DIVISION

ROBERT B. OAKLEY and)
IRENE OAKLEY,)
) CIVIL ACTION
Plaintiffs,)
VS.) NO.: 2:07-CV-351
)
AIR PRODUCTS AND)
CHEMICALS, INC.,)
)
Defendants.)

ORAL DEPOSITION OF
DAVID PYATT, PH.D.
November 7, 2008

ORAL DEPOSITION OF DAVID PYATT, PH.D., produced as
a witness at the instance of the PLAINTIFFS, and duly
sworn, was taken in the above-styled and numbered cause
on the 7th of November, 2008, from 8:29 a.m. to 3:24
p.m., before Shauna L. Beach, RDR, CRR, CSR in and for
the State of Texas, reported by machine shorthand, at
the law offices of Forman Perry Watkins Krutz & Tardy,
1717 St. James Place, Suite 600, Houston, Texas 77056,
pursuant to the Federal Rules of Civil Procedure and the
provisions stated on the record or attached hereto.

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P R O E E D I N G S

DAVID PYATT, PH.D.,

having been first duly sworn, testified as follows:

EXAMINATION

BY MR. LUBEL:

Q. Please state your name.

A. David Pyatt.

Q. Which diseases do you recognize and appreciate to be caused by benzene exposures?

A. By high dose chronic benzene exposure?

Q. Whatever benzene exposure.

A. Well, I can't answer that, whatever benzene exposure --

Q. Okay.

A. -- because we've all got benzene exposure and not every disease is caused by that. So you have to specify what kind of exposure you're talking about.

Q. You do? Okay.

So if you were asked what diseases are associated with benzene exposures, you can't answer that, correct?

A. Well, I think you need to -- in a formal setting like this, you need to specify what kind of exposure you're talking about. If you're talking about chronic occupational exposures, I can answer the

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1 question.

2 Q. Well, let's start with chronic occupational
3 exposures.

4 A. Okay.

5 Q. What -- what diseases do you recognize to be
6 caused by chronic occupational benzene exposures?

7 A. Okay. I think that science supports a link
8 with acute myelogenous leukemia. I think science
9 supports a link with some forms of myelodysplasia. I
10 think science supports a link with really high dose
11 exposures to aplastic anemia. I think there is an
12 association in the literature with pancytopenias or
13 other peripheral blood abnormalities.

14 Q. Such as what?

15 A. Lymphocytopenia, thrombocytopenia. And I'm not
16 sure if you would consider those diseases per se. But,
17 I mean, I think that is an established toxic effect of
18 benzene exposure.

19 Q. How about leukopenia?

20 A. Leukopenia, yes.

21 Q. You agree to that one, too?

22 A. Yes. Leukopenia is just a suppression of white
23 blood cells. Sure, that would fall into that category.

24 Q. At what levels of exposure can benzene have an
25 effect on the blood cells?

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1 A. I think the bulk of the literature -- and this
2 would be with animals and with people -- would suggest
3 that somewhere around 10 parts per million would be --
4 that would be a not surprising endpoint for someone who
5 is chronically exposed at 10 parts per million over a
6 time-weighted average, eight hour.

7 Q. And you would agree that there is absolutely no
8 literature that supports any effect on the blood level
9 from benzene exposures at less than 1 part per million,
10 correct?

11 A. I would not agree to that, no.

12 Q. Why wouldn't you?

13 A. Well, because the -- there has been a recent
14 paper published in Science, which has been relatively
15 controversial. That paper does indicate that they
16 reported some subtle changes in those workers at
17 exposures less than 1 part per million.

18 Q. Was that the first report of changes in the
19 blood from benzene exposures below 1 part per million,
20 that you had seen?

21 A. It's the first one that I have seen. And it's
22 been analyzed extensively on all sides. And there is
23 quite a bit of debate as to whether or not that is
24 accurate and whether the workers that were characterized
25 as having less than 1 part per million time-weighted

1 average really belong in that category. But, yes, it's
2 the first one that I've seen.

3 Q. Who wrote the article that you're referring to?

4 A. It was out of NCI. The first author is Lan. I
5 think it was in 2004 or 2003. Martin Smith was one of
6 the authors, Nate Rothman, a bunch of NCI investigators.

7 Q. What's the NCI?

8 A. The NCI? The National Cancer Institute.

9 Q. And so it's your opinion that the -- the work
10 they did was in error?

11 A. No, I didn't say that at all. I said it's my
12 opinion that that particular effect -- and I have the
13 study. We can get -- I'd be happy to get it out. But
14 if you look at the dose response, there is a suppression
15 in that low dose group, but then there is no additional
16 suppression in the next dose group, which goes from 1 to
17 10 parts per million. So that one datapoint is
18 inconsistent with everything else that I've ever seen in
19 the scientific literature. So that's a pretty important
20 point.

21 Q. And what were they studying?

22 A. They were looking at Chinese workers and the
23 health effects from benzene exposure.

24 Q. And how did they isolate people that had
25 exposures below 1 ppm?

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1 A. I don't recall those details. I mean, the way
2 they calculated their -- divided up the workers into
3 this and that, and some workers didn't have
4 time-weighted average that high, but there were some
5 excursions. They did some molecular analysis, some
6 genetic profiling. It's a pretty extensive study. But
7 I don't remember the details on how they divided it up.

8 Q. Have you reduced any criticisms you have of
9 that piece of literature to writing?

10 A. Me, personally?

11 Q. Yes, sir.

12 A. I haven't. There are plenty out there.

13 Q. But you have not done so?

14 A. I have not, no. There are several publications
15 and letters to the editor and discussions back and
16 forth. I mean, it was -- it was a very controversial
17 finding.

18 Q. Can you pull for us the literature that you
19 brought with you today that addresses the effects of
20 benzene exposures where they tried to isolate those
21 exposures below 1 ppm?

22 MR. GRAY: Objection; form.

23 MS. YATES: Objection; form.

24 A. Yeah, I mean, I'm not -- I think most of these
25 studies, when they've gone and looked at occupational

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1 workers, there are going to be people that have
2 exposures less than that. I don't know that they've
3 specifically gone after what you're asking about.

4 But in the refinery binder that I brought,
5 there's tons of refinery studies. I think I might have
6 35 or 40, maybe more. Practically all of those, if
7 they've quantitative exposure, there are going to be
8 groups of workers that are below that exposure level.

9 Q. (By Mr. Lubel) If you don't mind, just pull
10 out the articles where you think the focus of the study
11 was exposures at or below 1 ppm.

12 MR. GRAY: Objection; form.

13 MS. YATES: Objection; form.

14 A. Focus of the exposure?

15 Q. (By Mr. Lubel) Focus of the study. Take your
16 time.

17 A. Okay.

18 Q. I'm going to get you out of here for your
19 airplane.

20 A. Well, I appreciate that, but we can go as long
21 as you want. (Examination of documents.)

22 I don't -- I mean, I don't -- I cannot
23 think of a study where they have said, That's what we're
24 going to do. I mean, the studies where they've --
25 they've said, Okay, here's some workers, maybe they're

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1 exposed to benzene, what are the health effects
2 associated with those exposures? Then part of that
3 investigation is going to be a quantitative analysis of
4 the exposures. But I can't think of someone saying,
5 Okay, what are the -- what are the health effects of
6 people exposed less than 1 part per million, at least in
7 the purpose of the study.

8 Q. So that hypothesis has not been tested, to your
9 knowledge?

10 A. What hypothesis? That less than 1 part per
11 million -- oh, I disagree with that. I don't think you
12 have to say, I'm going to go look at workers
13 specifically to see what happens at less than 1 part per
14 million to be able to answer that question. If you're
15 going and looking at workers and there are workers
16 exposed to less than 1 part per million and you look at
17 the health effects of those workers, you're answering
18 the question --

19 Q. Oh, you are?

20 A. -- even though you haven't specifically said
21 that's what I'm doing.

22 Q. Well, you don't have to set the hypothesis
23 before you do the study?

24 A. Well, I think setting a hypothesis is an
25 important part of the scientific method. But no, I

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1 don't think if you don't say -- I mean, in the
2 hypothesis they don't say, And I'm going to compare
3 these to nonexposed controls and yet that happens in
4 every single study.

5 Q. Did any of the studies in the benzene
6 literature that you brought with you set forth the
7 hypothesis of exposures below 1 ppm don't lead to
8 disease or illness?

9 MR. GRAY: Objection; form.

10 MS. YATES: Same objection.

11 A. Well, I think several of them have said, We
12 know that our workers are exposed to low levels of
13 benzene. What's happening to them? You know, maybe
14 they didn't say 1 part per million, but I can think of
15 several refinery studies, one in particular -- which
16 we'll find as soon as we quit talking -- it was clearly
17 less than 1 part per million and there were no
18 hematopoietic effects, and that's what they were going
19 after.

20 Q. (By Mr. Lubel) Take your time, find all of the
21 studies in the -- you brought numerous books with you
22 today, correct?

23 A. Binders, yes.

24 Q. Yeah. Find all of the studies that you think
25 address the effects of benzene exposures at or below 1

1 ppm.

2 MS. YATES: Objection; form.

3 MR. GRAY: Same objection.

4 MR. LUBEL: What's your objection, Stacy?
5 Specifically, what's the grounds?

6 MS. YATES: It's -- you're asking for all
7 and I -- and I don't think he can speculate as to all of
8 that. You have 10 binders over there. Given the amount
9 of literature he has there, it's impossible given our
10 time constraints.

11 MR. LUBEL: Do you think he would have to
12 speculate to answer that question?

13 MS. YATES: I believe he would have to
14 speculate.

15 MR. GRAY: I think it's also vague as to
16 studies which address benzene exposures.

17 MR. LUBEL: He's just not qualified to
18 figure that out?

19 MR. GRAY: Well, I'm not sure who he's
20 addressing. As he reads it or is he supposed to read
21 the mind of the authors? He's smarter than me, so...
22 (Pause.)

23 MR. LUBEL: I'll stipulate to that. And I
24 just met him.

25 And we're off the record.

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1 (Discussion off the record.)

2 Q. (By Mr. Lubel) Do you have them?

3 A. I have some of them. Here's one that Jim
4 Collins published in 1991 and --

5 Q. What's the title?

6 A. A Study of the Hemapoietic Effects of Chronic
7 Low-Level Exposures to Benzene. And there are workers
8 in there exposed to less than 1 part per million and
9 there are no hematopoietic effects.

10 Q. Can we go through that article real quick?

11 A. Yeah, they --

12 Q. Just so we're clear: What do you mean, there
13 is no hematopoietic effects?

14 A. "Exposure" -- this is from the abstract --
15 "Exposures to low-level to benzene does not appear to
16 produce an increased level of abnormal hematology
17 measures detectable in the routine medical
18 surveillance."

19 Q. And what -- what were -- what were the
20 exposures here?

21 A. Umm, they're 0.1 ppm to a high of 1.4 ppm,
22 eight-hour-time-weighted average over a ten-year period.

23 Q. What type of workers were they?

24 A. Study of 200 workers, persons working with
25 benzene showed no difference in commonly measured

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1 hemapoietic outcomes when compared to 268 nonbenzene
2 workers in the same plant. These look like -- this is
3 from his Dow series, so this is from Monsanto.

4 Q. It's from Monsanto. Is it a refinery or
5 chemical plant?

6 A. I think this was a chemical plant.

7 Q. Where was it located?

8 A. I don't -- I don't know. The chemical -- from
9 the Monsanto Company, 800 North Lindbergh Boulevard,
10 St. Louis, Missouri.

11 Q. And how does it say they were exposed?

12 A. What do you mean by in their work, what they
13 were doing?

14 Q. Yes, sir.

15 A. I don't -- exposure estimates. "Exposure to
16 benzene was based on judgments of industrial hygienists
17 familiar with the plant using data on actual exposures.
18 Methods were described elsewhere."

19 Q. How were their measurements taken?

20 A. How were their measurements taken?

21 Q. Was there air sampling or were there -- were
22 these judgments modeling?

23 A. No, they were data. "Exposure estimates shown
24 in the final volume of Table 2 were constructed using
25 actual exposure monitoring data where monitoring results

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1 were available and industrial hygienists' judgments in
2 areas of very low exposure potential and little
3 monitoring data."

4 Q. What does that mean, judgments where there is
5 little monitoring data?

6 A. Umm, I guess in office environments and places
7 where they're not expecting anyone to be exposed, they
8 don't have monitoring stations set up. But out in the
9 facility, where there's potential for occupational
10 exposures, they have -- they do have monitoring data.

11 Q. So it was just in the offices that they didn't
12 have monitoring data?

13 A. It doesn't say that. That's -- that was my
14 paraphrasing when you asked what that means.

15 Q. Do they explain in that article what that
16 means?

17 A. They might.

18 MR. GRAY: Objection; form.

19 A. Well, okay. "Were constructed using actual
20 exposure monitoring data when monitoring results were
21 available." You know what that means, right?

22 "And industrial hygienists' judgments in
23 areas of very low exposure potential" -- you know what
24 that means. Okay?

25 "And little monitoring data."

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1 Q. (By Mr. Lubel) Do they break down as they look
2 at each individual worker's exposures where they fit
3 whether their actual monitoring data, whether they are a
4 judgment by the industrial hygienist if there is little
5 --

6 A. Oh, I don't know. I mean, I would suspect
7 that's described somewhere in the fine print in this
8 study.

9 Q. Do you recall what it says about that?

10 A. I don't.

11 Q. See if you can find that for us, Dr. Pyatt.

12 A. What am I looking for? Whether or not for
13 individual workers?

14 Q. Yeah, right. Whether -- sure. Whether for
15 individual workers they are addressing whether they have
16 actual monitoring data as opposed to making some
17 industrial hygiene judgment. You're familiar with
18 industrial hygienists making judgments, right?

19 A. I know what those words mean.

20 Q. Do you not know that industrial hygienists make
21 judgments sometimes when they do not have actual
22 monitoring data?

23 A. That's -- I mean, that -- that's what I mean.
24 I know what those words mean.

25 Q. Are you not familiar with them doing that?

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1 A. In my practice?

2 Q. Yes, sir.

3 A. What do you mean by "familiar"? I understand
4 what those words mean. I understand that that is
5 something that they do.

6 Q. You'd agree that it's not generally accepted in
7 the scientific community for industrial hygienists to
8 make judgments without actual monitoring data, correct?
9 They can't do that.

10 MR. GRAY: Objection; form.

11 A. Well, I'm not sure that's true. When you look
12 at job exposure matrices that are -- they usually
13 conduct these in epidemiological studies. They
14 frequently use panels of industrial hygienists to come
15 up with what they think those exposures might have
16 likely been.

17 Q. (By Mr. Lubel) Well, that's not appropriate,
18 though, is it?

19 A. I wouldn't say it's not appropriate. I mean,
20 that is pretty standard in some epidemiological studies.
21 But I'm saying that it's likely to have some uncertainty
22 about it and definitely a better procedure would be if
23 you actually have monitoring data.

24 Q. And so when we look through these
25 epidemiological studies that have been done on benzene,

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1 where they're relying on judgment as opposed to actual
2 monitoring data, we can throw those out, correct?

3 A. I wouldn't say that. I wouldn't say you could
4 throw them out. I mean, if a guy has been an industrial
5 hygienist for 20 years and he's worked in 15 refineries
6 where there is monitoring data and someone is asking him
7 about a question at the 16th refinery, which is set up
8 exactly the same way as the other 15, I think his
9 judgment as to what those exposures could be would be
10 relevant without actually having monitoring data.

11 So, no, I don't think you can throw them
12 out, but I do agree that having real data is better than
13 not having data.

14 Q. In any epidemiological studies that we look at
15 where they're taking surveys or questionnaires of people
16 that lead to exposure information, we can throw that
17 out, too, because that's not generally accepted, right?

18 A. I disagree with that as well. I don't think
19 it's not generally accepted. That's a pretty standard
20 process in epidemiology studies.

21 Q. To ask the people what their exposures were?

22 A. Absolutely.

23 Q. When was that recognized?

24 A. Good grief, Mr. Lubel, that's recognized --
25 you -- you'd have to -- sorry. You would have to ask an

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1 epidemiologist when it was recognized. But that is a
2 standard process for probably half of the
3 epidemiological studies that I've looked at. Where they
4 go out in the community and say, What were you exposed
5 to? Did you ever use this? Did you ever use that? How
6 often? Three times a week? Four times a week? There
7 is no monitoring data.

8 Now, yes, I think that weakens the
9 conclusions that you can draw from that study, but that
10 is a very common practice.

11 Q. What's better? The surveys or questionnaires
12 of the people or the industrial hygiene judgments when
13 you have no actual monitoring data?

14 A. That's a good question. I think that would
15 depend on the industrial hygienist and what their
16 experience was and -- but I think that probably is a
17 step more quantitative than just saying, Were you ever
18 exposed to benzene out in the community, where you've
19 got recall bias and other issues with that group of
20 people that you're studying.

21 But, clearly, if you've got area monitoring
22 or you've got personal monitoring, that's -- that's a
23 step in the right direction. If you have biological
24 monitoring, that's even better.

25 Here's another one --

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1 Q. Let me go back to this first one.

2 A. Okay.

3 Q. This is by James Collins.

4 A. Uh-huh. (Witness answers affirmatively.)

5 Q. Do you know him?

6 A. Yes.

7 Q. Do you know that he's the director of
8 epidemiology for Dow Chemical Company?

9 A. Yes.

10 Q. Have you talked to him lately?

11 A. What do you mean by "lately"? I mean, I've
12 talked to him in the last couple of years.

13 Q. You know him?

14 A. Yes, I know him.

15 Q. And when they authored this article, he was
16 working for Monsanto, correct?

17 A. When they published that author -- published
18 that paper? Yes, it appears that way.

19 Q. And do you know that about his work history,
20 that he was at Monsanto before going to Dow?

21 A. Yes -- oh, no, I didn't know that he was at
22 Monsanto prior to Dow, but whatever. I -- that wouldn't
23 surprise me.

24 Q. Is he a good epidemiologist?

25 A. He's an excellent epidemiologist.

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1 Q. Have you seen his opinions about the Chinese
2 study that Dr. Irons is doing for benzene?

3 A. I have not.

4 MR. GRAY: Objection; form.

5 MS. YATES: Objection.

6 Q. (By Mr. Lubel) Would it surprise you if
7 Dr. Collins has testified under oath that Dow Chemical
8 would not participate in the funding of that study
9 because he believed it was biased?

10 A. I don't know anything about that.

11 Q. Do you know anything about those Chinese
12 studies?

13 A. A little.

14 MR. GRAY: Objection; form.

15 MS. YATES: Objection; form.

16 Q. (By Mr. Lubel) Are you working on them?

17 A. No. I was involved really early on in some of
18 the planning stages when I was working with Rich Irons,
19 but then I left. It wasn't even -- it hadn't even been
20 funded at that point.

21 Q. Okay. Let's go to your next article you talked
22 about.

23 A. So this one, A Hematology Surveillance Study of
24 Petroleum Workers Exposed to Benzene.

25 Q. Who's the lead author?

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1 A. Tsai.

2 Q. Can you spell that?

3 A. T-s-a-i.

4 And the purpose of this study -- this is
5 directly from the abstract -- "The purpose of this study
6 is to evaluate hematology data from employees who have
7 ever participated in the Shell Benzene Medical
8 Surveillance Program compared to employees who have not
9 participated and to examine the sensitivity of CBCs" --
10 which is complete blood counts; I added that -- to
11 detect hematological changes in a low-exposure
12 occupational settings. This large study includes 1,200
13 employees who participated in the Shell Benzene Medical
14 Surveillance Program, with mean benzene exposure
15 time-weighted average eight hour of 0.60 part per
16 million from 1977 to 1988 and 0.14 ppm since '88."

17 And then the punch line, "At current
18 occupational exposure levels for benzene, no evidence of
19 adverse hematological effects was observed in the study.
20 These results raise the question of whether annual CBC
21 surveillance for benzene-exposed workers has adequate
22 sensitivity to detect meaningful hematological changes
23 due to low-level exposures."

24 Q. Does this study address their cumulative
25 exposures over their work lifetime, their ppm years?

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1 A. I don't think that was the purpose, but he may
2 have done that as he went through it. With regard to
3 hematology, that -- that is a relatively short-term
4 phenomenon. So what people are exposed to on a daily
5 basis is more relevant than what their cumulative
6 exposures were. So most of these studies that we are
7 talking about now, with hematology, have not gone and
8 taken that step to figure out what their cumulative
9 exposures were.

10 Since it's a -- it's a process that people
11 used to use -- occupational physicians used to use CBCs
12 and white blood count to monitor workers for excessive
13 benzene exposure. It's something that they said, Okay,
14 last week you were too highly exposed. It's not going
15 to be that helpful if they have to wait until the end of
16 their career to see what the basis of that cumulative --

17 Q. What you're saying is that these particular
18 studies that you pulled so far are dealing with more the
19 acute effects than the chronic effects?

20 A. Well, it's not acute in that you're not going
21 to see, like, a central nervous system depression or a
22 headache or something from a high exposure for 15
23 minutes. It's acute in that it -- it will show up
24 within weeks to months after that's occurring. So it
25 probably would more accurately be called subacute.

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1 Q. These -- these studies that we're talking about
2 right now don't address the chronic, long-term effects
3 is what you're saying? They're not supposed to?

4 MR. GRAY: Objection; form.

5 MS. YATES: Same objection.

6 A. Well, they're looking at a snapshot in time as
7 to what has happened to these workers from those
8 exposures in the workplace. It would take a follow-up
9 study to see if there were any long-term sequelae from
10 any hematological change that they observed.

11 Q. (By Mr. Lubel) Let's do it this way. The
12 first study, the James Collin study out of Monsanto, can
13 you find for me where in here they address the chronic
14 long-term exposures, the cumulative or aggregate
15 exposures over a work life.

16 A. I don't think they did that in here.

17 Q. Okay.

18 A. And I don't think they did that in the Tsai.

19 Q. Great. So what you're doing is you're pulling
20 articles to address my question of what effects to
21 low-level benzene exposures at or less than 1 ppm on the
22 blood.

23 A. Well, no. Actually, I'm pulling these in
24 response to your question as to can I point to any
25 studies where someone specifically went out to address

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1 the question of whether people exposed to less than 1
2 part per million are going to have hematological
3 effects.

4 Q. Okay. Let's keep going. Thank you.

5 A. Here's one, and this actually forms the basis
6 for EPA's noncancer toxicity value. This is Nate
7 Rothman's paper from 1996. Hematoxicity Among Chinese
8 Workers Heavily Exposed to Benzene. And they had --
9 they broke the workers up into two groups with 31 part
10 per million as kind of the exposure. In the less than
11 31 part per million group there were some workers that
12 had excursions above 31 parts per million, so there was
13 some question as to the validity of that breakdown.

14 Then they went to -- and I know the details
15 about this because it forms the basis of the EPA's
16 noncancer reference concentration. And the less than
17 31, they wanted to pull out all of the workers that
18 never went above 31 ppm at any time in their workplace.
19 And those workers had an average cumulative exposure or
20 time-weighted average of 7 -- approximately 7 ppm. And
21 they did see a change in lymphocytes. They saw
22 lymphocytopenia in those workers, but that was it.

23 Q. What is lymphocytopenia?

24 A. That is suppression of lymphocytes. Any
25 "penia" -- a cytopenia is a suppression. And then you

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1 can tell what cell is being suppressed that comes before
2 that.

3 Q. It's a white blood cell suppression?

4 A. Lymphocytopenia -- I mean, a lymphocyte is a
5 white blood cell. But there are other white blood
6 cells. So what you asked earlier in the deposition
7 about a leukopenia, that includes all white blood cells.

8 Q. In this Rothman article, the 1996 one, is
9 there -- can you point to me where it concludes that
10 exposures at or below 1 ppm are safe?

11 A. They never made that determination. I don't
12 think there are any workers that -- that would fit into
13 that category. These were pretty highly exposed
14 workers.

15 I want to make sure I'm not duplicating.

16 So here is another one from Jim Collins and
17 coworkers. Evaluation of Lymphopenia Among Workers with
18 Low-Level Benzene Exposure and the Utility of Routine
19 Data Collection. The abstract reads, "Whether low-level
20 benzene exposure produces health effects is
21 controversial. We used routinely collected data from
22 our medical/industrial hygiene system to study 387
23 workers with daily 8-hour time-weighted exposures
24 averaging 0.55 ppm".

25 Q. Was this at Dow or Monsanto?

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1 A. From the Monsanto Company. Address
2 correspondence James Collins, Ph.D., Monsanto.
3 "Examination of other measures of hematotoxicity" -- oh,
4 here it is. "We found no increase in the prevalence of
5 lymphopenia among benzene-exposed workers." There's
6 some confidence intervals there taking into account
7 smoking, age and sex. "There also was no increase in
8 risk among workers exposed 5 or more years."
9 Statistical analysis there. "Examination of other
10 measures of hematotoxicity, including mean corpuscular
11 volume and counts of total white blood cells" -- et
12 cetera, et cetera -- "produced similar results. We
13 conclude that risk of lymphopenia and other early
14 indicators of hematotoxicity are not increased among
15 workers in this study who were exposed to low levels of
16 benzene."

17 Q. Can I see that one while you're looking?

18 A. Sure.

19 I mean, the animal studies, I can't think
20 of a one where they would be concerned about what's
21 going to happen at less than 1 part per million. I
22 don't think they ever did that. There was no -- as I'm
23 pointing out, there is just no consistent evidence in
24 the literature that those levels of benzene would cause
25 cytopenia, so they wouldn't waste animals' lives to test

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1 it. Most of those studies, they were looking at pretty
2 high levels.

3 And in the older occupational literature,
4 they have really high -- really high exposures.

5 Q. What levels are they generally testing the
6 animals at?

7 A. Well, a lot of them aren't inhalation studies.
8 A lot of them have -- they're subcutaneous injections or
9 oral gavage and you can make that conversion, but they
10 frequently don't.

11 The inhalation studies -- or the
12 carcinogenic studies, they had to use a high enough dose
13 where they could actually see something. And so that
14 would be typically 300, maybe even 1,000 part per
15 million, over the lifetime of the animal. That's how
16 much they had to expose the animal to to be able to see
17 an increase in the tumor formation.

18 Q. Why were they using such a high dose on the
19 animal?

20 A. If they used something else, they didn't see
21 any tumors.

22 Q. What good is that?

23 A. That's the reason why they had to use a high
24 dose. It wouldn't be any good. That's really kind of
25 the problem with interpreting animal studies. You

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1 can't -- you can't afford, and really it's ethically
2 questionable, to have an animal study with 10,000
3 animals per treatment group. And that's how big it
4 would have to be in order for you to see really subtle
5 increases in tumors. In one in a million, okay, you'd
6 have to have a million animals to be able to potentially
7 see that kind of increase. So you'd need to see 30
8 percent increase or 40 percent increase in the number of
9 tumors. You're not going to do that with 1 part per
10 million or 10 or 200. So they had to use really high
11 levels.

12 Q. Do they also use higher levels on animals
13 because their life is shorter?

14 A. I don't think there is any rationale for that.
15 I mean, their life is shorter. They're different
16 hemopoietically. So there's all kinds of problems
17 interpreting those studies from a pharmacodynamic point
18 of view and how they respond. They have -- they have
19 extramedullary hematopoiesis. That are many differences
20 between rats and people. But I don't think their
21 lifespan is the reason why they had to use higher
22 exposures.

23 I mean, if a human is exposed for 30
24 percent of their life and a rat is exposed for 30
25 percent of their life, it's going to be different times,

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1 but there is no reason to think that they'll respond any
2 differently.

3 Q. And the truth is it's not -- it's generally
4 accepted in your field that we should not consider the
5 animal studies with respect to humans, correct?

6 A. I disagree with that.

7 Q. It's not relevant, is it?

8 A. I disagree with that.

9 Q. Well, tell me why it is that animals have any
10 part -- animal studies have any part in looking at
11 humans.

12 A. I think with the exception of maybe asbestos
13 and maybe benzene -- well, benzene for sure, and I think
14 arsenic -- maybe there's one or two other ones, but
15 those three for sure -- every other regulatory position
16 that the USEPA has set forth for a chemical is based on
17 animal data. So 99 percent of the chemicals for which
18 EPA has said, Here's what we consider to be the cancer
19 slope factor, is based on animal data.

20 Now, you could argue the relevance, and
21 people do all of the time, but it represents a very
22 important part of our public health decision-making,
23 animal data. There's no question about that. We don't
24 have enough reliable -- what you asked me earlier about
25 the epidemiological studies, they are problematic in

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1 terms of having quantitative exposure data. It's hard
2 to do.

3 So with an animal study, you have
4 definitive quantitative exposure data. You know exactly
5 what those animals were exposed to. And you know
6 exactly what those animals did on the weekend. You know
7 exactly their life. You know what they're doing. So
8 the data tends to be cleaner than what you get in a --
9 in a human population, less variability.

10 So EPA and regulatory agencies do most of
11 their mathematical modeling on animal data.

12 Q. What's more important, the epi studies or the
13 tox studies?

14 MR. GRAY: Objection; form.

15 MS. YATES: Same objection.

16 A. That's going to depend on what the question is.

17 Q. (By Mr. Lubel) With respect to benzene, are
18 both important?

19 A. Because of benzene, because there is relatively
20 good quantitative human data, we don't really have that
21 problem, because both the noncancer and the cancer
22 endpoints from EPA's point of view as well as OSHA's PEL
23 and the ACGIH and every other regulatory decision,
24 that's all based on human data.

25 Q. So with respect to benzene, the animal studies

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1 are not relevant?

2 A. I would not characterize it like that. There
3 are definitely some things that we have learned from
4 animal studies that have been borne out in humans. The
5 pharmacodynamics or the toxicodynamics seem to be
6 different. So there would be some issues in terms of
7 relevance there. The toxicokinetics seems to be right
8 on the money.

9 Q. Are the animal studies relevant to the issue of
10 how much benzene exposures cause disease?

11 A. You'd have to define disease. I think they are
12 very relevant when it comes to how much it takes to
13 damage the marrow and how much it takes to produce
14 cytopenias. Because it has been borne out that they
15 start seeing suppressions in lymphocytes at about the
16 same level that humans do. They respond approximately
17 the same way that humans do. Their marrow looks very
18 similar to the way that human marrow looks if it's been
19 highly exposed. All of those things seem to be true
20 when it comes to cancer, then it goes away. And then,
21 yes, I would say that the animals do not represent -- at
22 least today, they don't represent an appropriate
23 modeling for looking at the way humans respond to high
24 dose chronic benzene exposure.

25 Q. And why is that?

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1 A. Well, I don't know the answer to why. They
2 don't get AML for one thing. They get this sort of
3 chronic myeloproliferative disorder that is somewhat
4 different. They get an aggressive thymic lymphoma that
5 there is no precise analogy in humans.

6 They get zybal gland tumors that people
7 don't even have. We don't have a zybal gland. I mean,
8 they are different. They just do not respond the same
9 way.

10 Now, there is a couple of genetic inbred
11 strains of mice that people are working out that are
12 trying to develop that -- that are trying to develop a
13 good experimental model for chemical-induced leukemia.
14 It's a very important question that we'd all like to
15 have it. There are going to be a lot of things that we
16 can do molecularly to try and understand some of the
17 pathology. And there is at least one -- this TAG mouse
18 does seem to get as close as I've seen to a human AML
19 following benzene exposure.

20 Q. Well, if in fact the animal studies where
21 there's benzene exposures don't reveal leukemias, how do
22 we know that benzene exposures can cause leukemias in
23 humans?

24 A. Well, actually they get a --

25 THE WITNESS: Did you want to object?

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1 MR. GRAY: I did want to object to form.

2 A. They get a lymphoma, kind of an aggressive
3 T-cell lymphoma that sometimes spills over into the
4 blood. So you might could call that leukemia. It's
5 definitely not AML. It's different than acute
6 myelogenous leukemia.

7 So the first part of your question is not
8 exactly accurate. They do get some form of disease that
9 are kind of comparable. We know because there's
10 quantitative epidemiological evidence that high dose
11 exposure to benzene can cause AML in people. So we
12 don't need the animal data to support, confirm, argue
13 with -- I mean, that is why EPA and everyone uses human
14 data to -- to base their regulatory position on.

15 Q. (By Mr. Lubel) What human study, in your
16 opinion, was definitive on the connection between
17 leukemia and benzene exposure?

18 A. Well, I think the best one, and the one that
19 seems to be the best studied and has the fewest
20 confounders, is Pliofilm, the retrospective mortality
21 that Peter Infante first talked about in '78 and Bob
22 Rinsky reported on it in '81, and then kind of the
23 definitive risk assessment paper came out in 1987. I
24 think that, at least today, represents the best that we
25 have.

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1 Q. The most definitive body of work?

2 A. Yes.

3 Q. What would be the second best?

4 MR. GRAY: Objection; form.

5 MS. YATES: Objection; form.

6 A. Sorry. I was trying to think why they were --
7 okay.

8 The second best? I don't know. I haven't
9 ranked them.

10 MR. LUBEL: He denied your objection, by
11 the way.

12 THE WITNESS: I was confused by that.

13 MR. GRAY: I was confused by your question.

14 MR. LUBEL: He's saying there is no form
15 objection. It's not ambiguous. It's not compound.

16 MR. GRAY: It's definitely not compound.

17 A. Well, I mean, I think that -- the second best,
18 the second best. I mean, that one is the one that we
19 base all of our regulatory decisions on, because there
20 is fewer problems with that. I think we can learn
21 something from the older studies, Aksoy's work. I think
22 that has some relevance. The Vigliani studies, the
23 Italian shoe worker studies, I think that has some
24 relevance. The nested case control studies from Rushton
25 and Schnatter and Glass, I think that all has some

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1 bearing on this question. The NCI work that they did
2 all through the '80s, that has some bearing on this
3 question. I think their reanalysis and what they're
4 doing in China now, when it's published, will probably
5 matter, as will the Shanghai health study, the one that
6 Rich Irons is leading up that you brought up, I think
7 that will probably bear on it. But right now our formal
8 evaluation of this question is based on Pliofilm.

9 Q. (By Mr. Lubel) You can't rank them after that,
10 can you?

11 A. It's really hard to do. They're all going to
12 have pluses and minuses and problems. And even the
13 Pliofilm is not perfect. It's got a lot of problems
14 itself.

15 Q. And the truth is, it's really not appropriate
16 to just look at one epidemiological study. You really
17 need to consider the broad-based information we have to
18 form opinions, correct?

19 A. Well, I think to form any opinion you want to
20 look at all of the data that you can find.

21 Q. And if we look at the Hill Bradford -- the
22 Bradford-Hill Criteria or guidelines, so to speak --

23 A. Uh-huh. (Witness answers affirmatively.)

24 Q. -- those guidelines would recommend you look at
25 more than one epidemiological study in order to form an

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1 opinion, correct?

2 A. Absolutely.

3 Q. Causation, generally, doesn't turn on just one
4 study, does it?

5 A. It shouldn't, no.

6 Q. You're not taught that way.

7 A. No.

8 Q. I don't know if I missed it, but did you -- as
9 you were talking about the studies that had relevance,
10 did you mention the work that was -- that was done by
11 -- done in China in the '80s and '90s by Hayes and Yin,
12 that one?

13 A. Well, the Rothman study was part of that
14 process and I have all of those papers here, but I did
15 not see where they really had much bearing on less than
16 1 part per million.

17 Q. No, I'm talking about -- we kind of digressed
18 for a minute and talked about in general what studies --

19 A. Ranking them?

20 Q. Right.

21 A. Yeah, I said the NCI China.

22 Q. That's what you meant?

23 A. Right.

24 Q. With Hayes and Yin?

25 A. Yes. It started in '87 with Yin and -- this

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1 was the Chinese Academy of Preventive Medicine and they
2 started collaborating with NCI. And then they published
3 a whole series of papers through the -- through the '90s
4 on that workforce.

5 Q. Have you done any work for Dr. Collins?

6 A. Work for him? No.

7 Q. Any consulting work?

8 A. No.

9 Q. Have you done any work with him?

10 A. I've sat on a panel or two with him.

11 Q. What type?

12 A. The panel that I sat on with Jim was -- it
13 was -- it was with formaldehyde and some of the
14 regulatory scientific issues associated with
15 formaldehyde exposure and work.

16 Q. It wasn't related to benzene, was it?

17 A. No.

18 Q. Have you done any work with --

19 A. Well, actually, I take that back. We did -- my
20 company -- and I was involved -- we wrote a risk
21 assessment for children with benzene exposure. And Jim
22 came to at least one of our premeetings before we
23 presented this to EPA. So -- and then I think he was on
24 the phone two or three times when we had discussions.
25 So I mean, I've had interactions with -- with Dr.

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1 Collins.

2 Q. Not significant?

3 A. Not -- not like this.

4 Q. Like we're doing now?

5 A. Right.

6 Q. How about Dr. Rothman?

7 A. Nate Rothman? I met him at the benzene meeting
8 in 2004. And then, again, I was on one or two
9 conference calls where he was on it and I was on at the
10 same time. I know him. But, no.

11 Q. Good doctor?

12 A. I don't -- I mean, he's -- he's definitely
13 published some important work.

14 Q. Got a good reputation in the field?

15 A. Yeah, I'd say so, yes.

16 Q. How about Dr. Richard Hayes? Have you done any
17 work with him?

18 A. No. Same. I know him about the same degree
19 that I know Nate Rothman.

20 Q. They both enjoy pretty good reputations in your
21 field?

22 A. They're actually not in the toxicology field.
23 They're more epidemiologists. But, yeah, I mean, you
24 get into any scientific discussion about chemicals in
25 people, you're going to be discussing NCI data.

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1 Q. National Cancer Institute?

2 A. National Cancer Institute, which they are both
3 primary investigators for.

4 Q. Is this a good time for a break for you?

5 A. Uh-huh. (Witness answers affirmatively.)

6 Q. We're going to try to do it every hour and if
7 you need to do it sooner than that, let me know.

8 A. That would be great. Thank you.

9 (Short recess from 9:26 to 9:33 a.m.)

10 EXAMINATION (Continuing)

11 BY MR. LUBEL:

12 Q. Have you had a chance to pull the remaining
13 articles?

14 A. Well, I mean, I'm still looking. There is
15 going to probably be plenty that address the question.
16 Here's one by Quitt, Q-u-i-t-t, Autonomous Growth of
17 Committed Hematopoietic Progenitors From Peripheral
18 Blood of Workers Exposed to Low Levels of Benzene. And
19 they looked at CFU assays and some other hemopoietic
20 assays. "We investigated whether exposure to low levels
21 of benzene (geometric mean of exposures = 0.28-0.41
22 parts per million) results in perturbations of the
23 hemopoietic system found in patients with
24 myeloproliferative diseases. Erythroid, granulocyte, et
25 cetera, et cetera. "Colony growth from 17 workers

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1 exposed to low levels of benzene was compared with 20
2 healthy control subjects. Exposed workers had
3 significantly increased growth of autonomous BFU-E and
4 unstimulated CFU-GM when compared with the control
5 subjects."

6 Q. Can I see it?

7 A. Yes. And then here's Lan's paper, which we
8 started this discussion with. Tsai's paper.

9 Q. Is this a different one?

10 A. No, that's it.

11 Q. This is the one we started with?

12 A. No. I'm sorry. That's the one we started this
13 discussion with, the Tsai paper that reports that they
14 found changes below 1 part per million.

15 Q. Gotcha. Okay. You're pulling articles that
16 address it either way, both sides, right?

17 A. Yeah.

18 Q. These Collins articles that you've -- that
19 you've referenced, Dr. Collins --

20 A. Uh-huh. (Witness answers affirmatively.)

21 Q. -- out of Monsanto, do you -- do you recall
22 whether the route of exposure was skin or inhalation?

23 A. I don't recall specifically, but I'm 99 percent
24 sure it was inhalation.

25 Q. And so they -- they would not have any

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1 relevance to the skin exposures, correct?

2 MR. GRAY: Objection; form.

3 MS. YATES: Objection; form.

4 A. No, I would disagree with that.

5 Q. (By Mr. Lubel) Why?

6 A. I haven't ever seen any evidence to suggest
7 that the route of exposure has a pharmacodynamic
8 difference. By that I mean how the person would
9 respond.

10 Q. What do you -- go ahead.

11 A. With the exception of this genetic strain of
12 mouse, there does appear to be some differences there.
13 But in people, it doesn't -- well, no one has ever said
14 that it matters. And from a -- from a pharmacokinetic
15 point of view, I don't see why it would. Once the
16 benzene gets in the systemic circulation, whether it's
17 getting through first path, whether it's GI absorption,
18 whether it's getting there from lung absorption, whether
19 it's getting through the dermal absorption, once it gets
20 into the bloodstream, it's going to behave the same way.

21 Q. What did you mean by a pharmacokinetic
22 difference?

23 A. Pharmacodynamic difference.

24 Q. Dynamic difference.

25 A. The way modelers in pharmacokinetic break up

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1 the world is there is pharmacokinetic and
2 pharmacodynamic. Pharmacokinetic is how it's absorbed,
3 how it's distributed, how it's metabolized, how it's
4 excreted, all of the enzymes, all the kinetics, all of
5 the processes that go into how benzene is handled
6 kinetically by the body.

7 Pharmacodynamics is the physiological
8 response to the pharmacokinetics, how the animal
9 responds to that exposure.

10 So when we were talking earlier about
11 experimental animals, experimental animals do seem to be
12 very similar pharmacokinetically. They use the same
13 enzymes. The enzymes have the same KM values. There
14 just doesn't appear to be much difference in the way a
15 rat liver handles benzene and the way a human liver
16 handles benzene. But there are clearly differences in
17 the response of the whole animal to that
18 pharmacokinetics.

19 Q. So you're saying that the rats and the people
20 are alike with respect to whether it enters
21 the bloodstream via the air, ingestion, or dermal?

22 A. No. I mean, I think you're going to have some
23 pharmacokinetic differences there. Rats, for example,
24 are -- well, with benzene I'm not so sure. But rats are
25 obligate nose breathers, so there is going to be some

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1 differences in that in potentially how much benzene gets
2 absorbed. But I've never seen there being -- I've never
3 seen anyone report that they are higher or lower in
4 terms of their exposures. But they're different animals
5 so you're going to have some physiological differences.

6 The real -- the real issue is once it gets
7 absorbed, what's it going to do? And there is really no
8 reason to think that a molecule of benzene that gets
9 into the bloodstream from the GI tract or from the lungs
10 or from the skin is going to behave any differently than
11 any other molecule.

12 Q. Do they all go to the same place? Is that your
13 point?

14 A. Yes.

15 Q. Where do they go?

16 A. They go to the liver.

17 Q. And then where do they go?

18 A. Well, I don't think there's many molecules of
19 benzene that probably get out of the liver. Once it
20 goes to the liver, it's metabolized. And then you're
21 going to have the reactive metabolites of benzene. Some
22 are going to make it out. Assuming that you've got high
23 enough exposures, those are going to go -- they go
24 everywhere because they're in systemic circulation. But
25 where we're concerned where they're going is to the bone

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1 marrow where they undergo subsequent oxidation within
2 the marrow.

3 Q. How do they go from the liver to the bone
4 marrow?

5 A. Well, the liver is a highly perfused organ, so
6 there's all kinds of blood going through the liver. And
7 any time the liver metabolizes anything, it's either
8 excreted through the bile, depending on the chemical
9 characteristics of it, or it goes into the systemic
10 circulation.

11 Q. But benzene doesn't pass through the liver; the
12 metabolites do, correct?

13 A. That's what I said. Benzene you probably
14 wouldn't get, I mean, unless you just totally swamped
15 the metabolic capacity of the liver. And in that case
16 you would start seeing benzene coming back out in
17 exhaled breath. And that does happen some. So, you
18 know, there is some benzene that goes into the liver and
19 it doesn't get metabolized and it comes right back out
20 as parent compound. But the -- but the majority of it
21 is going to be metabolized.

22 Q. And those metabolites, then, head to the bone
23 marrow for benzene?

24 A. Well, they head all over the place. But in the
25 bone marrow is where we know that there's toxic effects.

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1 We don't see toxicity in the liver. You don't see
2 toxicity in the kidney. You don't see toxicity in the
3 lung, the brain, central nervous system, I mean. But
4 where you do see effects is in the bone marrow.

5 Q. That's where it targets?

6 A. That seems to be the case.

7 Q. What is the route? Maybe I'm not using the
8 right terminology that you're used to. What is the
9 pathway by which the metabolites travel to the bone
10 marrow?

11 A. The blood.

12 Q. So it's not the lymph system?

13 A. No.

14 Q. It's the blood?

15 A. Yes. And there is a whole series of --

16 Q. Let me ask you: This discussion we're having
17 about whether it's skin, ingestion, or inhalation, once
18 it gets into the bloodstream, there is no reason to
19 think that the -- the effects will be any different? Is
20 that generally accepted in your field?

21 A. There's no reason to think for benzene. Now, I
22 think for other chemicals that's not -- that's not going
23 to be the case.

24 Q. Why is benzene different?

25 A. Well, I think because it's so readily absorbed,

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1 at least through the GI tract and the lung, it's not too
2 readily absorbed through the skin. But I don't know the
3 answer to that. I think you have to just take it by a
4 chemical-by-chemical basis. But there's no reason for
5 me to think that once a benzene molecule gets into the
6 bloodstream -- now, okay. You could argue that benzene
7 through the lungs or benzene through the dermal
8 exposure -- the way the circulatory system works and the
9 way our -- the vasculature of the inside works, when
10 things are absorbed from the gut, it's called first pass
11 metabolism. And there's a whole series of vessels that
12 takes things from the gut straight to the liver. So
13 anything that's absorbed via the GI tract goes right to
14 the liver via the portal vein.

15 So there -- in some cases it's metabolized
16 to the point where it's not active. That's why a lot of
17 drugs don't work if you take them orally, because
18 they're -- they're broken down and metabolized before it
19 gets into the systemic circulation.

20 In the lungs and through the skin, it will
21 go around once before it gets to the liver. It will
22 still get to the liver, but it will take it two or three
23 minutes; whereas, in the gut it's the first thing that
24 it does. So you would have a slight kinetic difference
25 in benzene. It would hang around in the blood maybe two

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1 minutes longer before it goes into the -- into the liver
2 where it's going to be metabolized.

3 Q. So you think the lungs and the skin are more
4 alike than the GI tract from a kinetic standpoint?

5 A. No, not at all.

6 Q. I thought that's what you just said.

7 A. No. What I said was in terms of the
8 circulatory system and the way benzene is going to be
9 metabolized, that it would get to the liver at
10 approximately the same rate if it gets into the
11 bloodstream. But, no, they are actually completely
12 opposite. The lungs are set up for absorption. It's
13 their physiological purpose, is to absorb oxygen. So
14 they're very thin and they're made to have this -- the
15 skin is set up as a barrier against absorption, so
16 they're -- functionally they are almost completely
17 opposite of one another.

18 Q. What I meant was once it enters the
19 bloodstream, whether it's by the lung or by the skin,
20 you said in theory there is a difference between those
21 two and the GI tract.

22 A. Yes. I think probably once it gets in, because
23 of where it's going to have to go, yeah, it probably
24 would stay in there for another minute or two before it
25 makes it to the liver.

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1 Q. But based upon the information that you
2 currently have today, the scientific information,
3 there's no reason to believe that the effect inside the
4 body, once it enters the body via either of those three
5 routes, is different, correct?

6 A. There's none that I'm aware of.

7 Q. That's your field of toxicology, right?

8 A. Correct.

9 Q. Any more articles?

10 A. This one was just -- by Kipen, and there is a
11 whole bunch: Ward, Townsend, and these are the
12 Pliofilm. And it doesn't pertain directly to what you
13 asked, because they didn't look at 1 part per million,
14 but they saw changes at greater than 20 or 30 and they
15 didn't see changes at less than 10. So there are
16 some -- some studies that would --

17 Q. This is Kipen, Cody, and Goldstein, 1989?

18 A. Right. There's a bunch of those. There's
19 probably five or six papers that looked at -- the way
20 they set it up was, you know, fraught with some problems
21 because they -- they didn't know what the -- it was
22 difficult for them to correlate the blood work that was
23 taken in the -- in these work -- in these facilities
24 back in the '40s and '50s, with air monitoring. So they
25 tried to decide what the people might have been exposed

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1 to based on occupational levels and what the regulatory
2 levels were.

3 At any rate, they could see that as the --
4 as the benzene exposures decreased through time, the
5 blood counts increased through time. So that seemed to
6 be consistent with our understanding, that back in the
7 '40s they were exposed to enough benzene to where their
8 blood counts were suppressed, at least some of the
9 workers.

10 Q. Did you pull out some other articles?

11 A. Well, I am looking at my report and I reference
12 a sentence in here: "Most studies indicate that air
13 concentrations of benzene must be approximately 1-10 ppm
14 averaged over an 8 hour time-weighted average before
15 cytopenias are observed," which again is not consistent
16 with Mr. Oakley's exposure history.

17 And then the -- it looks like the ones that
18 I didn't -- Aksoy I reference. He doesn't have data at
19 1 part per million, but he has data at pretty high
20 levels where he didn't see any hematopoietic changes.

21 16 --

22 Q. You mean on a subacute basis?

23 A. On a sub Q basis?

24 Q. Subacute.

25 A. Subacute basis?

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1 Q. We're not addressing malignancies right now,
2 right?

3 A. No. No. Aksoy and the Turkish workers, he
4 looked at pancytopenias and other hematopoietic changes.
5 But those Turkish shoe workers studies, it was sad.
6 They were exposed to really high levels of benzene, 200
7 to 600 part per million on a time-weighted average with
8 peaks over 1,000. So, I mean, it gives us some idea as
9 to what high dose exposure can do, which is useful. But
10 it's not all that germane to the really low levels.

11 The two Collins papers which I pulled out.
12 Green, from 1981, that's an animal study.

13 Acute and Chronic Dose/Response Effect of Benzene
14 Inhalation on the Peripheral Blood, Bone Marrow and
15 Spleen Cells of CD-1 Male Mice. There's probably 50 of
16 those.

17 Q. Are those in your -- in your --

18 A. I didn't reference all of them. I have a bunch
19 of the animal studies here, which you can have. They go
20 back 50 years where people have been exposing animals to
21 benzene, and -- and I didn't reference all of them in
22 here.

23 Q. Let's do it this way.

24 A. The Lan paper I gave you. Go ahead. Keep
25 going.

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1 Q. Is this body of literature regarding the
2 low-level exposures, the subacute effects
3 from low-level exposures relevant to Mr. Oakley's case?
4 Are you relying on that body of literature for your
5 opinions?

6 A. Yes. I mean, because of Mr. Oakley's
7 pancytopenia, which I, you know, briefly discussed in
8 the report, and because of the established association
9 between benzene exposure and pancytopenia, I -- I was
10 very curious about that potential relationship. And so,
11 yeah, I -- I reviewed that literature very thoroughly to
12 see whether it matched what we saw with the
13 benzene-exposed workers or whether it did not.

14 Q. And so in your report, do you identify the
15 literature that you're relying upon for this for
16 pancytopenia topic?

17 A. Yes. Yes.

18 Q. And can you just tell me -- if you can point to
19 the area of the report.

20 A. That list of references. And I was going
21 through them. The only ones that -- I mean, I've pulled
22 out a lot of these that we've already talked about.
23 There's a few in here that are animal studies, but
24 they're still germane but we haven't specifically pulled
25 those out.

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1 Q. You're referring to Page 11, specifically the
2 citations 4, 16, 17, 31, 44, 46, 67, 77 through 79, and
3 92, correct?

4 A. Correct. Yes.

5 Q. What caused Mr. Oakley's pancytopenia, if you
6 know?

7 A. Well, I mean, people get pancytopenia for all
8 kinds of reasons. There's -- there's a very long list
9 of drugs that cause pancytopenia in an idiosyncratic
10 manner: Antibiotics, nonsteriodals, all kinds of
11 different drugs. There was some discussion in his
12 medical records that 'maybe his blood work might have had
13 something to do with his antidepressant use. I noticed
14 that he was on Allopurinol, which is an antioxi-dase
15 inhibitor that might have some hematopoietic effects.
16 So there's plenty of possibilities. But, no, I can't
17 tell you definitively what caused it.

18 Q. In your report, do you set out a specific
19 opinion as to what caused his pancytopenia?

20 A. No.

21 Q. That's not something you were asked
22 specifically to address in this case, was it?

23 A. I don't recall whether I was asked to do it or
24 not. I would have told them I couldn't do that.

25 Q. Is that generally left to a medical doctor?

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1 A. I don't think in this case a medical doctor
2 could tell you definitive. Matter of fact, there were
3 all kinds of medical doctors trying to figure out what
4 caused his cytopenia and none of them could do that.

5 Q. What information would you need to determine
6 what caused Mr. Oakley's pancytopenia?

7 A. Him personally? Mr. Oakley, based on his own
8 medical history, or any person with pancytopenia?

9 Q. Mr. Oakley.

10 A. I don't know that it can be determined with
11 that kind of certainty, particularly in a retrospective
12 fashion where you're just looking back in time and
13 saying he had it in '93 or '92. We don't have any
14 evidence of it beforehand. I don't know that it's
15 possible to say definitively what caused it, no matter
16 what else you have.

17 Q. Can you determine what caused his pancytopenia,
18 based upon reasonable medical probabilities, as opposed
19 to certainty or with the definitiveness that you've
20 referenced?

21 A. Well, I mean, I think if -- I mean, you look
22 back in his medical records. And in 1991, he was
23 treated with chloramphenicol or some other potent
24 antibiotic, and then in '92, he showed up with
25 pancytopenia. Yeah, then I think you could say that

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1 those are pretty likely to be causal.

2 Without that information, I don't know of a
3 way to do that. You could speculate and say maybe it
4 was this, maybe it was that, which seems to be what most
5 of the physicians were doing.

6 Q. As you sit here today, you're not in a position
7 to tell us, based upon reasonable medical probabilities,
8 what caused his pancytopenia, correct?

9 A. I can't tell you what caused it. The only
10 etiological -- potential etiological factor that I
11 evaluated thoroughly was benzene and that did not seem
12 to be a causal link.

13 Q. And did -- how did you rule out his benzene
14 exposures as causal -- causing his pancytopenia?

15 A. As outlined in the report, based on the timing
16 of when his exposures to benzene would have dropped off
17 precipitously, which would have been 1978, and you
18 didn't see this arise until '92. That -- that doesn't
19 work. That doesn't match. The fact that, really, his
20 air exposures weren't high enough, based on either
21 exposure estimate that I saw, to -- to probably cause
22 pancytopenia.

23 Q. What do you mean, either estimate?

24 A. Well, from Stephen Petty or from John Spencer.
25 The fact that when you looked at his blood work, it --

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1 well, the next one was that it was not reversible. Most
2 of the time when you remove -- because benzene, as
3 metabolites are damaging proliferating cells, cycling
4 cells, which is then manifest pretty quickly in the
5 periphery as pancytopenia -- when you remove them from
6 the benzene exposure, their blood counts return to
7 normal.

8 It's been shown in animals, people, for --
9 you know, for the last 100 years we've known that it's
10 typically a transient event. When you see it being
11 refractory to that and it persists, then it's an
12 indication of, okay, it's not associated with benzene
13 exposure or perhaps that there was some kind of damage
14 to the bone marrow.

15 At any rate, then, the last one is that the
16 most sensitive endpoint -- this is debatable and you'll
17 find papers on both sides -- but the most sensitive
18 endpoint in most studies is the lymphocytopenia. So the
19 lymphocytes -- including some of my own work --
20 lymphocytes are very sensitive to benzene exposure or
21 the metabolites. And at no point through his medical
22 records did I see a decrease in the lymphocyte number.
23 Which, you know, it's not a -- not an absolute telling
24 fact, but it -- but it does seem to indicate that it's
25 not consistent with benzene.

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1 Q. Anything else?

2 A. I think that's what I've put in -- yeah, that's
3 it.

4 Q. That's in your report?

5 A. Yeah.

6 Q. Can you find the section on -- the first issue
7 I wrote down was timing.

8 A. Uh-huh. (Witness answers affirmatively.)

9 Q. Can you find that section of your report?

10 A. Page 11. The very first thing at the top of
11 the page. "1) peripheral cytopenias are observed
12 relatively quickly following benzene exposures and would
13 not be expected to wait 12 to 14 years after his alleged
14 exposures to Liquid Wrench to manifest themselves." I
15 mean, that's -- that's not -- if you're just talking
16 about a peripheral cytopenia, that would never -- that
17 would never happen.

18 Now, if you're talking about a peripheral
19 cytopenia that may be related to some underlying disease
20 process, then you could probably find some literature
21 that would support it. But just a peripheral cytopenia,
22 it's going to happen within weeks. Sometimes it happens
23 within a week, which is why it's useful or was useful in
24 terms of being a biological monitoring system for
25 workers. If it took 12 years to manifest itself, it

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1 would be useless.

2 Q. So in order for somebody to have a pancytopenia
3 that you relate to benzene exposures, it has to be
4 diagnosed within weeks of the exposure?

5 A. Has to be diagnosed? Well, I mean, I'm not
6 sure that a subtle cytopenia would necessarily be
7 detectable. So perhaps someone, if they weren't part of
8 a medical monitoring system within the workplace, they
9 may have a subtle peripheral change two weeks after some
10 high dose time period and it goes undetected. And then
11 they never get it diagnosed. And two months later,
12 three months later, their blood counts are back to
13 normal.

14 Q. So how do we know when Mr. Oakley's cytopenia
15 first appeared?

16 A. We don't. I mean, the only evidence that I
17 have is when they first noticed it was in 1992. To
18 think that it persisted from when his high dose
19 exposures would have likely been, ending in 1978,
20 that -- that goes against the reversibility part. And
21 it wouldn't hang around for 12 years after he was no
22 longer being exposed to the levels of benzene that could
23 damage that blood system.

24 Q. What if he continues to have exposures after
25 '78?

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1 A. Well, if he continued to have exposures to
2 sufficient levels of benzene -- I mean, we're all
3 exposed to benzene, so if he did have exposures to
4 benzene after 1978 -- we all do.

5 Q. Occupational exposures.

6 A. Well, even occupational exposures. I mean,
7 there's plenty of studies that we've been talking about
8 where people that are exposed to 0.1 part per million on
9 a time-weighted average don't have any blood changes.
10 So he's going to have to be exposed to 1 or maybe 10
11 part per million years over an extended period of time,
12 and then, sure. I haven't seen any evidence of that
13 based on, you know, either John Spencer or Stephen
14 Petty.

15 Q. So you would expect to see in that '78 to '92
16 time period exposures between 1 to 10 part per million?

17 MR. GRAY: Objection; form.

18 MS. YATES: Same objection.

19 A. Well, I would expect if -- if -- if the
20 cytopenias, either way, if either -- if he was produced
21 -- if the exposures that he had prior to 1978 caused his
22 cytopenias, it is not consistent with the scientific
23 literature that they would have persisted for 12 years.

24 Q. (By Mr. Lubel) I've heard you say that. Now
25 I'm trying to find out if he'd got continued

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1 exposures --

2 A. I'm getting there.

3 Q. Sorry.

4 A. I'm kind of setting it up.

5 Q. Sorry. I didn't mean to interrupt.

6 A. Then the other part is if he didn't have a
7 peripheral cytopenia until 1992, but his exposures ended
8 in '78, that's not consistent.

9 In order for you to bridge that gap between
10 '78 and '92, there would have to be exposures that were
11 of long enough duration and high enough to where you
12 would think that cytopenias could occur.

13 Q. How long and how high?

14 A. Like we've been talking, somewhere greater than
15 1 part per million, maybe as high as 10. 200 would
16 certainly do it. But on a low end, probably more than 1
17 and not a single-use kind of thing, but, you know,
18 prolonged, over time-weighted average weeks, months.

19 Q. You're saying these cytopenias are nonmalignant
20 effects, correct?

21 A. Correct.

22 Q. When you look at benzene literature?

23 A. When you look at any literature.

24 Q. That's right. So -- but specifically when you
25 look at the benzene literature, there seems to be some

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1 distinction between nonmalignant effects and malignant
2 effects, correct?

3 A. Well, sure. Yeah. I mean --

4 Q. Exposures, the timing, it's kind of -- are they
5 different ballparks or fields of play?

6 A. I -- I think they are, but not everyone does.
7 And some people would argue that you have to have
8 evidence of hematotoxicity before you're at increased
9 risk of developing AML. There are some examples of
10 where people just get AML and there really was no
11 preceding anything, but that doesn't appear consistent
12 with most of our understandings. So I mean, I don't
13 think that's definitively worked out.

14 Q. So the latency period that troubles you about
15 Mr. Oakley is the one with relationship to his
16 cytopenias and his alleged benzene exposures?

17 MR. GRAY: Objection; form.

18 MS. YATES: Same objection.

19 A. No, that's troubling to me. The latency -- I
20 mean, the most important latency argument has it do with
21 his benzene exposures and his diagnosis of AML. And
22 that doesn't match at all. I mean, the -- they -- there
23 have been really good analysis of three, probably, of
24 the most important cohorts of benzene-exposed workers:
25 The Health Watch, NCI, and the Pliofilm. And all three

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1 of those have had analysis done to try to understand the
2 association between the timing of exposure and the risk
3 of disease. And they're all consistent. And they all
4 say the same thing; and that is, that it's only
5 exposures that occur within 10 to 15 years of diagnosis
6 that matter. Those are the only ones that influence the
7 risk of AML to any appreciable degree. Anything that
8 occurred earlier than 15 years does not affect the risk
9 estimate.

10 So that time frame, that time window, is
11 the most important in terms of understanding a person's
12 risk. It's consistent upon those three cohorts. It's
13 consistent with radiation, with chemotherapy. I mean,
14 it fits in with the biology of our understanding of the
15 chemically-induced disease.

16 So for Mr. Oakley, diagnosed in 2005,
17 really, the only exposures that based on that analysis
18 would matter occurred from 1990 post.

19 Q. (By Mr. Lubel) What caused his AML?

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

21 Q. Have you formed an opinion?

22 A. As to what caused it?

23 Q. Right.

24 A. No.

25 Q. It's not in your report, right?

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1 A. No. I think 80, 85 percent of the AMLs, at
2 least as we understand it today, are idiopathic. We
3 don't -- we don't know what caused them. And the other
4 10 or 20 percent are -- are definitively related to
5 chemotherapy.

6 Q. Chemotherapy did not -- did not cause his AML,
7 correct?

8 A. No, I did not see any evidence of that.

9 Q. Let's go back to the cytopenias for a minute,
10 because I want to make sure we're communicating.

11 It's your opinion that for the cytopenias
12 to be causally linked to the benzene exposures, the
13 exposures need to take place within a time period
14 relatively close to the diagnosis of the cytopenias,
15 correct?

16 A. Well, I mean, there could always be a
17 disconnect between the diagnosis and when it occurred.
18 I mean, diagnosis is a human thing where the guy went
19 into the clinic and said, I'm getting too sick too
20 often, or, I feel bad, or whatever and they look.

21 What I'm saying is that our understanding
22 of the temporal relationship is that cytopenias occur
23 pretty quickly after the benzene exposure, because of
24 the kinetics of the marrow and what the benzene is doing
25 in the marrow that presents itself as a cytopenia. So

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1 that's the first.

2 Second is they don't last. They're
3 generally reversible unless you've been exposed to high
4 enough levels and you've really done significant damage
5 to the bone marrow.

6 Q. So you're saying the cytopenias will persist?
7 They'll hang around, if you've had high enough exposures
8 to benzene?

9 A. That seems to be the case, you know, in a small
10 percentage of the people. And in some highly exposed
11 workers you don't see any cytopenias, you don't see
12 pancytopenias. In the Aksoy -- back to those Turkish
13 shoe workers, they were exposed to thousands of part per
14 million and pancytopenias, I think, were evident in 25
15 or 30 percent of those workers. So sometimes you don't
16 see it.

17 Q. How do we explain that?

18 A. Individual variability, I guess. I mean, he
19 was specifically looking for it. Now, it is possible
20 that his definition in '70 is different than our
21 definition in 2008, and that he saw subtle changes
22 and -- and didn't report them; or that, you know, now we
23 use automatic blood counting systems and flow cytometry
24 which is extremely accurate. In the '70s he would have
25 taken peripheral blood and counted on a microscope, and

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1 maybe he didn't see it. You know, the fact is -- and
2 then some of those recovered, some of them died from
3 aplastic anemia, which is really what they had, and a
4 small percentage of them went on to develop AML.

5 Q. But pancytopenia does not need to precede AML
6 for there to be benzene causation, correct?

7 A. Pancytopenia does not need to --

8 Q. Proceed AML?

9 A. That seems to be a consistent phenomenon
10 because the chemicals that cause AML also damage the
11 marrow. So you could argue that if you don't see it,
12 it's because you didn't look or because the person
13 didn't present with it or whatever. There are
14 definitely examples of where the AML just appeared as
15 frank disease.

16 Q. But as you sit here today, based upon the
17 current body of scientific and medical literature, do
18 you have an opinion as to whether pancytopenia must
19 precede AML for there to be causation?

20 A. No, I would not say that it must precede. No.

21 Q. If it does precede it, does it make it more
22 likely?

23 MS. YATES: Objection; form.

24 A. If you saw pancytopenia or myelodysplasia
25 that -- that was diagnosed prior to the -- to the

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1 diagnosis of AML, that would fit in with our
2 understanding of what a chemically-induced AML looks
3 like.

4 Q. (By Mr. Lubel) It would be more consistent
5 with it?

6 A. With the scientific literature, yes.

7 Q. Did Mr. Oakley have MDS?

8 A. I've seen no indication in the medical records
9 that he had MDS. In fact, there was a -- there was a
10 differential diagnosis in '93 where that was something
11 that they were looking at. And they -- and they found
12 no dysplastic changes. Again, in '98 I think they found
13 no dysplastic changes.

14 Q. Do you have any reason to believe that he had
15 MDS, based upon all of the information you've seen?

16 A. I'm not really qualified to make that
17 diagnosis, but no one has -- I've not seen in the
18 medical records and Dr. Levy's report didn't mention it.
19 I've seen no -- I know Ethan Natelson suggested it in
20 his report, that perhaps he had myelodysplasia.

21 Q. Possibility?

22 A. Possibility.

23 Q. As you sit here today, are you assuming that he
24 did have MDS or didn't?

25 A. I'm assuming -- as I'm sitting here right now,

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1 I'm assuming there was no evidence of it; that he did
2 not. It wouldn't change my opinions, really, either
3 way --

4 Q. Either way?

5 A. -- but... (Pause.)

6 Q. But he did have pancytopenia that preceded his
7 diagnosis of AML, correct?

8 A. Yes.

9 Q. So although not dispositive, it would be a clue
10 or something that would lead you to more consistency if
11 he's got the right benzene exposures, correct?

12 A. If he has the right benzene exposures and the
13 timing of those exposures were accurate, if -- I've
14 never seen it last that long, this pancytopenic latency
15 period. And if it's myelodysplasia, even less so. It
16 happens quickly. It's an aggressive disease and it goes
17 to AML pretty fast. I mean, six years has been the
18 absolute longest I've seen in the literature. And
19 usually it's a matter of months. So '92 to 2005, 13
20 years, that doesn't really match.

21 His cytogenetics absolutely do not match.
22 So there's other issues with -- with his -- his disease
23 presentation that leads me to believe that it had
24 nothing to do with benzene.

25 Q. We're going to go off the record for you to do

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1 this, but will you pull your literature on the -- on the
2 timing?

3 A. Okay.

4 Q. Do you mind?

5 A. The timing of what?

6 Q. The first point you made about the cytopenia.

7 MR. GRAY: Can you get the question out,
8 just so we don't -- because I'm not sure which timing
9 issue.

10 Q. (By Mr. Lubel) The point you made on Page 11
11 is the peripheral cytopenias are observed relatively
12 quickly.

13 A. Okay.

14 (Short recess from 10:12 to 10:17 a.m.)

15 EXAMINATION (Continuing)

16 BY MR. LUBEL:

17 Q. What folder did you pull out, Dr. Pyatt?

18 A. This is all the -- I mean, this is certainly
19 not all of them that have been published -- but all of
20 the animal studies on benzene exposure that I've
21 collected. And with regard to timing, that seems to
22 inform that question better than anything else.

23 Q. Better than human studies?

24 A. The problem with human studies, all you know is
25 that you've gone into the workplace and they've been

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1 exposed to benzene and then you go in and look at their
2 peripheral blood. And if you see a change, you don't
3 know if that was because of something that happened two
4 days before or a month before or whether or not that
5 was -- was an abnormally low value. Maybe that's just
6 the way that person always is. And you'd have to --
7 there's a lot of problems in interpreting that
8 literature.

9 Whereas, with the animals, you expose them
10 to benzene on Tuesday, you go in a week later, you look
11 to see what's happening, you can see cytopenias. So
12 it's clearly a very short time before you will start
13 seeing that.

14 Q. It's just more of a controlled test or
15 environment?

16 A. Exactly.

17 Q. Are all of the --

18 A. There's tons of them.

19 Q. Can you separate the timing articles, animal
20 articles, that you're relying on?

21 A. I mean, I will -- I will rely on the ones that
22 are in my report. But almost all of these, where
23 they've gone in and taken a time point, you will start,
24 like -- you know, here's where they started it. They
25 started seeing changes at 20 days. They didn't -- they

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1 even saw a little bit of change, probably, in day one.
2 Here they saw a change at day one or two, and by 30 days
3 it was -- you know, they had completely wiped out
4 whatever it was that -- that particular CFU assay. So
5 that's --

6 Q. What type of symptoms were the rats complaining
7 of?

8 A. I don't think the rats were doing much
9 complaining.

10 Q. What? You're kidding, aren't you?

11 A. You were kidding.

12 Q. No, I'm serious. There were no outward
13 manifestations of the animals?

14 A. Oh, you said what were they complaining of?
15 That was the part that --

16 Q. That was a -- that was in jest, that part.

17 A. Okay. All right.

18 No, you know, a lot of these do go
19 undetected because of that. You will sometimes see an
20 increased -- an increased frequency of infection, but in
21 these animal studies, they're in a controlled
22 environment where there's no infectious organisms. So
23 there have been studies on benzene that have actually
24 tried to infect the animals after high dose exposure to
25 benzene and have seen an increase in the infectivity

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1 because of this immunosuppressive effect at the really
2 high exposures. But that's one.

3 Q. Were these high exposures --

4 A. Most of them, yes.

5 Q. -- to the animals?

6 A. Yes.

7 Q. What range are we talking about?

8 A. Well, this one was 4 -- inhalation of 100, 300,
9 and 400 part per million produce dose dependent
10 decreases in blood lymphocytes, bone marrow cellularity,
11 marrow content of spleen colony forming units, and some
12 other things.

13 Q. Are you talking about 100 part per million was
14 the low range?

15 A. Yes -- well, no. In this one they used mice --
16 male mice have been exposed to benzene at air
17 concentrations of 10, 25, 100, 300, 400, and 3,000. And
18 they saw no effects at 10 or 25 part per million.

19 Q. Do we have any human studies that address the
20 timing issue at exposures lower than 10 ppm?

21 MR. GRAY: Object to the form.

22 MS. YATES: Same objection.

23 A. I -- the only -- I don't know of any. I mean,
24 the only way I think you could do that was if you found
25 a worker, took their blood, they came into the

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1 workforce, they were exposed at those levels, you took
2 their blood -- I mean, there might be some data like
3 that in these occupational surveys, but to my knowledge,
4 I haven't seen it published, other than the ones we've
5 already talked about. You're not going to get that much
6 information on the timing, because you are just taking a
7 snapshot of what's going on in that worker.

8 Q. (By Mr. Lubel) As you sit here, you can't
9 think of any human literature that specifically
10 addresses the timing issue?

11 MR. GRAY: Objection; form.

12 MS. YATES: Same objection.

13 A. I mean, I've referenced a couple. Aksoy, No.
14 4, he made mention of it. Hematological effects of
15 chronic benzene exposure in 217 workers.

16 22, I think that's -- that's Cronkite.
17 That's an animal study.

18 29, Goldwater. Goldwater is a human study
19 and there was some timing in there, but this was from
20 1941 and they -- that was really high. There wasn't any
21 data below 10 part per million. I mean, I think 10 --
22 at that time, the PEL might have been 100. So they
23 probably wouldn't even have paid attention if it was
24 just 10.

25 31, animal study.

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1 67 --

2 Q. If it's not --

3 A. Rothman. So --

4 Q. If it's not listed in your report specifically,
5 is it fair to say that you're mainly relying on the
6 animal studies for the timing issue?

7 A. I mean, there's -- it's not solely on the
8 animals, because that is generally accepted, that it's
9 going to be a relatively quick phenomenon. Which again
10 is why they have relied on it for -- they don't do it
11 that much anymore, but they've relied on it as an
12 occupational way to monitor the workforce. So they look
13 at a worker and they see a change in the blood; they
14 take that worker out and that represents something that
15 has happened within the last, you know, month.

16 Q. How often do they need to test these workers
17 for their blood to make this determination?

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

19 Q. What would you recommend, as a toxicologist
20 that's studied benzene?

21 A. I mean, if you really wanted to know whether
22 anything -- well, I mean, I think if -- if the
23 occupational conditions within a workplace is -- is
24 consistent, then it probably doesn't matter. You just
25 go in and look to see whether or not any of those

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1 workers are -- have suppressions. If -- if there are
2 events going on in the workplace where people are being
3 highly exposed, then I would say that you need to look
4 within two or three months after that or it's possible
5 to miss it.

6 Q. Oh, I thought people were using these
7 biological markers as clues to what the exposures were.

8 A. As clues to -- you mean, in terms of quantity?
9 Magnitude?

10 Q. No, just the effects.

11 A. Well, they are looking to see what the health
12 effects were in the workplace, but they're not
13 correlating -- I mean, the -- the modern studies where
14 they're doing it now, they know what the monitoring is.
15 They know what the air concentrations are.

16 Q. Are you saying that companies shouldn't
17 evaluate the blood of their own workers unless exposures
18 are high?

19 A. No, I didn't say that at all. And I think they
20 do evaluate the blood of workers if they're exposed to
21 benzene at all.

22 Q. And then they pull them out, if their -- if
23 they've got blood results that don't look good, right?
24 That's what they should do?

25 A. Yeah, that's -- I mean, based on the

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1 literature, what doesn't look good and where those
2 changes are, and how they -- that's -- I don't know what
3 they're doing in these occupational settings.

4 Q. What should they do?

5 A. Well, I think if they see a cytopenia, you
6 know, and it's -- and it's -- well, the first thing they
7 should do is run the test again, which they do. Because
8 you can get aberrant results back just from the lab.

9 But if they see a change in peripheral
10 blood numbers, then they take that person out of the
11 workplace. That's my understanding.

12 Q. Put them in a different area of the plant?

13 A. I don't know.

14 Q. And then you expect the cytopenia to go away,
15 once removed from the exposure?

16 A. Most cases, yes.

17 Q. And then if that person develops an AML, you
18 know, down the road, are you going to say that there's
19 no way that that AML was caused by the benzene exposures
20 because the cytopenia went away --

21 MS. YATES: Objection; form.

22 Q. (By Mr. Lubel) -- or those don't go together?

23 MR. GRAY: Objection; form.

24 MS. YATES: Objection; form.

25 A. Well, there's just too many other variables

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1 before you could make a determination as to whether -- I
2 mean, I think it's entirely possible that a person could
3 get a cytopenia in the workforce, find another job, live
4 a nice happy life for 40 years and develop AML, just
5 like all kinds of people do. And yeah, they are
6 absolutely unrelated.

7 Q. (By Mr. Lubel) Can they be related, as well?

8 A. Sure.

9 Q. How do we tell the difference?

10 A. Well, you can tell the difference based on the
11 latency, based on when their exposures were, how high
12 their exposures were, what the cytogenetics look like, all
13 of the things we would evaluate in any AML case.

14 Q. I guess the point I'm trying to isolate is: If
15 the cytopenias vanish, that doesn't mean that the AML is
16 not related to benzene down the road, correct, in and of
17 itself?

18 A. The cytopenias vanish?

19 Q. Once you are removed from the exposure, they're
20 gone.

21 A. That -- that question I don't know has been
22 really addressed very well.

23 Q. How do we --

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

25 Q. How do we do that?

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1 A. Well, I think you would have to look at some of
2 these studies where they have reported cytopenias. And
3 I think that's what NCI is doing. And I know that's at
4 least part of what the Shanghai health study is doing.
5 You see these peripheral changes. Does that in fact,
6 increase that person's risk of later developing some
7 malignancy, MDS or AML or whatever. So you have to
8 follow those people and see whether or not there's an
9 association.

10 My understanding, sitting here now, that
11 there's not. That -- that you can have peripheral
12 cytopenias from a whole bunch of different things
13 that -- that do not ultimately cause you to develop AML.

14 Q. I thought we agreed earlier that you didn't
15 need to have cytopenias, such as pancytopenia, to relate
16 an AML to benzene exposure; is that -- didn't we agree
17 on that?

18 A. Uh-huh. (Witness answers affirmatively.)

19 Q. Is that true?

20 A. Say it again.

21 Q. I thought we agreed earlier that an individual
22 did not need to have pan -- a diagnosis of pancytopenia
23 in order to relate their AML to benzene exposures.

24 A. Yes.

25 Q. We agreed on that, right?

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1 A. We did agree on that.

2 Q. And so in a broader sense, would you agree that
3 the person need not have any preceding cytopenias in
4 order to relate their benzene exposures to AML?

5 A. If there was no preceding cytopenia, that would
6 be -- in my interpretation, that would be one thing that
7 would argue against it being related to benzene. But it
8 would not say, Okay, he did not have a cytopenia, his
9 AML could not have been caused by benzene.

10 Q. It is not dispositive, either way?

11 A. I'm not quite sure what that words means,
12 but...

13 Q. It's not conclusive in and of itself?

14 A. Okay.

15 Q. True?

16 A. True.

17 Q. This green folder, can I mark this as Exhibit 1
18 as long as the court reporter agrees to -- to give you
19 your originals back?

20 A. You can have that.

21 Q. Okay. I'm going to mark it on the outside of
22 the folder.

23 A. That's fine.

24 {Deposition Exhibit No. 1 marked.}

25 Q. (By Mr. Lubel) What does the green folder that

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1 we marked as Exhibit No. 1 represent?

2 A. This is a collection of animal data, animal
3 studies; Cronkite, Snyder, Green, and Tunek and even
4 Rich Irons and some others who through the years have
5 done long- and short-term exposure studies on various
6 animal species and what they found. And I just haven't
7 gotten around to putting it into a binder yet.

8 Q. Are these animal studies focused on a
9 particular issue or are they just animal studies and
10 benzene? These are not all timing studies, for example,
11 correct?

12 A. No. No.

13 Q. Do you see what I'm saying?

14 A. Yeah.

15 Q. This is kind of your -- the -- the animal
16 studies that you generally like to refer to in the
17 benzene world, correct?

18 A. Yes.

19 Q. This is --

20 A. I mean, there are others. But, sure, that's a
21 good representative pile of them. When they do -- when
22 they do an animal study -- and this just has to do with
23 ethics -- you -- you want to get as much information and
24 data as you possibly can out of that study. So you will
25 answer as many questions as you possibly can to validate

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1 taking the life of these experimental animals.

2 So the timing issue, you know, maybe they
3 didn't specifically say, We're going to do this to see
4 when the cytopenias arise. But just like I was pointing
5 out to you in the very first paper, you know, they're
6 taking blood samples two days, five days, ten days, 20
7 days. Well, you deduce what the timing was from their
8 data.

9 Q. What are the best animals to study for benzene?

10 A. Well, that kind of goes back around to what we
11 were talking about earlier. Depends what your question
12 is. I mean, I'm sorry, depends on what your scientific
13 question is. You said what is the best animal to study?
14 What are you looking for when you say the word "study"?

15 Q. Well, are some animals better to answer a
16 particular question than others?

17 A. Well, yeah. Like we discussed earlier, the --
18 most people use rats and mice because they're cheap and
19 they're easy to deal with and we understand their
20 genetics very well.

21 Those are used predominantly for
22 pharmacokinetic studies, understanding absorption,
23 metabolism, that kind of thing. And they do seem to
24 correlate pretty well with humans and nonhuman primates.

25 Pharmacodynamically there doesn't seem to

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1 be -- humans seem to be pretty different in the way that
2 humans respond in terms of these chronic exposures.

3 Q. Well, what about rabbits and guinea pigs and
4 monkeys? Do they have any place in benzene studies?

5 A. I don't actually recall ever seeing a monkey
6 study. I know they've used rabbits and guinea pigs and
7 they tend to fall out quite a bit more similar to the
8 rodents than to humans.

9 It wouldn't surprise me if there's been --
10 I don't know if there's been a monkey study, a nonhuman
11 primate.

12 Q. All right. Do these animal studies within
13 Exhibit 1, will they express dermal exposures as well?

14 A. I don't know the answer to that. My guess is
15 no.

16 Q. Let me do it this way. Have you, as part of
17 your role in this case, done specific research on the
18 dermal exposures to benzene?

19 A. Based on some of the allegations in this case,
20 I've tried to educate myself on dermal absorption.

21 Q. And have you, essentially, told us the basis of
22 your opinions about dermal absorption today, already?

23 A. Well, there's other stuff in the report. And
24 that actually is germane to the -- to the amended report
25 that I -- which we can go over it.

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1 But it's more of a pharmaco/toxicokinetic
2 issue: How much benzene can get through the skin? Not:
3 What does the benzene do once it gets into the systemic
4 circulation.

5 Q. Have you brought with you the literature on how
6 much benzene can get through the skin into the blood?

7 A. I've brought some. I'm not -- I'm not an
8 expert on this, so I'm sure there's -- there's way more
9 literature out there.

10 Q. Do you mind pulling those? Thanks.

11 A. (Examination of documents.)

12 Q. You've pulled another green folder that's got a
13 label on it, Dermal Uptake.

14 A. Yes.

15 Q. And I'm going to mark it as Exhibit 2, right?

16 A. Okay.

17 (Deposition Exhibit No. 2 marked.)

18 Q. (By Mr. Lubel) Are all of these referenced in
19 your report --

20 A. I don't -- I don't think so.

21 Q. -- your updated report you told me about this
22 morning?

23 A. No. I mean, some of them aren't that relevant.
24 Some of them aren't -- a lot of them have to do with
25 kind of the flux of how much goes through skin, and

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1 that -- that wasn't really something that I formed an
2 opinion on. So some of them are; not all of them.

3 Q. Are your opinions about the dermal absorption
4 contained in your report, your amended report?

5 A. Yes.

6 Q. Can you show that to me?

7 A. Yes.

8 Q. We'll mark that as Exhibit 3.

9 THE WITNESS: I think, Hector, you have
10 the -- the highlighted one.

11 MR. GRAY: I think you handed me one as
12 well.

13 MR. LUBEL: Make sure.

14 THE WITNESS: Here it is.

15 MR. LUBEL: You've got it back?

16 THE WITNESS: Yeah. That's it.

17 (Deposition Exhibit No. 3 marked.)

18 Q. (By Mr. Lubel) Exhibit 3 is your report dated
19 November 4th, correct?

20 A. Yes.

21 Q. This is the report that you -- that you handed
22 to me this morning?

23 A. Yes.

24 Q. And on page -- looks like 12 -- is the portion
25 that contains your opinions about dermal absorption?

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1 A. Yes.

2 Q. Is that correct?

3 A. Uh-huh, yes.

4 Q. Now, does your report set forth both your
5 opinions and the basis for your opinions?

6 A. Yes.

7 Q. Have you done any original research on dermal
8 absorption?

9 A. No.

10 Q. Have you ever had the opportunity to do so?

11 A. I've never asked.

12 Q. But have you ever been presented with an
13 opportunity where you could have studied dermal
14 absorption?

15 A. No.

16 Q. Why not?

17 A. Well, when I was actually doing research on
18 benzene and benzene metabolites, I was interested in the
19 toxicodynamic part of the bone marrow, how is the bone
20 marrow responding to toxic insult from benzene or from
21 any type of chemical. So how it got into the systemic
22 circulation and what happened to it at that point, it
23 wasn't a research interest of mine.

24 I mean, it's a -- it's a unique field where
25 people are trying to figure out whether something can go

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1 through the skin. I'm not a dermatologist. It's just
2 never been an organ that I was all that interested in.

3 Q. Who are the prominent experts in that field?

4 A. I don't -- I mean, the only one I can think of
5 by name is Maibach, Dr. Maibach, somewhere in
6 California. I mean, I think he has probably published
7 the most on, not just benzene, but germane to this case:
8 The ability of benzene to go through human skin.

9 Q. What did he study?

10 A. What did he -- I think he used cadaver skin,
11 but I'm not positive about that. So I think he had
12 several different -- he may even have used some -- some
13 radiolabeled -- I'm not -- I'm not positive. He's
14 published a lot on it.

15 Q. Hasn't he studied the rhesus or rhesus monkey?

16 A. That sounds right, rhesus monkey, yes.

17 Q. Is that how you pronounce it? Rhesus?

18 The reason I ask you about this is because
19 I seem to recall you having something to do with
20 Dr. Maibach's study on Liquid Wrench. Am I mistaken?

21 A. Well, I was with -- right as I was
22 transitioning out Chem Rep when I worked with Dennis was
23 when the Liquid Wrench simulation study was kind of
24 being discussed. I don't -- I don't think even at that
25 point it had been funded yet. But I had nothing to do

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1 with the study.

2 Q. Do you don't take any credit for it?

3 A. No.

4 Q. Did you design it?

5 A. Well, I don't think I did. I mean, I don't
6 think it -- one of -- one of the things that I did with
7 the Liquid Wrench study was come up with some
8 preliminary scenarios that I thought -- not being an
9 exposure person so I was really kind of out of my
10 element -- but that -- that this ought to be bracket
11 what a normal working exposure to Liquid Wrench would be
12 from low to the high end. And -- and so I -- so I
13 recall generating some scenarios about, you know, very
14 generic kinds of terms, what people could be doing that
15 could potentially represent that range. How that got --
16 and if that got manifested in the actual study, I don't
17 know.

18 Q. Did you keep a file on that?

19 A. I had a file that I gave to Tim.

20 Q. You did? When?

21 A. To Mr. Gray. Whenever you asked for it. Six
22 months ago.

23 Q. That long ago? How voluminous was it?

24 A. Not very.

25 Q. Are we talking about inches?

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1 A. No. No.

2 Q. Feet?

3 A. No. No. No. This was -- let's see, it seems
4 I recall that I had an E-mail or two. I had a -- there
5 was a budget request or -- not request, but sort of
6 breakdown of who was going to be doing what.

7 MR. LUBEL: Did you give it to me, Tim?

8 MR. GRAY: I don't want to jump into your
9 deposition. We produced it and, frankly, we brought
10 those pages to this deposition.

11 MR. LUBEL: Well, thank you.

12 MR. GRAY: We objected to your --

13 MR. LUBEL: We even let you interrupt when
14 you have something productive to say.

15 MR. GRAY: We filed objections to your
16 request for production, but at the same time we thought
17 if you wanted to look at it, we'd bring those pages.

18 MR. LUBEL: We'll get to that in a minute.

19 Q. (By Mr. Lubel) Were you aware that
20 Dr. Paustenbach charged about \$600,000 for that effort?

21 MR. GRAY: Objection; form.

22 MS. YATES: Same objection.

23 A. I don't know. I don't know what the actual
24 budget was. But I thought it was more than that.

25 Q. (By Mr. Lubel) So it wouldn't surprise you if

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1 that three-day study and the paper that followed it
2 would cost in excess of \$600,000?

3 MR. GRAY: Objection; form.

4 MS. YATES: Objection.

5 A. Oh, \$600,000?

6 Q. (By Mr. Lubel) Yes, \$600,000.

7 A. I thought you said 60.

8 Q. \$600,000.

9 A. Oh, no, I don't have any -- I don't know.

10 Q. Does it sound like a lot to you?

11 A. \$600,000 is a lot to anybody. But people's
12 time is -- is expensive.

13 Q. What would you have expected that study and the
14 effort that went into it to have run?

15 A. I don't know. I mean, I -- I don't remember
16 what they did. I remember we were talking about the
17 publication and that was going to be about \$100,000.

18 Q. To write the paper?

19 A. That -- that was my recollection.

20 Q. \$100,000 to write a paper?

21 A. Based on -- based on the timing of people. You
22 know, in the consulting world, writing a paper is more
23 expensive than it is in the academic world, for sure.

24 Q. How long does it take to write a \$100,000
25 paper?

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1 A. Well, that's going to depend on how many people
2 are working and what their hourly rate is.

3 Q. Wow. That's a pretty good business to be in.

4 A. Well, depends. I mean, it depends on how long
5 it takes you to write it.

6 Q. That's right up there with Tim Gray's billing.

7 MS. YATES: Objection; form.

8 A. I mean, yeah.

9 MR. LUBEL: I withdraw that one. You don't
10 need to answer that one.

11 THE WITNESS: Okay.

12 Q. (By Mr. Lubel) You don't know how much it
13 costs?

14 A. I don't.

15 Q. You don't have an opinion about what's
16 reasonable?

17 A. I've never done a simulation study.

18 Q. You were involved before the study actually got
19 started?

20 A. Peripherally, yes.

21 Q. And so did you recommend to the group that they
22 should do some dermal absorption part of that study?

23 A. No, I don't remember ever discussing dermal.

24 Q. You could have?

25 A. I guess if I thought of it.

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1 Q. Did anybody in the group discuss it?

2 A. Not that I -- not that I am aware of, but that
3 would -- gee, that would totally change the dynamics of
4 the -- of the study, to account for dermal absorption.
5 That's a hard, hard -- I don't even know how you would
6 do it.

7 Q. Is it possible?

8 A. Not in that scenario, I don't know how it would
9 be possible. I mean, I guess if you could -- if you
10 were looking at urinary metabolites, which they didn't
11 do, I don't know. That would be a hard thing.

12 Q. Could you have designed a study from this
13 Liquid Wrench simulation where you address the dermal
14 absorption in the study?

15 A. I don't know that I could. I mean, I could
16 take a stab at it and then have to talk to some
17 physicians and Maibach and some other people to see if I
18 was even close, but it certainly is way outside of my
19 area of expertise.

20 Q. How about Paustenbach? Was it within his
21 expertise?

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

23 Q. He was your partner, wasn't he?

24 A. He owned the company.

25 Q. You worked together?

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1 A. Not very much.

2 Q. Well, not anymore at all now.

3 A. Not anymore at all, but even then, not very
4 much. I had my own thing; he had his own thing. We got
5 together for -- for periodic sort of budgetary meetings.
6 But, no, I didn't work with Dennis too much.

7 Q. Is he considered an expert in the field of
8 dermal absorption?

9 A. I guess that would depend on who you ask.

10 Q. Well, I'm asking you.

11 A. Oh. I think Dennis knows more about dermal
12 absorption than I do. I would allow that.

13 Q. Short of Maibach, is he considered an expert in
14 the field of dermal absorption in the United States?

15 A. Well, they approach it from two different
16 directions. Dennis is kind of a paper exposure
17 assessment reconstruction kind of person. I think he's
18 very well-known and respected in that field.

19 Maibach is trying to understand exactly how
20 much benzene can actually go through the skin. I don't
21 think Dennis has ever conducted those kinds of in vitro
22 analyses.

23 Q. What you're saying is Paustenbach -- that's
24 Dennis, right?

25 A. I'm sorry.

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1 Q. -- he's relying upon people like Maibach to
2 give him information that he can use in his modeling?

3 A. I would assume that's correct.

4 Q. Or --

5 A. He had a book chapter that he wrote on dermal
6 absorption that I read a long time ago. And there are
7 many variables in there; some of which I'm sure he's
8 going to have to get some input somewhere.

9 Q. Other than Maibach and Paustenbach, do you know
10 of anybody else that is considered to have an expertise
11 in that area of dermal absorption?

12 A. It's just not my field. I mean, there's --
13 there's plenty of people out there that are very
14 knowledgeable about dermal absorption.

15 Q. Who are they?

16 A. Well, I mean, every pharmaceutical company that
17 applies drugs through the skin are going to have
18 dermatologists and people that understand the ability of
19 chemicals to transfer through the skin. I just don't
20 know who those are.

21 Q. I'm specifically -- specifically talking about
22 benzene. Who are the people, other than Maibach and
23 Paustenbach, who you consider to have sufficient
24 expertise in dermal absorption of benzene? Let me be
25 more specific.

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1 A. Okay. Me, personally, I don't know that many.
2 So I -- so I can't really answer that.

3 Q. Can you think of any other than Paustenbach and
4 Maibach?

5 A. Well, there was one, Susten, S-u-s-t-e-n, that
6 published a paper that -- that I thought was -- it's the
7 one that ACGIH seems to base some of their decisions on,
8 but I don't know that person.

9 Q. Susten, he wrote the paper on the estimates of
10 tire builders, correct?

11 A. Yes.

12 Q. That was discussed in the OSHA hearings?

13 A. I don't know about that last part, but that
14 wouldn't surprise me.

15 Q. Do you recall that the percentage content of
16 the product used in tire building was 0.5 percent --

17 A. No, I don't.

18 Q. -- benzene?

19 A. I don't. I don't remember.

20 Q. You just don't have a recollection of that?

21 A. No.

22 Q. Skin absorption is an accepted route of
23 exposure for benzene in humans, correct?

24 A. It's possible, yes. It's accepted.

25 Q. There's three, right?

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1 A. Yes.

2 Q. What are they?

3 A. Oral, inhalation, and dermal.

4 Q. And oral means drinking it?

5 A. Eating it.

6 Q. Ingestion?

7 A. Ingestion.

8 Q. And dermal means skin, right?

9 A. Right.

10 Q. Are you familiar with any industries where
11 their exposures to benzene are primarily skin versus the
12 other two?

13 A. No.

14 Q. Is that anything you've researched?

15 A. No.

16 Q. Do you know if mechanics typically have more
17 dermal exposures than they do the other routes? No
18 opinions?

19 A. I recall one paper, Laitinen, I think was the
20 author, and that was the group of workers they looked
21 at. And in that case he estimated -- she; I don't know
22 whether it's a he or she -- but that author estimated
23 that dermal represented, I think, somewhere between 1
24 and 80 percent, with the -- the best guess was 60
25 percent of that person's cumulative exposure.

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1 Q. But the route of exposure really depends on the
2 person and what they're doing, doesn't it?

3 A. The route of exposure depends on the person and
4 what they're doing?

5 Q. Right.

6 A. Yes.

7 Q. Most people, when you look at studies, are not
8 drinking benzene, correct?

9 A. Not that I've seen.

10 Q. We haven't seen those in the studies reported,
11 have we?

12 A. Well, there's a few, actually --

13 Q. Okay.

14 A. -- but not many.

15 Q. Most people are not eating it?

16 A. A few. But, no.

17 Q. Right?

18 It seems that most of the studies are
19 addressing people breathing it.

20 A. That's absolutely true.

21 Q. That's a true statement, right?

22 A. Yes. That is clearly the predominant pathway
23 in occupational studies.

24 Q. But when we look at an individual, his or her
25 particular exposures, and which pathways are dominant

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1 for them, really depend on what they do.

2 A. I would say that's accurate. I mean, you can
3 certainly come up with a hypothetical situation where a
4 person's overall benzene exposure is going to be driven
5 by dermal and not inhalation.

6 Q. In other words, you could have -- you could
7 have -- pick a mechanic that's wearing a respirator all
8 day, that's using his hands and is not wearing gloves,
9 what would be their predominant exposure?

10 A. I don't know. I mean, the -- I don't know how
11 well the respirator worked. But for sure, if you had
12 someone breathing contained air, and they were soaking
13 their feet in benzene, yeah absolutely dermal would be
14 driving that person's exposure.

15 Q. And the truth is, we have to examine that
16 person's individual job tasks in order to determine
17 which particular pathway is their most dominant?

18 A. Well, I would say that's true with -- with the
19 caveat that you can get generalizations from
20 understanding the industry and understanding what people
21 are doing. And like the ACGIH, their position is it's
22 likely to represent 20 to 40 percent. That seems
23 reasonable. And I think that's a fair estimate. I've
24 seen some language that -- maybe it was what you were
25 referring to in OSHA -- where they suggested that maybe

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1 a person's dermal might be as high as the inhalation.
2 So it would be kind of an even contribution. So you're
3 still within that same -- same ballpark. Going further
4 outside of that, and I don't think there's any evidence
5 to support it.

6 Q. Was it -- was it Susten -- are you familiar
7 with that article?

8 A. Uh-huh. Well, peripherally.

9 Q. Right. What were those people's dermal
10 exposures as compared to their inhalation exposures?

11 A. I think it was 20 to 40 percent.

12 Q. 20 to 40 percent?

13 A. Yeah, I think -- I'm not sure. It was at least
14 referenced as part of the document -- of the support for
15 the ACGIH making that determination in their TLV
16 documentation.

17 Q. And how were they exposed dermally?

18 A. Through their hands, I guess. I'm not sure.

19 Q. What were they doing? Do you remember?

20 A. It was -- they were using rubber-making
21 solvent, was my recollection.

22 Q. And what part of their hands were exposed to
23 the solvent?

24 A. I don't -- yeah, I don't know. We'd have to
25 get it out. I don't know the answer to that.

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1 MR. GRAY: Is that in one of these papers?

2 THE WITNESS: Yeah, I'm sure Susten is in
3 there.

4 Q. (By Mr. Lubel) Laitinen? Is that another
5 article that you referenced?

6 A. I think I referenced it, yeah.

7 Q. Were those mechanics?

8 A. Here's the Susten one. Seems to -- my
9 recollection is, yes, they were mechanics working in
10 a -- in a filling station.

11 Q. Have you relied on that article?

12 A. I read it. I mean, I think it's -- I think
13 it -- it contributes to sort of an understanding -- I
14 don't -- it's on the disk. Is it in my report? I think
15 I reference it. If it is, it's on the disk. But I
16 didn't print it out and put it in this binder.

17 Q. What are you talking about?

18 A. Laitinen.

19 Q. But you're familiar with it?

20 A. Yeah. Yeah. I've read it.

21 Q. Do you recall what they were exposed to that
22 contained benzene?

23 A. I think it may be gasoline, but I'm not
24 positive about that.

25 Q. And do you remember what they were doing with

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1 the gasoline or whatever the benzene-containing product
2 was to get it on their skin?

3 A. Parts washing. I don't remember. Just in
4 their normal work.

5 Q. And do you have an opinion as to whether a
6 parts washer using a benzene-containing product would
7 have predominantly more inhalation exposures or skin
8 exposures or do you know?

9 A. I don't think there is any way to answer that
10 question.

11 Q. You need more info?

12 A. Sure.

13 Q. And typically, you're going to rely on
14 industrial hygienists for that --

15 A. Yes.

16 Q. -- right?

17 A. Yes.

18 Q. Because that's kind of outside of your field.

19 A. You bet.

20 Q. Are you saying that dermal exposures to benzene
21 don't -- don't contribute to cause disease?

22 A. I don't think that's what I said.

23 Q. Do you believe that?

24 A. Well, what I believe is that dermal absorption
25 of benzene through vapor is considered negligible. You

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1 don't even need to consider that if you're doing risk
2 assessments and trying to calculate an exposure for a
3 person in an environmental environment. So that is how
4 I make -- I just didn't write that section very clearly.
5 And that's where I wanted to specify what I was
6 referring to.

7 Q. Dermal absorptions to vapor?

8 A. To vapor. To benzene in the air.

9 Q. As opposed to liquid?

10 A. Is negligible.

11 Q. As opposed to liquid?

12 A. If there is benzene in the liquid and you have
13 dermal absorption of the liquid, that's a different
14 issue.

15 Q. That can cause disease, correct?

16 A. I've never seen an example of where it caused
17 disease, but it can cause absorption. And now you're
18 right back to the same thing that we've been talking
19 about all day.

20 Q. Same as inhalation?

21 A. Once the benzene molecule is in, right.

22 Q. I just want to make sure if -- if we actually
23 see you at the courthouse that -- that you're not
24 telling the judge and the jury that skin absorption of
25 benzene cannot contribute to cause disease. That's not

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1 your position, is it?

2 MR. GRAY: Objection; form.

3 A. I would never -- I mean, inhaled, ingested, or
4 dermally-absorbed benzene is benzene. The quantitative
5 literature that -- that I would base a comparison of Mr.
6 Oakley or anyone's risk of AML to the literature, they
7 used inhalation. They did not account for dermal. And
8 when they did account for it, there was one or two, I
9 think, where they looked at it, and it was -- it made a
10 relatively negligible contribution.

11 So that is the basis of my opinion that
12 inhalation is the predominant pathway. But if a
13 molecule gets in through the skin, it's going to do the
14 same thing that a molecule that gets in through the
15 lung.

16 Q. (By Mr. Lubel) Okay. You've lived in the
17 United States for how long?

18 A. Ever, my whole life.

19 Q. For your lifetime. Are you generally familiar
20 with -- with football being more popular at least today
21 than soccer, in the United States?

22 A. Sure.

23 Q. That will change one day, possibly, correct?

24 A. I guess it's possible.

25 Q. Right?

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1 A. It's possible.

2 Q. But right now, it seems as though football is
3 more popular in the United States than soccer, correct?

4 A. I would agree with that.

5 Q. More people that have been studied had
6 inhalation exposures versus skin exposures, correct?

7 MR. GRAY: Objection; form.

8 A. Well, I think the -- the quantification in
9 these studies has been inhalation. I mean, ostensibly
10 they had dermal absorption. I don't know what they were
11 doing in the Chinese workplace. But I'm assuming that
12 they would have had the same kind of exposures that
13 anyone would have.

14 Q. (By Mr. Lubel) Is it harder to measure the
15 skin exposures than it is the inhalation exposures?

16 A. I think for sure it is. It's certainly going
17 to be more uncertain.

18 Q. It's a more difficult task, correct?

19 A. Yes.

20 Q. If it was as simple when Paustenbach and your
21 former company did the Liquid Wrench study, they'd just
22 gone ahead and done the dermal absorption test at the
23 same time, right?

24 A. Well, it's not simple. That's for sure.

25 Q. But if it was that simple, they would have done

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1 it?

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

3 Q. When we look at the reported epidemiological
4 literature, we see more quantitative reports of
5 inhalation exposures to benzene, right?

6 A. Yes.

7 Q. No doubt about it, right?

8 A. Yeah, that is the predominant exposure pathway
9 that people have quantitated.

10 Q. Do we know why it's reported more often than
11 dermal exposures?

12 A. My understanding is because dermal absorption
13 or exposure wasn't considered to be important.

14 Q. In other words, it's not dangerous?

15 A. Well, a molecule is a molecule. So a molecule
16 is going to be as dangerous as a molecule is depending
17 on however it gets in there. But overall, it wasn't
18 considered to be a pathway that would contribute
19 significantly to that workforce cumulative exposure.

20 Q. Why?

21 A. Because benzene isn't very permeable through
22 the skin.

23 Q. So that's why they didn't study it?

24 A. I think that's one reason. And they have
25 looked at it -- I mean, Dennis Paustenbach and Pamela

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1 Williams looked at it when they reevaluated the Pliofilm
2 cohort and they published it in 2004 and they included
3 that in their new Monte Carlo, which everyone agrees is
4 probably the best, most rigorous exposure assessment of
5 that cohort. And they did include dermal. And it made
6 2 or 3 percent, maybe 10 percent difference in their
7 cumulative exposures for those various tasks.

8 Q. Why is that important?

9 A. Well, because I'm saying that in -- in that
10 case it did not contribute very much to their cumulative
11 exposures.

12 Q. Why did they report it?

13 A. Because they studied it.

14 Q. So they took dermal absorption tests of that
15 cohort?

16 A. Well, I think they've made modeling assumptions
17 and did a risk-assessment-type approach. It's a
18 retrospective cohort. They didn't look at the workers
19 themselves. Those workers are gone. Most of them are
20 dead. They went back and said, What were they doing?
21 What were the skin absorptions to those solvents would
22 have likely been. How much benzene gets in, et cetera,
23 et cetera. And then calculate a quantitative dermal
24 exposure from that process.

25 Q. What -- what was their purpose in modeling

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1 benzene?

2 A. Well, their purpose was to go back -- and
3 there's always been some uncertainty and there's always
4 been some debate as to what the cumulative exposures
5 really were in this Pliofilm cohort. And that's --
6 that's important because the Pliofilm cohort drives our
7 regulatory positioning on benzene. So the exposures
8 that those people had that did and did not get disease
9 is critical in being able to accurately extrapolate to a
10 risk.

11 Q. So the skin exposures were important to
12 Paustenbach in that study?

13 A. Pamela Williams was the first author. It was
14 one of the changes that they made where they went in and
15 -- I think Dr. Paustenbach actually tried to include it
16 to some extent in 1992, when he did his original
17 exposure assessment. Rob Rinsky did not.

18 Q. So Paustenbach and Williams?

19 A. Pamela Williams.

20 Q. Is she a doctor, too?

21 A. She's a Ph.D.

22 Q. In what?

23 A. She got her Ph.D. or S, science, of doctorate
24 from Harvard School of Public Health. I'm not exactly
25 sure what her degree is in. She's good at exposure

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1 assessment. That's what she did when I was working with
2 her.

3 Q. So did you tell them when they did this dermal
4 modeling for benzene exposures of this cohort that it
5 wasn't necessary because dermal exposures don't matter?

6 MR. GRAY: Objection; form.

7 MS. YATES: Same objection.

8 A. I didn't tell them not to.

9 Q. (By Mr. Lubel) Why not?

10 A. I think part of why we know that it's not a
11 significant exposure route in most of these workplaces
12 in part is because of that.

13 Q. So we need their article to tell us whether all
14 workers in America have sufficient dermal exposures to
15 cause disease?

16 A. I didn't follow that question.

17 Q. What is it about those workers that they
18 studied -- what is it about their skin exposures that
19 tell us about Mr. Oakley's skin exposures? Were they
20 doing the same task or similar task?

21 A. How you can extrapolate those findings from the
22 Pliofilm cohort to Mr. Oakley, I'm not prepared to say.
23 But it -- but it just -- it just indicates that in
24 that -- in that environment, the dermal absorption
25 didn't contribute very much to their cumulative

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1 exposures.

2 Q. What -- what is that environment that you're
3 referring to?

4 A. The Pliofilm manufacturing process.

5 Q. Right. And I'm trying to get you to tell me
6 how does the Pliofilm manufacturing process in any
7 dermal exposures that take place in that process, have
8 to do with Mr. Oakley.

9 A. And I'm not qualified to do that. John Spencer
10 could perhaps do that. You know, there are others in
11 this case that I think would be better able to address
12 that question.

13 Q. Has anybody done that?

14 A. Done what? Taken -- taken the Pliofilm data
15 and tried to apply it to Mr. -- I don't know the answer
16 to that.

17 Q. To Mr. Oakley?

18 A. Yeah, I don't know.

19 Q. Have you brought with you any studies that
20 address dermal exposures where you -- where you intend
21 to say, This is the same or similar to Mr. Oakley?

22 A. I'm not going to say anything about dermal
23 absorption or very little. It's just not my field.

24 Q. So you hadn't picked out studies to say, This
25 study is like Mr. Oakley's exposures to the skin, or

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1 anything like that?

2 A. No. The only thing that I've said about dermal
3 absorption is that Stephen Petty's assumption that it
4 represented 20 times his inhalation is -- is outside of
5 anything that anyone has ever published. That's the
6 only real comment that I made.

7 Q. Okay. And so have you compared Petty's work to
8 the published literature to see if there's any reasons
9 that account for that difference?

10 A. I have not, no.

11 Q. Do you know anybody that has?

12 A. I would assume that that's going to be -- well,
13 I don't know.

14 Q. Don't assume. I'm asking you if you know if
15 anybody's done that.

16 A. I don't, no.

17 THE WITNESS: I'm sorry. You've been
18 really good with breaks. Can we take a quick one?

19 MR. LUBEL: Yes, sir.

20 (Short recess from 11:06 to 11:11 a.m.)

21 EXAMINATION (Continuing)

22 BY MR. LUBEL:

23 Q. The determination of whether an individual's
24 benzene exposures causing disease had more to do with
25 the skin versus breathing is really an individual

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1 consideration, correct?

2 A. I think on an individual basis you will see
3 that percentage varying.

4 Q. But the point is that for you, as the
5 toxicologist, to form an opinion as to whether the more
6 damaging exposures came via the skin as opposed to
7 breathing it or vice versa, you really have to look at
8 that individual's exposures?

9 A. As a toxicologist, I would rely on someone else
10 to do that, an industrial hygienist or someone with
11 expertise in that.

12 Q. But for you to give an opinion about which
13 exposures were more important, you're going to have to
14 rely on somebody else to give you that information,
15 right?

16 A. The only thing that I would say is what the
17 published literature on the topic has suggested. And if
18 Mr. Oakley or anyone falls outside of any work
19 experience that anyone has ever published, I -- I can't
20 speak to that. So I -- so I am familiar with the
21 literature enough to see where that -- where the
22 relative percentages are and what people are saying
23 about dermal absorption. But the answer to your
24 question is, for him specifically, no, I can't.

25 Q. No, you can't what?

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1 A. I can't -- it's not my job. I don't have the
2 expertise to determine which was the most important for
3 him.

4 Q. Well, you can -- you can form an opinion based
5 upon which ones are most important for him as long as
6 you've got reliable information on the relative
7 exposures of the different pathways, correct?

8 A. Exposure assessment is a difficult thing. And
9 it's one that some toxicologists are very good at, but
10 I'm not one of them. So I -- I just don't do it.

11 Q. But you'd agree, there is some amount of
12 benzene exposures that somebody can have via the skin
13 that can cause AML?

14 A. In the absence of any other exposure?

15 Q. Right.

16 A. I've never seen that.

17 Q. So it can't happen?

18 A. There is no evidence that it ever has.

19 Q. Okay. And so it's your opinion that skin
20 exposures alone cannot lead to a benzene-induced
21 malignancy, correct?

22 A. Not in a human I've ever seen, no.

23 Q. I'm not asking whether you've ever seen it.
24 I'm saying from a -- from the standpoint of
25 toxicology -- that's your field, right?

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1 A. Yes.

2 Q. -- skin exposures from benzene cannot induce
3 malignancy.

4 A. No, I wouldn't say that. I mean, if we went
5 back to this hypothetical made-up situation where a guy
6 is not breathing -- he's breathing air out of a tube and
7 he's soaking his feet in benzene for 20 years, he may be
8 able to get enough dermal absorption to benzene to cause
9 a cytopenia and give him AML. I don't know the answer
10 to that.

11 Q. Well, what if the man has got both exposures,
12 via breathing it and skin, can that induce a malignancy?

13 A. I think if the exposure is sufficiently high.

14 Q. And so what -- what are the exposures that
15 the -- that the man or woman are going to need to be
16 exposed to from both skin and inhalation to lead to
17 malignancy?

18 A. Well, if you look at the best study that we
19 talked about earlier, the Pliofilm study, and what the
20 Pliofilm study says is that if you only conduct an
21 analysis on AML, you need something approximating 200
22 part per million years before you get a statistically
23 significant increased risk in leukemia.

24 That -- that '95 paper was based on Paxton,
25 which was based on sort of a combination of all three

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1 exposure assessments. There may be a little dermal
2 mixed in there, but that's mostly an inhalation
3 exposure.

4 If you also assume there were dermal
5 absorption or dermal exposure that was not accounted
6 for, then that's going to go up. And instead of being
7 200, it's going to 250 or 300, whatever you're going to
8 add because of the dermal.

9 Q. So --

10 A. It was 200 part per million years, purely
11 inhalation.

12 Q. What exposure is necessary, via the skin, to
13 cause a benzene-induced malignancy?

14 A. I -- I don't know the answer to that. I mean,
15 you could calculate -- you could take 200 part per
16 million years and you could calculate what an internal
17 dose would be, and then you could go backwards and
18 figure out what an internal dose would be through dermal
19 and you could make some comparisons.

20 Q. Have you done that?

21 A. I haven't, no.

22 Q. How would you do it?

23 A. Like I said, I mean, you -- in a part per
24 million year or a part per million, you know how much is
25 absorbed. About 50 percent of all inhaled benzene is

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1 absorbed. So a part per million is 3.2 milligrams per
2 meter cubed. You can figure out how much air the person
3 is breathing. You can do the math and calculate what
4 that would equate to in terms of an internal dose of
5 benzene. How much was taken in.

6 Then you could compare what person's dermal
7 absorption through the flux and all of this other math,
8 which I don't pretend to understand completely, and you
9 could calculate what that would equal in terms of an
10 internal dose and then you could compare them. And you
11 could say, Okay, here's how -- maybe it's 50 grams. I
12 don't know how much it would be. But some number of
13 benzene. And that's where you would have to get
14 dermally absorbed to equal that 200 part per million
15 year of the Pliofilm.

16 Q. Is this a formula you're referring to?

17 A. Well, it's just standard risk assessment
18 practice. It's not magic. I'm just not very good at
19 explaining it. You can definitely pretty easily
20 determine an internal dose, how much benzene got into a
21 person from inhalation.

22 Q. Can you draw this out for me?

23 A. Well, sure. I could try. I mean, again, I'm
24 not that good at exposure.

25 Q. Take your time.

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1 A. So look, 1 part per million exposure to benzene
2 is equal to 3.2 milligrams per meter cubed. That is a
3 part per million. That is a straight-across conversion.
4 All right? So if you were exposed to this 1 part per
5 million over an eight-hour-time-weighted average, then
6 you could figure out, Okay, that's fine -- if that's
7 true, how much do you breathe in an eight-hour period?
8 All right? Let's say you breathe 10 meters cubed. You
9 don't; I'm just throwing that out there. Your meters
10 cancel out. So it in that case, you would have 32
11 milligrams. So you would have been exposed to 32
12 milligrams of benzene breathing 1 part per million over
13 10 meters cubed for that day. All right? We think that
14 about 50 percent is absorbed. So you would have taken
15 in about 16 milligrams in that example. So it's not
16 hard.

17 Q. And then how would you convert that for the
18 skin?

19 A. Well, then you would do the same process with
20 skin absorption. You'd say how much is on there?
21 What's the percentage of benzene in the -- how it goes
22 through? And then calculate what an internal dose would
23 be from those absorptions.

24 Q. You could do this with -- with reliance on
25 others?

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1 A. I couldn't do it, if that's what you're asking.

2 Q. You could do it if people gave you reliable
3 information?

4 A. If they gave me -- did the math for me.

5 Q. You can do the math, can't you?

6 A. Not with much certainty.

7 Q. Who would you get to do the math?

8 A. Someone better at math than me.

9 Q. But you do recognize that it can be done?

10 A. I think you could calculate an internal dose,
11 sure.

12 Q. Via the skin or inhalation?

13 A. I think there's going to be some uncertainty
14 with the skin because there's -- there's all sorts --
15 there's uncertainty about the flux and whether or not
16 that's a legitimate -- you know, if we really know
17 enough about those, if the models that predicted those
18 have been validated. I think you can come up with an
19 answer, but it's going to have very wide confidence
20 intervals.

21 Q. How wide?

22 A. I don't know. It would be a pretty uncertain
23 answer.

24 Q. And you'd agree that Dr. Paustenbach would
25 admit that modeling benzene exposures for dermal

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1 absorption are not reliable, right?

2 A. I -- I do not agree to that. I don't -- I
3 don't know the answer to that.

4 Q. Well, hadn't he written about dermal exposure
5 modeling?

6 A. Well, he wrote a book chapter that I read a
7 while back that talked about the methodologies sort of
8 from the standard risk assessment process. But it had
9 some uncertainties that had not been validated. And my
10 understanding of John Spencer's report is that's why he
11 didn't consider it to be a legitimate thing to do,
12 because those models have not been completely validated.

13 Q. What I'm asking you is: Does Dr. Paustenbach
14 agree that it's not legitimate to model dermal exposures
15 to benzene?

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

17 Q. What did his book chapter say?

18 A. Well, his book chapter was giving instructions
19 how to go about doing it. So I would assume he would
20 think it's a legitimate step, given, you know, the
21 caveats that it's not validated and there's going to be
22 uncertainty.

23 Q. It's not perfect?

24 A. It may not be right.

25 Q. Okay. And so when we look at these

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1 epidemiological studies, how many do you think I could
2 pull out of your stack where the inhalation exposures
3 that they've assumed are based on uncertain information?

4 MR. GRAY: Objection; form.

5 MS. YATES: Objection; form.

6 A. Let me think about that one for a minute.

7 Q. (By Mr. Lubel) Every one of them, correct?

8 A. The exposure data and the epidemiology studies
9 is uncertain, absolutely.

10 Q. Every single one of those articles will have
11 some level of uncertainty as to the exposures even via
12 inhalation, correct?

13 A. I would say that's probably true with the --
14 well, you know, the animal data isn't over there
15 anymore. You've removed that from that pile. So taking
16 the animal data out of the pile, everything that you're
17 looking at there would be some uncertainty to it, yes.

18 Q. You're talking about the human data is
19 uncertain, correct?

20 A. The exposure part, you bet.

21 Q. Why is the animal data more certain than the
22 human data?

23 A. Because you put them in a cage. You know
24 exactly what you exposed them to. There is not any
25 guesswork.

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1 Q. Before we take the lunch break I'm going to try
2 to cover another topic. I'm sure you've testified on
3 this before. It's the how much question. Have you got
4 a set of literature on how much exposures are necessary
5 to benzene to induce AML? Have you got a series of
6 articles that you rely on?

7 A. Yeah, I think this binder -- the ones I
8 reference in here and then the binder Benzene and AML.
9 I assume you have these. If you don't have these we can
10 make copies, but this one I need to take with me.

11 I mean, this has -- in this binder I have
12 accumulated -- well, let's -- depends on the study. If
13 you're talking about Pliofilm, that was one study that
14 is in here and that's Rinsky '87. But there's been ten
15 other studies of that cohort that are going to be in its
16 own binder. Same with Health Watch. Same with the NCI
17 China. So there will be one or two representatives in
18 this binder of that big collection of literature. And
19 that's why I brought these. So that literature is
20 germane to the question. Sometimes that's where they
21 did the quantitative analysis; sometimes that's where
22 they did the histopathology. It just depends. It
23 wasn't in the original study.

24 Q. Let's start with --

25 A. But this is the studies that have quantitated

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1 or tried to quantitate the risk of AML and benzene.

2 Q. Based upon these uncertain levels of inhalation
3 exposures?

4 A. Well, I think there's always going to be some
5 uncertainty.

6 Q. We can agree on that, right?

7 A. Yes.

8 Q. The data is just not as precise as we'd like?

9 A. There is uncertainty.

10 Q. And especially when you're looking back and
11 doing retrospective analysis, it's harder to go back in
12 time and to re-create what happened in the past,
13 correct?

14 A. That's true.

15 Q. And for that reason, you'll have less precision
16 than if you start here and you go forward in time,
17 right? A prospective analysis?

18 A. Not -- not necessarily, but -- but in general,
19 I think that's probably true. Many studies that are
20 being conducted now, they are going forward in time, but
21 they're still not collecting air monitoring data.
22 They're still asking people, What were you exposed to?

23 Q. Why would they do that? Save money?

24 A. We'll have to ask the epidemiology world why
25 they do that. It's cheaper. It's quicker.

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1 Q. That's not they way you prefer to do it.

2 A. It makes it less useful.

3 Q. Isn't that what their --

4 A. If you don't have quantitative data, it makes
5 the findings less useful.

6 Q. Aren't we trying to get information?

7 A. Yes.

8 Q. Have you told them, Guys, ladies, give me some
9 quantitative information?

10 A. When they've asked --

11 Q. Okay.

12 A. -- I have told them that.

13 Q. Okay. What's the range, when you look at the
14 literature, on the low end, starting with the low end,
15 of exposures to benzene that can induce AML?

16 A. That have been statistically significantly
17 associated with an increased risk?

18 Q. Start there.

19 MR. GRAY: Objection to form of that
20 question.

21 MS. YATES: Same objection.

22 A. You don't -- you know, there are broad ranges.
23 So the Glass study suggests that in something greater
24 than 8.

25 Q. 8 what?

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1 A. Part per million years. It goes up to 60 part
2 per million years is what they reported. Guenel.

3 Q. Did you say "60" or "16"?

4 A. From 8 to 60.

5 Q. Okay. I just wanted to make sure I heard.

6 A. Yeah. And Guenel, I think, is something
7 greater than 17 part per million years up to infinity.

8 In the Pliofilm cohort it had to be greater
9 than 200 part per million years.

10 In the NCI, they saw a quirky dose
11 response, but they saw an increase in MDS and ANLL at 40
12 to 90 or 40 to 100.

13 So the range is somewhere greater than 50
14 to about 200 in general.

15 Q. Well, the range that you just gave us is -- is
16 8 ppm years to 200 ppm years.

17 MR. GRAY: Objection; form.

18 MS. YATES: Same objection.

19 A. I would disagree with that. I don't think the
20 Glass study supports that it's 8. The next level down,
21 the 7.9, the relative risk of AML is 0.5. So if you
22 take that literally and ignore the confidence intervals,
23 7.9 would protect you from getting AML. So I think it's
24 pretty hard to justify that it's going to be 8 or even
25 8.1.

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1 Q. (By Mr. Lubel) But the article reports 8?

2 A. No. The article reports greater than 8.

3 Q. I'm sorry. The Glass article, that's
4 peer-reviewed and published, correct?

5 A. Yes.

6 Q. Reports a statistically significant increase of
7 AML from benzene exposures at greater than 8 part per
8 million years, correct?

9 A. Yes.

10 Q. And you -- and you made some reference to 8 ppm
11 years to 60 ppm years. Can you elaborate on that?

12 A. That -- because it's a wide exposure range, you
13 don't -- you don't know where they are and she doesn't
14 either. What she said was, in her estimation, no one in
15 that cohort had greater than 60 part per million years
16 exposure. Now, there is a lot of debate as to whether
17 or not it was done correctly and if she missed people,
18 but -- so the range, in her study, is going to be
19 somewhere between -- in that study -- between 8 and 60.

20 Q. Is this study flawed?

21 A. Umm, there was quite a bit -- well, I think any
22 epidemiology study or any science study is going to have
23 problems.

24 Q. There's no perfect epi study, is there?

25 A. Not to my way of thinking.

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1 Q. Is Glass more flawed than you think it should
2 be?

3 A. Glass has some issues with the exposure
4 assessment, and she was concerned about it. Other
5 investigators who have done comparable analyses that she
6 did, they got different results. They were concerned
7 about it. Either they missed something or she missed
8 something. And so there has been a collaborative group
9 to try to figure out why her exposures were lower than
10 what they saw in the Canadian workers, in the UK
11 workers, and in other petroleum workers.

12 Q. What cohort were they studying in Glass? Do
13 you remember?

14 A. Well, it was the Australian Petroleum
15 Institute. So there were all kinds of different workers
16 in there: Distribution, transport, marketing, refinery
17 workers.

18 Q. Industrial workers?

19 A. Yes.

20 Q. Were any of those workers, as you recall it,
21 exposed to gasoline?

22 A. I think for sure there were some gasoline
23 exposures. In fact, she said at one point in her paper
24 that she saw a higher risk when people were exposed to
25 concentrated forms of benzene as opposed to more dilute

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1 forms of benzene, such as in gasoline, even if the -- if
2 the exposures were the same.

3 Meaning, 10 part per million from benzene
4 and 10 part per million from gasoline, you saw a higher
5 risk in the benzene than you did if it's in a mixture
6 like gasoline.

7 Q. And what's the scientific basis for that?

8 A. Well, my understanding of the scientific basis
9 and my interpretation of that data is that you could
10 have competitive inhibition in the metabolism with other
11 chemicals present in the gasoline.

12 Q. And is that what she concluded?

13 A. She's not a toxicologist. She didn't make any
14 statements about it at all.

15 Q. So is it your opinion that the benzene from --
16 from the carrier gasoline can't lead to a
17 benzene-induced malignancy?

18 A. I've not seen definitive evidence that gasoline
19 can give a person enough benzene exposure for them to
20 get AML.

21 Q. But assuming the exposure is high enough.

22 A. If the exposure is high enough and you've
23 overcome this competitive inhibition, then you start
24 pumping up against, well, how much toluene can a person
25 be exposed to before that kills them? And I don't know

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1 whether you can get enough benzene exposure from
2 gasoline to give them AML or they're going to die from
3 the hexane exposures or something else. I don't know
4 the answer to that.

5 Q. You just don't have an opinion on that right
6 now?

7 A. As to whether or not it's possible?

8 Q. Whether a benzene-induced malignancy can occur
9 from high enough exposures to gasoline.

10 A. My -- my opinion on that is based on the
11 scientific literature, and that does not support that it
12 happens.

13 Q. Can't happen?

14 A. That it does happen. I can't say that it
15 can't. Anything can happen, I guess. But there -- the
16 scientific literature taken collectively does not
17 support an increased risk in AML in gasoline workers.

18 Q. So you're saying in the Glass study, none of
19 the -- of the AMLs that were studied were exposed to
20 gasoline?

21 A. They might have been exposed to gasoline. I
22 don't know what they did on a daily -- we're all exposed
23 to gasoline. But whether they did that in the workplace
24 or not, she doesn't break out exactly where all -- well,
25 she does. She breaks out where the AMLs were and some

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1 of them were in distribution. So that might have been
2 carrying gasoline. But some of them also were carrying
3 BTEX. She has some people in her cohort that she
4 describes that would have had very high exposures to
5 pure benzene. That could have underlie her AML risk.

6 Q. Did she conclude one way or the other as to any
7 of the AMLs that were exposed to gasoline, whether those
8 were causally related or not?

9 A. She didn't make -- she did not conclude that,
10 no.

11 Q. She didn't -- she didn't conclude either way?

12 A. She did not make that conclusion, either way.

13 Q. Were any of the AMLs in the cohort in the -- in
14 the gas -- the primarily gasoline-exposed occupations,
15 so to speak?

16 A. I think that data is available and I don't
17 remember it right off.

18 Q. Now, Guenel, you said, was greater than 17 part
19 per million?

20 A. 16.8 I think is how they broke it out. Don't
21 ask me why.

22 Q. What were they studying in Guenel?

23 A. Give me a minute and I'll tell you.

24 Q. Tell me the title of that article. I just
25 forgot it.

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1 A. Leukemia in Relation to Occupational Exposures
2 to Benzene and Other Agents: A Case Control Study
3 Nested in a Cohort of Gas and Electric Utility Workers.
4 It's from 2002.

5 Q. Is she -- is Guenel the lead author?

6 A. Uh-huh. Pascal Guenel.

7 Q. Okay.

8 A. G-u-e-n-e-l. I know that's in my report.

9 Q. Can I look over it real quick? I'll move away
10 from you as soon as I look at it.

11 A. You can take it if you'd like.

12 Q. Were these exposed workers exposed to pure
13 benzene or -- or benzene-containing products?

14 A. Gas and electric utility workers. I don't
15 know. She -- she lists -- he lists it as benzene, but
16 I'd have to just go through and read it in detail to
17 see. I would guess that it's benzene-containing
18 products just based on the title, but I don't know that.

19 The only -- the other issue with this
20 paper, which is an important issue, is that this is
21 acute leukemia. So you would have to take those numbers
22 with a grain of salt.

23 Q. This was in your AML stack, was it not?

24 A. Yes.

25 Q. It's one of the articles you've relied upon?

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1 A. Uh-huh, yes. But I don't know how much it has
2 to bear specifically with regard to the amount of
3 benzene to give a person AML, because they don't break
4 out AML in that cohort.

5 Q. You're saying because they looked at the
6 broader group of acute leukemia?

7 A. Right. If you take out the acute lymphocytic
8 leukemias, then I don't know what that would do to their
9 exposure numbers, what that would do to their risk.

10 Q. The odds ratio is 3.6, correct?

11 A. I -- I don't --

12 Q. (Indicating.)

13 A. Greater than 16.8. It was 3.6, yes.

14 Q. What does that mean?

15 A. 3.6 means that for an odds ratio, that they
16 observed 3.6 times more than they expected with the
17 caveat that the real answer lies somewhere between their
18 confidence intervals.

19 Q. Of... (Pause.)

20 A. 1.1 to 11.7. And because it's greater than
21 1.1, that -- because it's greater than 1, then it would
22 be statistically significant.

23 Q. So you could actually have odds ratios as high
24 as 11.7?

25 A. I think when they -- when they did the

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1 calculation that was one of the possibilities.

2 Q. In other words, 11 times what was expected from
3 a nonexposed?

4 A. Or 1.1 times.

5 Q. And so how do they calculate an odds ratio of
6 3.6? Do you know? Is it somewhere in between this
7 confidence interval that they calculate the odds ratio?

8 A. Yes, it's somewhere between. And some
9 epidemiologists would say that's their best estimate.
10 There's variability about the number. There's a number
11 that they observed based on diagnostic criteria, et
12 cetera. There's variability in the control population
13 they compared it to. So that's why you have that
14 confidence interval.

15 When you see a confidence interval that
16 wide -- and that's not as wide as some -- but 1 to 11,
17 that means that the numbers were very small and that
18 they were pretty uncertain as to that analysis.

19 Q. So you're saying you'd rather see a tighter
20 confidence interval if you can get one?

21 A. Yes.

22 Q. In other words, 1.1 to 4 is better than 1.1 to
23 11?

24 A. It would certainly indicate that they had a
25 more stable database.

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1 Q. Does it typically mean that you have more
2 people to study?

3 A. I think as a general rule, that's true. The
4 larger your study, the more stable it's going to be.

5 Q. If you look at Table 6 it actually does a
6 breakdown for acute myeloid leukemia. That's AML,
7 right?

8 A. Uh-huh. (Witness answers affirmatively.)

9 Q. And assume with me that it breaks it down by
10 the level of exposures and gives you the number of
11 cases, the number of controls. I'm going to hand it
12 back to you.

13 A. Okay.

14 Q. It shows five cases, nine controls, with an
15 odds ratio of 2.4, but it reflects a 95 percent
16 confidence interval of 0.7 to 8.5. And that confidence
17 interval would indicate to you that it's not
18 statistically significant, correct?

19 A. Absolutely. It would not be statistically
20 significant.

21 Q. It's not?

22 A. Right.

23 Q. But that range of confidence interval, the 0.7
24 to 8.5, would be suggestive of an association, correct?

25 MR. GRAY: Objection; form.

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1 A. Yeah, I don't think you can say that. I mean,
2 the -- what the confidence interval tells you is that --
3 that we are 95 percent sure. And even with the
4 confidence interval that's greater than 1, so now you've
5 moved it up here, we're 95 -- we don't know what the
6 right answer is. No one does. But we're 95 percent
7 sure that the right answer is somewhere in here. We
8 think it's 3.6, but it may not be 3.6. Maybe it's 11.7.
9 Maybe it's 1.1. We're 95 percent sure. So within this
10 range, if you asked the question 20 times, 19 times it
11 would show up somewhere in this range. One time it
12 might be below or it might be higher. You don't know.

13 If this is below 1 and you have the end of
14 your confidence interval going below 1, you just don't
15 know. You know, it's not suggestive of an association.
16 It's not suggestive of a not association. It just tells
17 you that it was insufficient to answer the question.

18 Q. (By Mr. Lubel) Well, if your upper range of
19 your confidence interval is below 1, too -- is also
20 below 1?

21 A. Yes.

22 Q. Then it does answer the question?

23 A. If it's below 1 in the upper end, then it would
24 be statistically significantly deeper -- decreased and
25 you would make the same argument. We're 95 percent sure

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1 that every time -- we're 95 percent sure when we ask
2 this question it will be negative. 5 percent of the
3 time it might be positive.

4 Q. Do you know whether or not epidemiologists
5 consider their confidence interval -- the 95 percent
6 confidence interval, such as the one we see in that
7 table, to be suggestive of an association?

8 MR. GRAY: Objection; form.

9 A. I think that's going to vary widely depending
10 on what epidemiologist you ask.

11 Q. (By Mr. Lubel) You've heard that before?

12 A. Well, this is a debate, argument, that goes on
13 in all kinds of circles, not just this one.

14 MR. LUBEL: Is this a good time for a
15 break?

16 THE WITNESS: Sure.

17 (Lunch recess from 11:41 to 12:32 p.m.)

18 EXAMINATION (Continuing)

19 BY MR. LUBEL:

20 Q. We had been talking about -- I wrote it down --
21 four studies addressing this range of exposures related
22 to AML. And we've talked about the Glass article, the
23 Guenel article, and then you referenced Pliofilm and
24 NCI. And we had not gotten to Pliofilm or NCI. Is that
25 your recollection?

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1 A. That sounds right, yes.

2 Q. Let's move to the Pliofilm article, if you
3 would pull that for us.

4 A. Actually, there are several.

5 Q. Well, the one that -- are you -- are you
6 relying on all of them?

7 A. Well, two for sure that are quantitative. Bob
8 Rinsky conducted a qualitative risk assessment and
9 calculated the exposures.

10 Q. Okay.

11 A. But in -- in there -- well, let's find it. I
12 mean, this is kind of the -- probably the single most
13 important paper that's out there. At least it's
14 referenced the most.

15 And he finds a statistically significant
16 increase at 200 to 400 part per million years. There's
17 not one from 40 to 200. So that's --

18 Q. Can I look at it real quick?

19 A. You bet. And then what he and everyone else
20 did is included all of the leukemias together and then
21 Otto Wong redid the analysis, where he only included
22 defined, confirmed cases of AML. And in that analysis,
23 he found a very clear threshold and there was no
24 elevated risk below 200 part per million years.

25 So that is probably the single best

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1 analysis to answer the question how much benzene does it
2 take to give a person AML.

3 Q. All right. Now, the Rinsky article you're
4 referring to is titled: Benzene and Leukemia. And it's
5 April of 1987, correct?

6 A. Yes. In the New England Journal of Medicine,
7 yes.

8 Q. You were referring to Table 2, Observed and
9 Expected Deaths From Leukemia?

10 A. Yeah.

11 Q. Do you remember how many cases there were of
12 AML?

13 A. I think it's on that table. I don't. I can
14 tell you. So that's what this ratio is. So from the 0
15 to 0001 to 0.1 there were two cases. He expected 1.83
16 in this 40 to 200. There were two cases. He expected
17 0.62, here there were --

18 Q. Can I see that real quick before you move on?

19 A. -- 24 cases or -- yeah, I think that's what
20 that is. Total maybe 11, 12, and then in the updates in
21 the -- in the Silver paper, I think they found three or
22 four more. So you might be up to 15.

23 Q. Let me do it this way: In the top of Table 2
24 gives you the exposures and the ppm years, correct?

25 A. Yes.

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1 Q. And the second column is 40 ppm years to 200
2 ppm years?

3 A. Right.

4 Q. And they found two -- is that leukemias or
5 AMLs?

6 A. This is leukemias.

7 Q. Two leukemias, but expected 0.62, right?

8 A. Right. Based on the number of people and using
9 external control population.

10 Q. So you had more than a doubling of the risk,
11 relative risk, or is this an odds ratio?

12 A. This is SMR.

13 Q. So this is mortality, deaths, right?

14 A. Yes.

15 Q. So we refer to that as OR, odds ratio?

16 A. No.

17 Q. SMR?

18 A. SMR, Standardized Mortality Ratio.

19 Q. But we saw more than a doubling of the risk,
20 correct?

21 A. Right. Standardized mortality ratio, SMR, and
22 then the confidence interval will go down to 0.36.

23 Q. So it's not statistically significant, correct?

24 A. No, right. Correct.

25 Q. What was the power calculation?

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1 A. You mean whether or not it could detect?

2 Q. Yes. Exactly.

3 A. I don't know.

4 Q. Does he report it?

5 A. No. No. I think others might have gone there.

6 I don't know the answer to that.

7 Q. Why don't they do it?

8 A. A lot of them do.

9 Q. A lot of them don't, too.

10 A. Right. I don't know.

11 Q. Does it help you?

12 A. Well, I mean, I think if you read enough
13 epidemiological studies and you're in this business very
14 long, I think that's almost a given that studies have
15 limited capacity to detect small changes. So when you
16 see something where the relative risk, however you
17 define that relative risk, of 1.2 or -- I mean an odds
18 ratio or SMR, whatever, relative risk of 1.2 and there
19 is a 20-percent increase, they're just not accurate
20 enough to really rely on that, in my view.

21 Q. Well, here's -- here's a broader question I'm
22 trying to find out.

23 If you, as a scientist, are really trying
24 to see if the relative risk is doubling or better --
25 okay? If the study was designed so that it would not

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1 detect a relative risk of less than 50, what use is that
2 study to you? Do you see where I'm going with that?

3 A. I do. And there is some truth to that. The
4 Pliofilm would not fall into that category. And I'm
5 basing that on the fact that it is probably the most
6 analyzed cohort on the planet. And we base all of our
7 regulatory and public health decisions on it. In those
8 lower categories they were looking at a lot of -- a lot
9 of workers. It just was not a statistically significant
10 finding.

11 Q. Okay. Do we know why?

12 A. Why it was not statistically significant?

13 Q. Yes, sir.

14 A. Because it may not have been truly elevated.

15 Q. Or... (Pause.)

16 A. Or it was elevated and there wasn't enough
17 people in that group to find a statistical association.

18 Q. Is leukemia considered a rare disease?

19 A. Depends on which kind of leukemia.

20 Q. AML?

21 A. I don't know. I mean, I think it's rare
22 compared to breast cancer or lung cancer, but rare is
23 kind of a relative term. There are 12 or 13,000 new
24 cases of AML in the U.S. every year. A lot of people
25 know people that have AML or it's touched a lot of

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1 people's lives. It's not totally unheard of.

2 Q. I'm talking about from an epidemiological
3 standpoint, is it rare such that the study needs to be
4 designed carefully if it's going to try and detect a
5 statistically significant doubling of the risk or
6 better?

7 A. I think epidemiologists should always pay
8 attention to the potential power of their study. But
9 that is an epidemiology question, and I'm not really
10 qualified to -- to address it.

11 Q. But you're generally familiar with the
12 importance behind the power calculation?

13 A. I'm familiar with what it does.

14 Q. And -- have I mixed all these up?

15 A. I don't think so.

16 Q. For two reasons --

17 A. There might be two copies. No, it's all the
18 same.

19 Q. All right.

20 A. And then the other one is Otto Wong's right
21 there (indicating).

22 Q. All right.

23 A. Are you giving this one back to me?

24 Q. Yes, sir. That's the Rinsky article.

25 A. Yes. The first one -- well, one of the first

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1 ones.

2 Q. Did any of the Pliofilm studies find a risk
3 below 200 ppm years?

4 MR. GRAY: Objection; form.

5 A. What do you mean, a risk? I mean, they
6 calculated the risk for every dose. Are you talking
7 about a statistically significant increased risk or just
8 a risk?

9 Q. (By Mr. Lubel) Well, let's start with a risk.

10 A. Well, yeah, they calculate -- I mean, the risk
11 was not elevated, but they calculated the standard
12 mortality rate. Are you talking about a relative risk?
13 That's just a ratio of what you saw versus what you
14 would expect. You can express that as an odds ratio or
15 relative risk, SMR, SIR. There's not mortality. They
16 calculated the risk for all of these doses.

17 For 001 to 0.1 -- or up to 40 part per
18 million year, the relative standard -- the standardized
19 mortality ratio was 109. So they calculated a risk
20 below 200.

21 Q. Did -- did any -- there's a number of different
22 authors that have written about the Pliofilm cohort on
23 benzene, right?

24 A. Paxton, yes, several.

25 Q. Have any of them found a statistically

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1 significant risk at or above 40 ppm years?

2 A. At or above?

3 Q. Right.

4 A. I think they've all found a statistically
5 significant risk at or above 40 part per million years.

6 Q. Okay. At 40 ppm years or at 50 or --

7 A. Well, you said "at or above."

8 Q. I mean -- I'm trying to be more specific now.

9 A. Okay.

10 Q. Where is that lower range in the
11 Pliofilm-specific studies?

12 A. There's not an increase at 40.

13 Q. 45?

14 A. Paxton took all of the various exposure
15 assessments and kind of lumped them together and went to
16 a slightly different bent and it was 0 to 5, 5 to 50,
17 and 50 to 500. And there was a statistically
18 significant risk in some of the exposure assessments in
19 the 50 to 500. That's -- if you'd like to look at that
20 paper, it's right here.

21 Q. The exposures in the Pliofilm studies, were
22 they quantitative in the sense that they were actual
23 measured exposures or was there judgment involved in
24 analyzing data?

25 A. I think there was judgment involved.

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1 Q. Was there any modeling done?

2 A. I don't -- I would assume there was, yes.

3 There was also quite a bit of air monitoring data that
4 they tried to correlate.

5 Q. Actually, from the process or from some other
6 similar process?

7 A. No, from the -- I mean, it passed -- I should
8 know the answer to this because I've looked at that
9 data. Maybe pre-1945 they had no air data -- or might
10 have been pre-1950. But after that point, they did have
11 air data.

12 Q. Did they have air data such that they didn't
13 need to make any judgments after whatever that time
14 period is, '50 or '55?

15 A. I don't know how they -- they parsed that out.

16 Q. But your recollection is that they had
17 sufficient documented records of the exposures for the
18 employees to -- to express it in quantitative terms as
19 opposed to modeling?

20 A. That's my recollection, yes. And they've
21 definitely done modeling, too. What Williams did in
22 2003, she used Monte Carlo techniques for a variety of
23 the various exposure input parameters.

24 Q. Do you know who was paying Otto Wong for his
25 work?

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1 A. I don't.

2 Q. He didn't disclose that, did he?

3 A. I --

4 MR. GRAY: Objection; form.

5 MS. YATES: Same objection.

6 A. I don't know. He didn't on the paper that I
7 remember seeing.

8 Q. (By Mr. Lubel) I take it that -- that you
9 didn't know that Otto Wong had been doing work for the
10 American Petroleum Institute and the Chemical
11 Manufacturing Association.

12 A. Well, I didn't say that.

13 Q. You knew that, didn't you?

14 A. Yeah.

15 Q. And that's generally known, now, isn't it?

16 A. Otto does work for the Petroleum Institute,
17 sure.

18 Q. But did you recall that he was specifically
19 doing work for companies that cared about the results
20 when he wrote this article?

21 A. That, I don't know. In 1995 I was a graduate
22 student, so I don't know who he was being paid by.

23 Q. Okay. So we got Paxton next?

24 A. Sure.

25 Q. Is that right?

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1 A. Well, I mean, Paxton is just sort of a
2 rehashing of all of this same data.

3 Q. That's what I'm trying to figure out. Why are
4 all of these different authors writing on the same data?

5 A. Well, because it's not absolutely certain what
6 the exposures were. And Kenny Crump did it, Dennis
7 Paustenbach did it in 1992, and Bob Rinsky did it, and
8 there is a lot of variability in how they approached the
9 process.

10 Q. And didn't all of them come up with different
11 exposure numbers?

12 A. Uh-huh. (Witness answers affirmatively.)

13 Q. Is that a "yes"?

14 A. Yes. They came up with different exposure
15 numbers. I mean, they were -- they were not as far off
16 as you might imagine. There was some continuity there.
17 But what EPA said was they were all equally valid. EPA
18 couldn't really decide which was the most appropriate,
19 so when they established their cancer potency factor,
20 based on this study, they expressed it as a range. And
21 that it's somewhere between here and here, based on
22 Crump and Allen and Rinsky.

23 Q. So they don't use Paxton's work?

24 A. Well, Paxton didn't -- what Paxton did was just
25 re -- she didn't do a new exposure assessment.

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1 Q. Do they -- did they -- did they use Wong's
2 work?

3 A. No.

4 Q. Why not?

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

6 Q. Probably because he was bought and paid for by
7 the industry?

8 A. I would think that's not the case.

9 MS. YATES: Objection; form.

10 Q. (By Mr. Lubel) Well, did they say why?

11 A. They never -- it wasn't addressed.

12 There has been quite a bit of discussion
13 within the agency and within IARC as to whether or not
14 they should redo this and they should pay attention to
15 the fact that in the Pliofilm and in the original they
16 were using all of the leukemias that were present. So
17 there is some discussion about that. But they haven't
18 acted on it, yes.

19 Q. Are you talking about a subtype analysis?

20 A. Correct. What Otto did -- what Dr. Wong did,
21 which was different than anybody else, was went back in
22 and analyzed for just the AML cases.

23 Q. Did he -- did he analyze for the other
24 leukemias?

25 A. No.

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1 Q. Why not?

2 A. You'd have to ask -- ask -- ask him that. I
3 don't know. I think because it's generally accepted
4 that AML is the one that is linked to benzene, so that
5 was the one that he would have been the most concerned
6 about.

7 Q. Did he do an analysis of the subtypes of AMLs?

8 A. In this particular paper?

9 Q. Right.

10 A. No.

11 Q. Why not?

12 A. I think based on what I just told you. There
13 weren't very many.

14 Q. Weren't very many what?

15 A. Weren't very many cases that weren't AML, so he
16 didn't have to take out -- I think he took out three.

17 Q. Do you --

18 A. So I mean, I don't know what you would do with,
19 you know, one CML and a couple that haven't been
20 diagnosed. That's too few to conduct any kind of
21 analysis on.

22 Q. What subtypes of AML are causally related to
23 benzene?

24 A. Subtypes of AML?

25 Q. Yes, sir.

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1 A. The only one that I think you could make a
2 credible argument that would not be linked to benzene is
3 an M3 or acute promyelocytic leukemia.

4 Q. You've written on that?

5 A. I think I've include it. I've included it in
6 that book chapter. And there I think you can make that
7 argument because of the cytogenic changes have never
8 been reported. But more to the point, it's a unique
9 enough subtype of AML where people have done
10 epidemiology on it as an independent disease; whereas,
11 for an M2 or an M4, no one -- no one's done that.

12 Q. So at this point, based upon the information we
13 have, are you of the opinion that all of the subtypes of
14 AML, except for possibly M3, can be causally related to
15 benzene exposures?

16 A. Yes.

17 Q. The Paxton article from Pliofilm is dated 1994,
18 correct?

19 A. Yes.

20 Q. I'll give you that back.

21 A. Okay.

22 Q. Do you have the Infante article?

23 A. From '78?

24 Q. Right.

25 A. Probably.

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1 Q. Do you rely on that one as well?

2 A. Well, he didn't do much quantitative -- really,
3 anything quantitative in his paper. So, no, not from a
4 quantitative point of view. I mean, he gets credit for
5 being the first person to step up and say this might be
6 an issue. I think even here's an earlier one from '77,
7 his Lancet paper.

8 Q. Is it different than the one you handed me?

9 A. Yeah, I think it's -- I don't know. Is it?
10 That might be a book chapter, the one that I just gave
11 you.

12 Q. Why do some individuals succumb to a
13 benzene-induced malignancy at different exposures than
14 others? Do we have an answer to that?

15 A. Well, I mean, I think the -- the short answer
16 is there's going to be genetic susceptibility issues
17 with -- with individuals. That's the reason why you
18 always have such a wide dose response any time you use a
19 human population for a study. Which kind of goes back
20 to what we talked about earlier, why animals will
21 sometimes give you a cleaner answer than -- than humans
22 will.

23 The exact polymorphisms that can lead to
24 susceptibility are -- are starting to be identified, but
25 we don't know precisely what -- what role they play and

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1 how big of a role they play, but we're getting there.

2 Q. Can you draw the dose response curve for us for
3 benzene?

4 A. For benzene and AML?

5 Q. Benzene, period.

6 MR. GRAY: Objection; form.

7 MS. YATES: Objection.

8 A. I don't know how you draw a dose response if
9 you don't have -- what's your endpoint?

10 Q. (By Mr. Lubel) Just disease.

11 A. Which disease?

12 Q. Any of them.

13 A. Well, I mean, if we're talking about aplastic
14 anemia, I think that dose response might look pretty
15 different than if we're talking about a cytopenia or
16 AML.

17 Q. How so?

18 A. I think it only really happens at really,
19 really high doses. I think it --

20 Q. How high?

21 A. 600 part per million years, 1,000 part per
22 million years, high dose. That's a high dose
23 phenomenon.

24 Q. Higher than leukemia?

25 A. Yes.

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1 Q. Higher than cytopenias?

2 A. Yes.

3 Q. Can you draw the dose response curve for us for
4 AML? I've asked Irons to do it and he says it can't be
5 done. That's why I'm asking you.

6 A. It would be -- there would be a lot of
7 uncertainty, but I think it would go up with increasing
8 doses and then at some point it will likely plateau out.

9 Q. Well, I think he'd agree with that. But can
10 you draw it?

11 A. Well, no -- no more specifically than that.

12 Q. Can anybody?

13 A. Can they draw a -- well, I guess they --
14 anybody could draw it.

15 Q. Would it be accurate? That's what I mean.
16 Draw it accurately.

17 A. That, I don't know the answer to.

18 Q. Do you imagine there will be sometime in the
19 future that we'll have enough information to do it?

20 A. Well, there's a lot -- I mean, benzene is
21 something that people are continually interested in.
22 And there are new studies coming out, so I think,
23 perhaps, that we will get a better understanding of
24 mostly the low dose effects, not the high dose effects,
25 because people aren't really being exposed to those.

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1 Q. Were you aware from your limited participation
2 in the Shanghai studies with Dr. Irons that the primary
3 reason that they were conducting those studies in
4 industry was -- over in China was to try and better
5 define the low dose threshold, if there was one?

6 A. I don't remember that being a specific research
7 aim, but I think that is something that would likely
8 come from that study.

9 Q. Why?

10 A. Well, because that's what they do when they're
11 evaluating risk of various malignancies associated with
12 air concentrations. You know, I think -- in my view,
13 anyway, the -- the existence of a threshold is -- is
14 pretty clear. It just makes sense on every level that
15 I've analyzed it on. Where exactly that threshold might
16 lie is -- is open to discussion.

17 Q. There is no generally-accepted answer to that
18 right now, is there?

19 A. Well, I guess it depends on what you mean by
20 generally accepted. I think my answer of somewhere
21 between 40, 50, and 200, with the best estimate being
22 about 200, I think that is a fair representation of the
23 data.

24 Q. I'm saying there is no scientific consensus on
25 that point.

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1 A. I would say that's probably true.

2 Q. And possibly, additional studies going forward
3 may lead us to general acceptance on -- on where that
4 threshold is, correct?

5 A. The more data you have, the better. Yes.

6 Q. And so for that reason, you actually support
7 the work that Dr. Irons and industry is doing over in
8 China to get more information?

9 MR. GRAY: Objection; form.

10 MS. YATES: Objection; form.

11 Q. (By Mr. Lubel) True?

12 A. I don't know what you mean by "support." Think
13 it's a good idea?

14 Q. Yeah, that's what I mean.

15 A. Well, I had some reservations about some
16 aspects of it, but I think the more data the better.
17 And there are other things going on besides -- that's
18 not the only ongoing benzene study in the world. And I
19 think all of them, when they get published, I will look
20 at them just like you will look at them and the rest of
21 the scientific community will look at them and we'll
22 evaluate how it fits in with what we know.

23 Q. What other ongoing benzene studies are there?

24 A. Well, NCI has got things going on in China.
25 There's at least one study in Brazil that I know of, a

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1 researcher named Rego. They are doing some things in
2 Germany. Rob Snyder is doing some in vitro work.
3 Martin Smith continually does in vitro work. David
4 Eastman is doing in vitro work. Neal Obernoff is
5 doing -- I mean, there's -- there's API in conjunction
6 with Australia and the UK are doing a pooled analysis of
7 the case control study. You know, there's -- those are
8 just the ones I'm aware of.

9 Q. The Rego work in Brazil --

10 A. Uh-huh. (Witness answers affirmatively.)

11 Q. -- who's funding that?

12 A. I have no idea. My -- I've talked to Dr. Rego.
13 He was -- what he approached me on was whether or not
14 they should include non-Hodgkin's lymphoma as a
15 compensatory illness.

16 Q. In a worker's compensation scenario?

17 A. Right. Whether or not they should consider NHL
18 as a benzene-induced malignancy.

19 Q. What did you tell them?

20 A. I told them in my view they should not. And
21 then we argued about the literature. I sent him some.
22 He sent me some. And that was the end of it.

23 Q. You-all never reached an agreement on that?

24 A. No. No.

25 Q. Is he a knowledgeable expert?

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1 A. I -- I don't know who he is. I mean, he's
2 published several papers. I've seen two or three papers
3 that have come out of his lab.

4 Q. On benzene?

5 A. Yeah. Well, he's more of an organic solvent
6 kind of guy. Most of his epi studies are not focused on
7 benzene, but focused on workers exposed to all kinds of
8 organic solvents.

9 Q. Of which benzene could be one?

10 A. One. And that was part of the discussion that
11 we had about his own work and whether or not it fit in
12 with this question.

13 Q. So --

14 A. He was a very nice guy. Don't get me wrong.
15 When I say "argue," that probably wasn't the most
16 appropriate word.

17 Q. You had a professional dispute?

18 A. Yeah, we just discussed the literature.

19 Q. Don't worry. Tim and I have those every day.

20 A. I can believe that.

21 Q. But Dr. Rego, he believed that the evidence was
22 sufficient to link benzene to NHL, correct?

23 A. He thought, at least at that point -- I don't
24 know what their final decision was -- but at that point,
25 based on whatever their bar was for the Brazil working

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1 comp, that it was significant to -- to go over that bar.
2 I -- I think that was where he was going.

3 Q. Are you involved in any ongoing benzene studies
4 right now?

5 A. Studies? Like, real --

6 Q. Now, you got me nervous because --

7 A. Well, I mean, there's lots of --

8 Q. Because you haven't been parsing words all day
9 and now, What's a study? Tell me what you think --

10 A. Yeah, I'll tell you what I'm doing and you can
11 decide whether it's a study or not.

12 We have been involved in reanalyzing the
13 Plioform data back to the Williams and Paustenbach
14 exposure estimates because that, at this point, has not
15 been incorporated into a risk assessment. Everyone
16 likes it; everyone agrees that it's good data, EPA
17 included, but it's never been -- you know, is that --
18 are the exposures in Paustenbach and -- Williams and
19 Paustenbach, 2003, is it going to change the -- the
20 slope factor for benzene? My guess is it's not, but it
21 hasn't been taken to that next step and incorporated
22 into an epidemiological analysis. So we are involved in
23 that. We were the first -- we put it into the form
24 where you could analyze it. And now we've been asked to
25 submit a proposal where we're going to analyze it. So

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1 that -- if that's a benzene study, then, yes, I'm
2 involved in a benzene study.

3 Q. Who are you doing that work with?

4 A. I'm doing it with Lisa Alyward, who is a
5 partner in my firm, the three of us. David Garabrant at
6 the University of Michigan will be the epidemiologist.
7 Peter Shields might be involved from Georgetown.
8 Michael Goodman might be involved from Emory. These
9 are -- these are -- this is an epidemiology question and
10 I'm not an epidemiologist, so we had to get some help.

11 Q. Who -- who introduced you to this question?

12 A. Actually, I introduced them to the question.

13 Q. Okay. Just --

14 A. "Them" being API. I said, You guys need to
15 take the Williams and Paustenbach exposure estimates and
16 do something with them. Right now it's just been
17 published and no one has analyzed that data.

18 Q. For the Australian Petroleum Institute?

19 A. No. API is American Petroleum Institute.

20 Q. Oh, American Petroleum Institute.

21 A. Yeah. I don't have anything to do with -- they
22 may call themselves API, too.

23 Q. So are they going to fund this work?

24 A. Well, I hope.

25 Q. In theory?

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1 A. They funded the first part.

2 Q. You mean Paustenbach's work?

3 A. No. No, the first part where we took
4 Paustenbach's data and put it into a form where you can
5 evaluate it epidemiologically.

6 Q. Have you published that?

7 A. It's not publishable. It's very mundane. It's
8 taking them out of this form and putting them into this
9 table where you can then take this table and analyze it.
10 It was very boring work.

11 Q. It hasn't gotten more exciting since then, has
12 it?

13 A. Well, this part is going to be interesting.
14 The epidemiological evaluation I think could be very
15 interesting.

16 Q. And does this address something that has not
17 been answered before?

18 A. It's going to address many of the same
19 questions that people have. It's going to use it with
20 the most updated and the most rigorous exposure numbers,
21 which is why Williams and Paustenbach published their
22 paper in '03.

23 Q. So you're going to take the Williams and
24 Paustenbach '03 data and do something different with it,
25 correct?

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1 A. Yes. Do the same thing in some ways that's
2 already been done, but now we're going to be using the
3 most current and rigorous exposure numbers.

4 Q. But you're not going to change their data,
5 right?

6 A. The Williams and Paustenbach data?

7 Q. Right.

8 A. No. No.

9 Q. You're going to import their data into your
10 work?

11 A. Yes, do an epidemiological analysis of that
12 data.

13 Q. Of the Pliofilm cohort?

14 A. Correct.

15 Q. With the new exposure information?

16 A. Correct.

17 Q. And that exposure information will include the
18 dermal assessment, correct?

19 A. Yes.

20 Q. Both inhalation and dermal?

21 A. Correct.

22 Q. All right. Any other work specific to benzene,
23 other than that one, currently ongoing?

24 A. I was doing some work with the Texas Commission
25 on Environmental Quality that had to do with benzene,

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1 but I'm pretty sure they've made -- it was an air mobile
2 source emissions and they were setting standards and --
3 so I was just working with them informally to provide
4 papers; if they didn't have them, point out papers that
5 maybe they were missed. But I think they're done. That
6 was an open-ended, ongoing thing. And I've talked with
7 them a few times, but I think they're done.

8 Q. Who over there were you talking to?

9 A. Kip, Kip Howard I think is his name. Haney,
10 Kip Haney.

11 Q. Kip Haney?

12 A. Uh-huh. (Witness answers affirmatively.)

13 Q. How were you introduced to Kip Haney?

14 A. He actually introduced himself to me at SOT.
15 He came up to a poster I had and we discussed benzene
16 and what I do. And then how this arrangement was set up
17 is Stewart Cagen, who is a toxicologist with Shell,
18 asked me if I would make myself available to Kip if he
19 had any questions about the benzene literature.

20 Q. Who from Shell?

21 A. Stewart Cagen. He's a toxicologist with Shell.

22 Q. Shell the oil company?

23 A. Yeah.

24 Q. And how did Stewart Cagen know Kip Haney? Do
25 you know?

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1 A. Well, I think they had interactions over
2 this -- this process. He's a state regulator,
3 so...(Pause.)

4 Q. Who was paying you for your time?

5 A. No one.

6 Q. You were volunteering?

7 A. Yeah.

8 Q. Do you do any work for Shell on the side?

9 A. No.

10 Q. Have you ever?

11 A. No. Well, other than what part they are of
12 API, the American Petroleum Institute.

13 Q. I got you.

14 A. But never to them directly.

15 Q. They get some benefit from your API work,
16 though?

17 A. I would hope so, yes.

18 Q. Because they're one of the --

19 A. Right.

20 Q. -- petroleum companies?

21 A. Yes.

22 Q. All right. So Stewart Cagen asked you to
23 continue to help Kip Haney on the mobile source
24 emissions for benzene?

25 A. Yeah. They were setting a standard that was

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1 based on how much benzene can come out of a car. I
2 think that's what they were doing. I never was involved
3 in the regulatory process. It was purely science.

4 Q. They --

5 A. What do you think about these studies? Are
6 there problems with these studies? Make sure you
7 consider this. It was that kind of discussion.

8 Q. And Cagen is a toxicologist?

9 A. Yes.

10 Q. Why couldn't he do it for Mr. Haney?

11 A. I don't know.

12 Q. Was he qualified to do it?

13 A. He doesn't know as much about benzene as I do.
14 I mean, he's got a bunch of chemicals on his regulatory
15 plate.

16 Q. He has more on his plate than you do?

17 A. At least different kinds of chemicals, yes.

18 Q. Any other work?

19 A. Related to benzene, no.

20 Q. Currently?

21 A. Currently, no.

22 Q. How long would it take you to --

23 A. Oh, I'm sorry.

24 Q. Yes, sir?

25 A. Our company -- and this is mostly my two

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1 partners, Shawn and Lisa -- we just did a biomonitoring
2 equivalent where they're in the process of doing a
3 biomonitoring equivalent for benzene.

4 Q. For who?

5 A. Benzene -- oh, for who? I think it was the
6 so-called benzene task force, which is a combination of
7 API and ACC.

8 Q. And who?

9 A. ACC, the American Chemical Council.

10 Q. Okay.

11 A. They might call themselves something different
12 now. Anyway, they -- they have -- there's overlap in
13 industries and groups that -- that are concerned about
14 benzene. And so they formed, at least temporarily -- I
15 don't know if it's going to hold or not -- sort of this
16 collaborative group between those two. It was mainly
17 just to fund products. So I think that's the group that
18 has asked Shawn, us, to do the biomonitoring equivalent.

19 Q. What type of companies are on this benzene task
20 force? Are these like the Shells and Exxons of the
21 world?

22 A. That's on one side. And then the other side is
23 mostly chemical companies that aren't part of the
24 American Petroleum Institute.

25 Q. Like Dow?

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1 A. Dow is one big one, yes. Lyondell. There's a
2 lot.

3 Q. I got you.

4 And so you called it a biomonitoring
5 equivalent for benzene?

6 A. Right.

7 Q. Put that in regular people terms, what that
8 means.

9 A. It would take a little explanation, but --

10 Q. Do your best.

11 A. Okay.

12 Q. I don't need you to write a book on it.

13 A. I understand.

14 Right now there is a ton of biomonitoring
15 data that's in existence. CDC is doing it, University
16 of Michigan is doing it, University of Minnesota,
17 California, all over the place. They are producing
18 biomonitoring data which says we went into the
19 population, we looked, we found this much DDT, we found
20 this much dioxin, we found this much of this, we found
21 this much of that.

22 Okay. Then the CDC backs away, rightfully
23 so. That's their purview and that's their job to report
24 it, but they don't interpret it. So people are left
25 wondering, Okay, well, this is bad? I have this much

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1 DDT in me. Am I at risk? What's the health effects of
2 having this amount of DDT in my body? An understandable
3 question.

4 So what we propose to do -- and I'm not
5 going to take much credit for it, but it was my company;
6 it was mainly Shawn -- is form pharmacokinetic modeling.
7 You can take the EPA's reference concentration, which
8 EPA says this is a safe dose: 24/7, 70 years, you can
9 be exposed to this dose with no adverse effects, right?

10 Q. What do they call that dose?

11 A. Reference concentration or reference dose. You
12 accept it or no. Believe that it's true or not. Up or
13 down. Doesn't matter. That's what they call it. You
14 can take that dose and with pharmacokinetic modeling you
15 can say, Here is what your blood levels would be if you
16 were exposed at the reference concentration. Are you
17 with me?

18 Q. Yes.

19 A. So that is a biomonitoring equivalent of the
20 reference concentration. So that means that if you have
21 some level of cadmium or something in your body, and
22 you're concerned, Does this increase my risk of disease?
23 Does this increase whatever? You can look at the
24 biomonitoring equivalent and say, Okay, I am below the
25 reference concentration; therefore, based on EPA's

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1 calculations, it's not a problem.

2 Q. But you're doing this specific for benzene?

3 A. Well, they've done -- they've done, I think, 12
4 chemicals. Benzene is just one of them.

5 Q. What do you call it? The reference dose for
6 benzene.

7 A. It's the -- yeah, reference dose or reference
8 concentration.

9 Q. What is it for benzene?

10 A. I think it's 20 part per billion is about what
11 it amounts to.

12 Q. 20 part per billion?

13 A. Uh-huh. (Witness answers affirmatively.)

14 Q. Is that a "yes"?

15 A. Yes. I think.

16 Q. Part per million or billion?

17 A. Billion. So they're saying that at 20 part per
18 billion, you can be exposed to that your whole life, 24
19 hours a day, forever, and you would not expect
20 cytopenia, aplastic anemia, no disease from that
21 exposure.

22 Q. What is the purpose of that figure? What do
23 they do with it?

24 A. It's -- it's really important. They -- they do
25 it for -- because then regulatory groups and agencies

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1 and smaller public health agencies can use those
2 reference concentrations as standards and say, All
3 right, we -- we need our air levels to be below these
4 reference concentrations.

5 Q. Is this for, like, pollution standards? That's
6 what I'm trying to find them out.

7 A. Well, they call it a reference concentration.
8 I think it's probably used all over the place. You
9 would certainly use it in risk assessments. In a risk
10 assessment for a noncancer toxicity endpoint, I would
11 take your exposure and I would divide it by the
12 reference concentration to get a ratio. And if that
13 ratio is 1, then my interpretation of that risk
14 assessment would be you're not at risk. You were
15 exposed to the reference concentration.

16 If that ratio was 1,000, then the
17 interpretation would be you're exposed to 1,000 times
18 what EPA says is a safe dose, you know, take that for
19 whatever it's worth.

20 Q. But -- but the EPA doesn't define this 20 part
21 per billion figure, this reference dose or
22 concentration, as a fine line between safe and
23 dangerous, do they?

24 A. Oh, absolutely they do. Without a question.

25 Q. That's not right, though?

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1 A. Sure it is. I mean, it's right that they do
2 it. They absolutely -- no, they're not -- they don't
3 account in that number -- because there's a cancer slope
4 factor. But based on noncancer toxicity, they say 20
5 part per billion and you will suffer no adverse effects,
6 noncancer.

7 Q. Do they have a cancer figure?

8 A. Uh-huh. (Witness answers affirmatively.)

9 Q. What's the cancer number?

10 A. Cancer number is a different animal and it's a
11 risk that is associated with exposure. And I -- I'd
12 have to look it up.

13 Q. Can you find it real quick while we take a
14 break?

15 A. Sure.

16 (Short recess from 1:12 to 1:26 p.m.)

17 EXAMINATION (Continuing)

18 BY MR. LUBEL:

19 Q. Were you able to find it on the Internet, the
20 IRIS?

21 A. Yeah. We couldn't print it out. But the --
22 but the way they do it, as I was explaining earlier,
23 based on a range of exposure, their cancer slope factor
24 is 7.1 times 10 to the minus 3 to 2.5 times 10 to the
25 minus 2 at 1 part per million.

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1 Q. Okay. So their noncancer reference
2 concentration is 20 part per billion, correct?

3 A. Approximately, yes.

4 Q. And then this cancer reference concentration
5 seems a little bit more complicated.

6 A. It is.

7 Q. You said 7.1 times 10 to the negative 3?

8 A. And then it's a range to 2.5 times 10 to the
9 minus 2.

10 Q. At 1 part per million.

11 A. Right.

12 Q. What does that mean?

13 A. They say, Here is what the risk -- the
14 increased risk is going to be at a given concentration.
15 Perhaps a more useful way, they also use this
16 calculations and say, Here's what a 10 to the minus 4
17 risk is; here's what a 10 to the minus 5 risk is; here's
18 what a 10 to the minus 6, which would be a one in a
19 million risk. So they tell you what the concentration
20 would be at a one in a million, if you want to know for
21 regulatory purposes.

22 Q. So they don't have a -- a fine line between
23 safe and dangerous, the EPA, on cancer effects?

24 A. No. No.

25 Q. Why?

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1 A. For most chemicals, EPA uses the linearized
2 multistage model and that's how they model their data.
3 And there's certainly huge amounts of controversy over
4 whether that's appropriate or not, but it is a linear
5 model.

6 Q. Why is -- why is there a bright line, if you
7 will, on the noncancer effects and there is not a bright
8 line between safe and dangerous on the -- on the
9 malignancies?

10 A. Because the -- the endpoint that they're
11 modeling and using is a cytopenia based on Rothman's '96
12 paper. And cytopenias are unequivocally thresholded
13 events. There is definitely a threshold. In fact,
14 there is a threshold for almost all noncancer toxicity.

15 Q. You mean cytopenias, you're talking about
16 noncancer effects?

17 A. Yes. That is what their -- that reference
18 concentration, the way EPA does it, is they say, Okay,
19 here's all of our data about noncancer toxicity and
20 benzene or any chemical. And they say, All right, what
21 is the most sensitive endpoint that we can find? Is it
22 dizziness? Is it nerve conduction studies? Is it --
23 whatever, the most sensitive endpoint. And then they
24 say what -- for that endpoint, what is the most
25 sensitive species? Where do we see that effect

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1 occurring at the lowest level?

2 Q. People or animals?

3 A. It doesn't matter. Across the board. Then,
4 based on the most sensitive species, that's -- then they
5 take that -- and that's called the NOAEL, no observable
6 adverse effect level -- and they start dividing it by
7 uncertainty factors. They'll divide it by a factor of
8 10, if it's an animal study, because animals aren't
9 people. They'll divide it by a factor of 10 if it's a
10 short term and it needs to be chronic. They'll divide
11 it by a factor of 10 to account for sensitive
12 subpopulations like elderly or children.

13 So they'll just keep lowering it, lowering
14 it, lowering it, and then they'll come up with their
15 reference concentration.

16 Q. Why weren't they willing to do that for the
17 cancerous effects?

18 A. It's a whole different regulatory process. The
19 way they do -- because you've got cancer data at these
20 upper doses. You know, you see an increase in leukemia
21 at 200 part per million, you don't see it at these low
22 doses. Well, what -- what does that slope look like?
23 You can come up with a lot of explanations. Does it
24 drop off completely and there is no risk below a certain
25 point? Which is what a definition of a threshold would

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1 be. Does it flatten out? Is it supra or super linear?
2 There's all kinds of permeations. EPA, as a
3 precautionary principle, makes it linear, draws a
4 straight line through it. That's -- that's what they
5 do.

6 Q. So why don't they have -- "they" being the
7 EPA -- this 200 part per million as their threshold
8 level for benzene?

9 A. They don't use the 200 part per million at all.
10 They use the -- the raw data from the Pliofilm based on
11 the Paustenbach and Kenny Crump and Rinsky's analysis.
12 And then they put that into their model, confined by
13 using the upper 95th percent confidence interval and
14 confined by this multistaged linearized process, and
15 then they come up with a slope. And that slope is those
16 numbers that I gave you. It's called a slope factor.

17 Q. Is it lower than 200 ppm years?

18 A. I mean, you can calculate what the risk would
19 be at 200 part per million years. This is not -- these
20 aren't risk numbers -- well, they are -- these are
21 numbers that they -- based on that modeling, the risk of
22 AML increases per unit of exposure. That's the way they
23 calculate a slope factor.

24 Q. Do you agree with that?

25 A. No.

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1 Q. You don't agree that the risk increases with
2 each exposure?

3 A. Well, I think for benzene there is good enough
4 data -- they take this approach as a gene -- for any
5 gene toxicant, genotoxicity, they assume this linearized
6 approach. For some chemicals they are starting to move
7 away from it and to model the data slightly different.
8 For benzene, they have not done that yet. I think there
9 is sufficient data to support using a nonlinear model
10 for cancer.

11 Q. So what -- what does the EPA tell us -- what
12 would they tell us if we said that an individual's
13 exposures were over a work life 25 ppm years?

14 A. There would be a number that you could
15 calculate, based on the slope factor, that that would
16 represent an increased risk. It might be 10 to the
17 minus 6, but you could do it.

18 Q. You could do the calculation?

19 A. Sure.

20 Q. How long would it take you?

21 A. Me, personally?

22 Q. Yes, sir.

23 A. I don't know. A couple of days.

24 Q. Well, we won't start that today.

25 A. Yeah.

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1 Q. How's that?

2 A. That's why I said that.

3 Q. Is there a publication that we can look at for
4 various exposure levels? You know, that's -- where you
5 don't have to do the math, you can just look at it and
6 say --

7 A. Sure. The IRIS, the EPA has that. And then --
8 then you can go -- when you go to their Web site you can
9 have the EPA IRIS document and then you can have the
10 supporting documentation for cancer, and that will be a
11 250-page document where they have gone through all of
12 their discussions.

13 Q. But just so I'm clear with you, for
14 noncancer-causing effects, such as -- cytopenias was the
15 example you gave me, they draw a fine line between safe
16 and dangerous at 20 part per billion, correct?

17 A. That's their reference concentration. And
18 their definition is to -- it is not anticipated to be --
19 adverse effects over the lifetime of exposure.

20 Q. If it's at 20 or lower?

21 A. 20 part per billion or lower, correct.

22 Q. But for cancer, they were unwilling to kind of
23 draw that fine line and say everything above the line is
24 dangerous and everything below it is safe?

25 A. At this point, that's true. And not just for

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1 benzene. For just about every one of their chemicals.

2 Q. Do you agree with that concept, generally?

3 A. No. It's the -- they -- they will tell you --
4 "they" being the EPA -- is that there are things they do
5 that are science based. There are things they do that
6 are policy based. The policy is in the absence of
7 definitive data: If we're going to make a mistake,
8 we're going to make a mistake on the side of protecting
9 the public. It's called a precautionary principle.

10 This linearized multistaged model is a
11 policy decision. Tony -- Tony Cox and plenty of other
12 modelers have come up with alternatives to fit the AML
13 data in Pliofilm without a linearized model and you will
14 get widely different results.

15 Q. How much benzene exposure does it take for an
16 individual to get MDS?

17 A. My position on that would be -- well, first of
18 all, MDS is -- that's kind of like saying leukemia. So
19 there's a lot of different forms of MDS.

20 Q. An MDS that you believe can be causally related
21 to benzene.

22 A. Okay. Okay. So then you -- when you get those
23 myelodysplasias, they transform so rapidly to AML and
24 they have the same cytogenetic profile as the AML that
25 follows it that, you know, arguably, they are just

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1 different phases of the same disease. And so when they
2 have been evaluated, it is the same cutoff that you
3 would see with -- with AML. But there's less data for
4 MDS than there is even for AML. Like, in the Pliofilm,
5 there is no mention of a myelodysplasia.

6 Q. But you treat MDS and AML the same?

7 A. In terms of quantification of benzene, I do.

8 Q. In terms of the disease?

9 A. You mean treat them?

10 Q. No.

11 A. Like clinically treat them?

12 Q. No, I'm not talking about treating them. I'm
13 talking about in causation with benzene.

14 A. As long as it's the form of MDS that I think
15 has been, you know, linked.

16 Q. All of the MDSSs, excluding ringed sideroblasts.

17 A. That is certainly one. The refractory anemia
18 by itself, that's -- that's not as clear to me as the
19 excess blasts and excess blasts and transformation.

20 The CMML I don't believe is linked with
21 benzene. In fact, that one is no longer even considered
22 a myelodysplasia syndrome. They've moved it into the
23 myeloproliferative disorders.

24 Q. So the only two that you are clear on right now
25 that you don't associate with benzene exposure are the

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1 ringed sideroblast and the CMML?

2 A. Yes.

3 Q. You have questions about RAAB?

4 A. No. I think that one is probably a benzene,
5 possibly --

6 Q. RA. You have questions about RA?

7 A. Right. Refractory anemias.

8 Q. What -- what's the best literature on the
9 connection between MDS and benzene? Is it the NCI work?

10 A. There's -- that's -- they evaluated it. They
11 never separated them out. But you can go through in the
12 Travis '94 paper and see how many of each one they saw.
13 You could do the analysis, if you wanted. I think
14 probably the single best study is a Strom, S-t-r-o-m,
15 that was published in Blood in 2005, where they looked
16 at a variety of etiological factors with the subtypes of
17 MDS. And they found smoking was associated with RA and
18 with RA and ringer sideroblast. They found benzene was
19 not. And that benzene was associated with excess blasts
20 and excess blasts and transformation. The CMML they
21 took out of the equation because that's not even really
22 a myelodysplastic disease.

23 Q. So you think the Strom article is the best
24 article that illuminates --

25 A. It has -- it has specifically addressed this

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1 paper. And Blood is a heck of a good journal.

2 Q. Do you think the Strom work is better than
3 the -- than the body of the NCI work?

4 A. Oh, they are totally different. They're very
5 different.

6 Q. As it relates to MDS?

7 A. Even in that sense, they're so different.
8 Because the -- because the NCI has all of the
9 quantitative exposure and all -- I mean -- and it's got
10 its own huge set of problems. It would be hard to
11 compare those. But on this one particular issue of
12 subtypes and benzene, the Strom paper is pretty clear.

13 Q. What's the best piece of work on the level of
14 exposure to benzene necessary to be causally related to
15 MDS?

16 A. I would say then you probably are talking about
17 NCI. With the caveat that there are huge problems with
18 that exposure assessment in that study. So I mean, I
19 think the pathology and I think, you know, overall, it
20 was a credible piece of epidemiology. And I think they
21 did the best they could. But to rely on that, draw a
22 hard and fast line and say 40 to 90 you see a risk, I
23 don't think you can do that. I think those exposures
24 have been grossly underestimated in -- in many cases.
25 Not in every a case, but in many cases.

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1 In other words, the risk that they -- that
2 they reported from 40 to 90, I think those people have
3 much higher exposures than that.

4 Q. Actually, if you -- if you look at the latest
5 NCI publication --

6 A. Uh-huh. (Witness answers affirmatively.)

7 Q. -- which was, what, '01 or '03?

8 A. No, they published one in '05.

9 Q. '05?

10 A. And I think in '06.

11 Q. Well, if you look at '01 or '03, one of those
12 talks about excess risk at greater than 10 ppm years.
13 Do you recall that?

14 A. I -- vaguely. I don't. That certainly is not
15 evident in the '97 Hayes paper.

16 Q. Because you said the '97 Hayes paper has that
17 range of 40 ppm to 90 to 100 ppm?

18 A. Something like that.

19 Q. Something like that?

20 A. Where they saw an increased risk of MDS/AML.

21 Q. What subtype of AML did Mr. Oakley have, if we
22 know?

23 A. Based on my understanding of the medical
24 records, it was an M1 or perhaps M2 -- no, maybe M0. M0
25 and 1, I think is what they said.

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1 Q. Are you satisfied with that?

2 A. I -- I rarely will question that. I'm not
3 qualified to.

4 Q. You're just going to rely on what the record
5 says, right?

6 A. Yes.

7 Q. Earlier you said you had -- you either said, I
8 have some reservations or some problems with the work
9 that Dr. Irons had set out to do in China. Can you
10 elaborate on that?

11 A. Well, I just think there were some concerns
12 about, you know, this notion of -- and this was very
13 early on, and I think they've addressed all of these --
14 they've probably addressed it by pulling in Helm McRhine
15 and Richard Larson and a lot of others for an ethical
16 review board. But identifying a high -- a high-risk
17 population without making steps to rectify the
18 situation, and I think they are doing that.

19 Q. You mean studying the people?

20 A. Without -- without saying, you know, if you
21 guys take benzene out of the manufacturing stream, you
22 might -- it might be better. And I think they are
23 definitely doing that.

24 Q. You were told they were doing that?

25 A. Oh, I think -- that's my understanding, yes.

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1 Whenever they -- whenever it's possible to do it. And
2 discussing respiratory and personal equipment.

3 Q. What do you mean, whenever it's possible to do
4 it? What would be impossible about telling somebody
5 their exposures are dangerous?

6 A. Yeah, I don't know -- that probably wasn't the
7 most appropriate way to put it.

8 Q. I think we -- correct me if I'm wrong -- but
9 what you mean is that there may be some employers that
10 won't let them in the facility to run any tests if they
11 are going to be talking straight with their workers.

12 A. I don't know.

13 MS. YATES: Objection; form.

14 A. I don't -- I don't know the answer to that. I
15 mean, there's going to be -- there will be a variety of
16 responses based on the fact that they're looking at so
17 many different factories or were.

18 Q. (By Mr. Lubel) And what -- what limited role
19 did you have in that study?

20 A. It was very limited. I mean, I taught Rick
21 Irons' etiology class when he went and did the initial
22 -- what do they call it? -- kind of a screening study
23 when he went over -- feasibility study when he went to
24 China for several months to decide, you know, are there
25 enough? Is it feasible to do this?

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1 And then I was working with him in the lab
2 when he was discussing kind of the study design. But
3 I -- but I had even less to do with this than the --
4 than the Liquid Wrench simulation study.

5 Q. You didn't really participate in the Shanghai
6 health study study design, correct?

7 A. No.

8 Q. You won't take credit for that?

9 A. No, I will not.

10 Q. Nor the blame if something goes wrong. That
11 goes both ways, right?

12 A. Right.

13 Q. The exposure levels necessary to induce the
14 cause of benzene malignancy, we've talked about the
15 Glass article, the Guenel article, the Pliofilm set of
16 articles, that cohort, and you generally talked about
17 the NCI article. Are there any other articles that need
18 to be considered in that topic?

19 MR. GRAY: Objection; form.

20 A. I think so.

21 MS. YATES: Same objection.

22 A. Sure. I think the Glass paper, from the Health
23 Watch, there is a whole -- again, there is a whole
24 series of studies.

25 From the NCI, there's a whole series of

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1 studies. All of those are important.

2 Schnatter from the Canadian Petroleum
3 Worker published a couple of cohorts. And then nested
4 case control studies that bear on this study that did a
5 quantitative analysis of AML.

6 Leslie Ruston did the same thing with the
7 UK.

8 Rob Snyder evaluated the Pliofilm cohort
9 and calculated risk based on air concentrations and
10 found that in that cohort there really was no increased
11 risk at an air concentration below 20 part per million.
12 So it's getting at another -- we've been almost
13 exclusively talking about cumulative exposures, but he
14 wanted to know whether or not there were issues
15 associated with, you know, what the air concentrations
16 were.

17 So there's -- there's going to be overlap
18 and they're going to be important together. But
19 that's -- that's another thing that bears on this
20 question.

21 Q. What -- what causes disease? Is it the -- is
22 it the concentrations or is it the cumulative exposures
23 over a lifetime?

24 A. By disease I'm assuming you are talking about
25 AML.

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1 Q. Yes, sir.

2 A. I would think that it's likely to be a
3 combination of those two. And it's likely to be a
4 combination of those two within an appropriate time
5 window.

6 Q. And so how do we weigh which of those is more
7 important: Cumulative exposures versus a particular day
8 of concentration?

9 A. I think to some extent you can say that that is
10 somewhat of an open-ended question, but in studies where
11 they've tried to evaluate which of those were the most
12 important in terms of risk, you'll find that some -- you
13 would find an answer on both sides of that.

14 Q. Where do you fall?

15 A. Umm, I don't -- I don't know the answer to it.
16 I mean, I think that if you just look strictly at
17 cumulative exposure, I don't think that tells you the
18 whole picture. 200 part per million years, that could
19 be 40 part per million for five years or 200 part per
20 million for one year. Okay, that's going to -- that
21 will represent very different exposure parameters and
22 probably a very different biologic response. So I don't
23 think that's all there is to that equation.

24 Q. Well, what difference does the amount of
25 exposure have on the latency period?

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1 A. That's a good question. I think for most
2 chemicals, the argument would be supportable that the
3 higher the exposure, the longer the latency.

4 Q. The longer?

5 A. The longer. No, I'm sorry. You're right.

6 Q. Shorter?

7 A. You're right.

8 Q. Reverse that, right?

9 A. Yes. Yes.

10 Q. So if you have somebody that's got chronic
11 lower level exposures to just chemicals in general, you
12 would expect the time period between the last exposure
13 and the onset of disease to be longer than you would the
14 higher the exposures that they have?

15 A. Well, I don't know about that last exposure to
16 the onset of disease, but I think the overall process
17 might take longer.

18 Q. So -- so the heavier the exposures, the shorter
19 the latency?

20 A. In general.

21 Q. In general?

22 A. In general.

23 Q. There is no formula, is there?

24 A. No. And I think that's probably -- that's
25 probably fair with benzene. You know, they have -- like

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1 we talked about earlier, they have gone in and evaluated
2 when the exposures have to occur to increase the risk of
3 AML. And I think you have to factor in all of those.
4 You have to factor in the cumulative, you have to factor
5 in the air concentrations, and then you've got to put it
6 within that appropriate window, which is about 10 to 15
7 years prior to diagnosis.

8 Q. Have you seen any of the observed cases falling
9 outside that 10 to 15 years, that window?

10 MR. GRAY: Objection; form.

11 MS. YATES: Same objection.

12 A. I have not. When you -- when you look at the
13 regular epidemiology, the way that epidemiologists will
14 sometimes define latency can be very different. And
15 some of them will define latency at the end of the
16 exposure period; some of them will define latency at the
17 first day of employment. So when you have that much
18 variability in the description, yeah, you're going to
19 have people that -- 34, 40 years, all over the place.

20 The other way of looking at it is: When
21 did the exposures matter for the AML risk? And you get
22 away from when things started and you just figure out
23 how it ended. And that's a more comprehensive -- it's
24 certainly is a more consistent pattern with the three
25 big cohorts that it's been done in.

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1 Q. (By Mr. Lubel) So some scientists
2 calculate the latency from the beginning of the
3 exposures, correct?

4 A. Some epidemiologists will use that definition,
5 yes.

6 Q. Some calculate it from the last exposure?

7 A. Yes.

8 Q. And some calculate it from the most significant
9 exposure that can cause the event, the disease.

10 A. Yeah, I don't even know if they call what I'm
11 describing the latency. It's more been termed as the
12 etiological window, or at least that's what I think is
13 the more appropriate term. They may use the word
14 "latency."

15 Q. Etiological window?

16 A. Right. When do the exposures -- when are they
17 likely to be involved in the etiology of the disease?
18 In other words, if you get -- if you -- if you catch AML
19 in 2005, but you were exposed to benzene in 1950, that's
20 probably not related, based on this analysis.

21 Q. Can you pull the articles that -- that address
22 this point?

23 A. Yeah. You can't --

24 MR. LUBEL: Take a break.

25 (Short recess from 1:51 to 1:58 p.m.)

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1 EXAMINATION (Continuing)

2 BY MR. LUBEL:

3 Q. Did you pull those articles?

4 A. I did.

5 Q. Okay.

6 A. The first one -- and she did publish a paper in
7 '05, and I don't have it in this binder, but it's
8 somewhere -- but this is some of the supporting
9 documentation from the Health Watch Technical Report and
10 Appendices, May 2001. And there is a whole section in
11 here starting on Page 75, exposure time frame and
12 lymphohematopoietic cancer.

13 Q. Is this Page 75 of the Glass --

14 A. This is Page 75 of the Lymphohematopoietic --
15 Lymphohematopoietic Cancer and Exposure to Benzene in
16 the Australia Petroleum Institute Technical Report and
17 Appendices May 2001. And she's published this as well.
18 I just -- in 2005. I just don't have that paper in
19 front of me.

20 And what they did was, that exact thing.
21 What are the risks of exposures five years prior to
22 diagnosis, ten years prior to diagnosis, or 15 years
23 prior to diagnosis? And at exposures 15 years or more
24 prior to diagnosis, there is no longer a statistically
25 significant increase in AML.

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1 Q. In that cohort?

2 A. In this cohort.

3 Q. How many total AMLs were they looking at?

4 A. I think 11, 12, something like that.

5 Q. Can you make sure?

6 A. That would be in -- in the Glass paper. Well,
7 then they updated it. In the Glass paper, the 2003, I
8 think they were actually --

9 MR. GRAY: You-all are welcome to look at
10 mine. I would not like to mark it because it has my
11 highlights.

12 A. Total leukemia cases, looks like they have 33.

13 Q. (By Mr. Lubel) How many of those were AMLs?

14 A. They had 11 ANLLs and I think two of those were
15 AUL, kind of unspecified, maybe, biphenic --
16 biphenotypic. So 9, probably confirmed diagnosed cases
17 of AML, but 11 that went into their calculation.

18 Q. Did they do a subtype breakdown of the AMLs?

19 A. No.

20 Q. They did not?

21 A. No.

22 Q. So --

23 A. Go ahead.

24 Q. -- out of the -- should we say 11 or 9 AMLs?

25 A. You can use 11, that's fine.

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1 Q. Out of the 11 AMLs, how many fit within the
2 different categories of the exposures prior to
3 diagnosis? You were doing -- you were looking at some
4 chart.

5 A. Uh-huh, it's in there.

6 Q. On Page 75, wasn't it?

7 A. Well, there's a whole bunch of different ones.

8 Q. Just tell me what table we need to look at and
9 what page.

10 A. I would say you should probably pay attention
11 to Table 46, LH Cancer Odds Ratio of Lag Exposure to
12 Groups PPM Years. Lymphohematopoietic Cancer in Lag
13 Cumulative Exposures. Proximity of exposure within five
14 years, within ten, within five years, within 15 years.

15 Q. Does it give you the numbers that fit into each
16 category of the cases?

17 A. No. You could calculate that based on the odds
18 ratio. And I think somewhere they do say how many they
19 actually had.

20 Q. Because 11 isn't that many, is it?

21 A. It's more than you'll find in some of these
22 other studies that we've been talking about, some of
23 these epidemiology studies.

24 Q. Would you base your opinion solely based upon
25 this article?

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1 A. You mean in terms of latency? In terms of the
2 etiological window?

3 Q. Right.

4 A. If this was the only one to it, I think that
5 would be a fair criticism that they didn't have that
6 many and that it's kind of new and no one had really
7 seen that before. But the fact that it is so consistent
8 with our understanding of chemotherapy-induced leukemias
9 and there the numbers are hundreds, thousands. You
10 know, we know a lot about that. And then these other
11 two cohorts, which we'll get into because that is
12 consistent across the board, yeah, I believe it.

13 Q. Okay. Let's look at the other two cohorts.

14 A. Okay. This is a paper by Murray Finkelstein,
15 Leukemia After Exposure to Benzene: Temporal Trends and
16 Implications For Standards.

17 And what he says, "In the case of benzene,
18 it would appear that risk is attributable primarily to
19 exposures incurred within the previous 10 to 20 years,
20 with exposures in the most recent 10 years being the
21 most potent. To limit risk, exposures must be
22 controlled during that interval."

23 And then there is another one that just
24 came out this year, 2008, from David Richardson.
25 Temporal Variation in the Association Between Benzene

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1 and Leukemia Mortality. And it just really confirms
2 that. "The association between benzene mortality and
3 benzene exposures was of the greatest magnitude in the
4 ten years immediately after exposure."

5 Q. After what exposure? The last exposure or the
6 first exposure?

7 A. The last exposure. Ten years prior to
8 diagnosis.

9 Q. That's what Richardson says?

10 A. Yes.

11 Q. Could you find that for us?

12 A. No, this one is time since exposure.

13 Q. Since what exposure?

14 A. The last exposure.

15 Q. So 10 to 20 years after the last exposure?

16 A. That's what this says, yeah. With the -- with
17 the strongest being within ten years.

18 Q. Okay.

19 A. Ten years immediately after last exposure.

20 Q. All right. Now, let's go back to
21 Finkelstein -- was it Finkelstein?

22 A. Uh-huh. (Witness answers affirmatively.)

23 Q. Do you know that author, Finkelstein?

24 A. No. I don't know this one either.

25 Q. You don't know Richardson either?

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1 A. No.

2 Q. Finkelstein, can you find how they calculate
3 that window, whether it's first exposure, last exposure,
4 or some other event?

5 A. Let's see. More recent exposures were
6 strong -- more recent exposures were more strongly
7 associated with risks than were distant ones. There
8 were no significant relation between leukemia death and
9 benzene exposures incurred more than 20 years
10 previously. I'm looking for what you asked. Death.

11 Q. What was that?

12 A. Prior to death. The windows were prior to
13 death. One to four years, five to nine, 10 to 14, 15 to
14 19, et cetera, so... (Pause.)

15 Q. Prior to death?

16 A. Right. So that would be close to diagnosis.

17 Q. Can you show me what page you're referring to
18 in Finkelstein?

19 A. Uh-huh. (Witness answers affirmatively.)

20 Q. And give me the page number.

21 A. Table 1, Individual Exposure Windows 1 to 4
22 Years Before Death.

23 Q. Can you help me read that? What does that tell
24 you?

25 A. Well, this is the coefficient where they --

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1 they looked at how much the risk changed the slope of
2 their cumulative risk estimates. And no, I can't give
3 you any more than that. I would rely solely on his
4 interpretation in the conclusions.

5 Q. You can't get it -- you can't parse it from his
6 table of data? You just -- you're just relying on his
7 words --

8 A. Yeah.

9 Q. -- correct?

10 A. Yeah, I mean, I don't know what -- and even the
11 Richardson paper, it's got some math in it that -- you
12 know, I'm not an epidemiologist. I don't understand it.

13 Q. But those are the three --

14 A. No. No. There's another one.

15 Q. There's another one that you're relying upon?

16 A. Uh-huh. (Witness answers affirmatively.)

17 The Hayes paper, '97, does the same kind of
18 thing. And --

19 Q. What page?

20 A. Table 2. And what they find is that this is
21 less than five, five to ten, of duration. So less than
22 five years' duration the risk is much higher than in
23 greater than ten years' duration.

24 Q. Greater than ten years' duration of what?

25 A. Of working in this benzene environment.

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1 Q. So you're saying the more you're exposed, the
2 less risk you're at?

3 A. No. I'm saying --

4 Q. The longer you're exposed?

5 A. -- that the risks were the highest for the
6 short-term worker than they were for the longer-term
7 worker.

8 Q. That doesn't make any sense.

9 A. Yes, it does. It's entirely consistent with
10 everything that we've just been talking about.

11 Q. I'm talking about just Bradford-Hill Criteria.
12 Doesn't that tell us that -- that the dose response you
13 expect to see is the -- is the greater exposures you
14 have, the more risk you have?

15 A. No. What this is saying is that the exposures
16 were more important and gave a higher risk if they
17 occurred -- in five years than they did if they occurred
18 within ten years or if they occurred within 20 or ten.

19 Q. Okay. Where does the article say that in order
20 for your malignancy to be benzene-induced the window has
21 to be within this 10- to 20-year period that you're
22 talking about?

23 A. I think their assumption in this table is they
24 were all benzene-induced.

25 Q. Where is the conclusion that they say it can't

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1 be benzene-induced if it doesn't happen within the
2 window of 10- to 20 years?

3 A. I don't think anyone has ever been that
4 literal.

5 Q. You are.

6 A. No, I'm not. I'm saying that that time window
7 is most consistent with our understanding. The latency
8 seems to be within 15 years prior to diagnosis. 16
9 years prior to diagnosis, I mean, absolutely, that's not
10 going to count? I couldn't say that. There's just too
11 much variability.

12 But within ten years, that makes the most
13 sense. And they do say in there, probably down on the
14 bottom, that most recent exposures gave a higher risk
15 than more distant exposures. Look at it this way --

16 Q. I hear what you're saying. So what you're
17 saying is that the more recent exposures seem to play
18 the biggest role?

19 A. Yes.

20 Q. You're not saying that the more distant
21 exposures play no role whatsoever.

22 A. Well, in this -- in this particular study, what
23 you said is true in that they didn't do that kind of
24 analysis. They're just saying that the more recent ones
25 are more important. In some of these other analyses

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1 they say that outside of this 15-year window, they don't
2 matter. They just don't impact on the risk. When a
3 patient -- when a patient is treated with leukemogenic
4 chemicals, alkylating agents, or whatever in the clinic,
5 their risk goes up at about six months, it peaks at
6 about five years, and then their risk starts dropping
7 until after about ten years, the treating physicians
8 will generally tell them that your risk of developing a
9 leukemia now from your treatment is no different than
10 the general population. That's what I'm talking about.
11 That at some point out, your exposures no longer
12 increase your risk of developing that disease. It's
13 like people that their risks of lung cancer go down
14 after they stopped smoking. Their risk doesn't stay
15 elevated through their whole life. It precipitously
16 starts dropping off.

17 Q. But there's a difference between dropping off
18 and saying it reaches zero, you have no risk anymore.
19 No study defines no risk.

20 A. Well, what I'm saying is that the Health Watch,
21 they say that those exposures don't matter.

22 Q. That's one study.

23 A. That's one analysis. That's true.

24 Q. You've already told us one study doesn't get
25 you there in science.

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1 A. No, but I think it is totally consistent with
2 all of these other ones. And that is the most important
3 window is within ten years of diagnosis.

4 Q. Those exposures are the most important?

5 A. That's right.

6 Q. But you're not taking the position under oath
7 that the exposures greater than ten years are not
8 important at all?

9 A. No.

10 Q. You wouldn't do that, would you?

11 A. No.

12 Q. Not at this point?

13 A. At some point 20 years out and I would take
14 that position.

15 Q. At 20?

16 A. 20.

17 Q. A fine line at 20?

18 A. Well, because that's where the Pliofilm did it.
19 They said, ten to 20, and outside of 20, we cannot
20 tell -- what they did was they looked at all of the
21 exposures and the risk. And they did a sensitivity
22 analysis. And the math I don't get. But basically what
23 they said is, If we start removing exposures in these
24 different time windows, what does that do to the risk of
25 leukemia? And if you remove the exposures that occurred

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1 very soon, prior to diagnosis, that changed the risk.
2 If you removed the exposures that occurred very
3 distantly to diagnosis, that did not change the risk.

4 Q. 20 years from last exposure, though, correct?

5 A. In that study.

6 Q. Is that the one you're following or not?

7 A. Well, that's the one that where the 20 would
8 kind of be a bright line. The Health Watch is ten to
9 15.

10 Q. I need you to tell me whether from your
11 calculation you're using this fine line of 20 years
12 from -- from first exposure, last exposure, or some
13 other event.

14 A. My -- my position, under oath, would be that
15 the most important exposures are within ten to 15 years
16 of diagnosis.

17 Q. But I'm asking you a different question. You
18 told me that you draw a line at 20 years, do you recall
19 that, just a second ago?

20 A. Yes.

21 Q. What I'm trying to find out is how do we --
22 from where do we calculate that 20 years from last
23 exposure?

24 A. No. 20 years prior to diagnosis. There
25 doesn't seem to be any evidence that 20 years prior to

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1 diagnosis or death, you're going to have any risk.

2 Q. Any risk or significant risk?

3 A. They say it doesn't affect the risk numbers.

4 Q. Who says that?

5 A. All of this literature that we've been
6 discussing.

7 Q. Health Watch, Finkelstein, Richardson, and what
8 else?

9 A. The NCI.

10 Q. And Hayes, '97?

11 A. Hayes, '97.

12 Q. Any other studies that you're relying upon for
13 this point?

14 A. Sure. Every clinical study with chemotherapy.
15 And there's probably 10,000 of them -- well, say 1,000
16 of those. There's lots of them.

17 Q. Did you bring any with you?

18 A. I brought some.

19 Q. Which ones did you bring? Can we use those?

20 A. No. I mean, you can use them. You can put
21 them into the record, if you'd like.

22 Q. But you need to take them with you when you go.

23 A. And I'm not sure I brought the therapy-related
24 leukemia binder. But I will tell you under oath that it
25 is unequivocal that they -- the risk of developing

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1 leukemia after their -- there the numbers are much
2 cleaner, because we know exactly when they were exposed,
3 right? They were exposed during treatment. Following
4 that exposure, if they don't develop leukemia within ten
5 years, their risks are back to normal.

6 Q. Within ten years?

7 A. Right.

8 Q. Is that for all chemotherapeutic agents?

9 A. Sort of the long end is, yes. I mean, some,
10 like topoisomerase drugs you would see a lot quicker
11 than that.

12 Q. Three to eight years?

13 A. Sometimes you can see leukemia associated with
14 topo drugs in six months.

15 Q. Have you done a comparison of the dose that the
16 patient is given to the chemo and compared that to the
17 exposures that these benzene-exposed workers have in the
18 epi studies?

19 A. That is actually a great idea. I have not done
20 it, but I --

21 Q. You're going to write an article on it now,
22 aren't you?

23 A. If I do --

24 Q. Do I get credit for it?

25 A. No, because I already had the idea.

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1 Q. Okay. Not bad for a lawyer to think of it,
2 though?

3 A. It's a good idea. It's an important question.

4 Q. That will take some time?

5 A. It will and there's going to be a ton of
6 variability.

7 Q. Why?

8 A. Well, because people are on different drugs.
9 There's all kinds of different drug cocktails and they
10 can't even definitively say which one of the four drugs
11 they treated this person with is the one that gave them
12 leukemia. So, you know, it's -- it's a fuzzy concept.

13 There, however, the timing is very clear
14 and you can get the quantitative dose information.
15 Whereas, in all of this other literature with the
16 benzene, well, the timing isn't all that clear, but the
17 dose is even less clear.

18 Q. We've covered the timing issue, correct? We've
19 covered the latency, right?

20 A. (Witness nods head.)

21 Q. Have we covered the exposures are too low by
22 virtue of the epi that you've talked about and what's in
23 your report?

24 A. (Witness nods head.)

25 Q. Is that "yes"?

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1 A. Yes, I think we have. Yes.

2 Q. We have not covered in detail your blood counts
3 returning to normal after removed from exposure, have
4 we?

5 A. I think we have.

6 Q. You think we have?

7 A. Yes.

8 Q. Is that also in your report in detail?

9 A. Yes.

10 Q. Now, when you say the lymphocytopenia very
11 sensitive, no decrease, not consistent. Do you recall
12 that being one of the opinions?

13 A. Yes.

14 Q. Can you elaborate on that?

15 A. Sure. In the animal data, as well as in people
16 data, human epidemiology, while you do see changes in
17 the white blood count, most of the changes --

18 Q. Sorry.

19 A. -- most of the changes in the white blood count
20 are accounted for by lymphocytes. They seem to be the
21 most sensitive subtype; not always. You'll see
22 neutropenias and thrombocytopenias, but lymphocytes tend
23 to be the one that most people consider the most
24 sensitive cell type.

25 Q. To benzene exposure?

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1 A. To benzene exposure. And their reference
2 concentration that we were discussing from IRIS is based
3 on the Rothman '96 analysis. And that was the only
4 thing that they saw in those workers exposed to seven
5 part per million over a six-year period was a change in
6 their lymphocytes. So that is the basis of our
7 regulatory noncancer endpoint, a change in lymphocytes.

8 Q. What's a lymphocyte again?

9 A. A lymphocyte is a lymphocyte. It's a form of a
10 blood cell. It's a white blood cell that is part of
11 your immune system. There could be T lymphocytes and B
12 lymphocytes.

13 Q. He did not have a decrease in lymphocytes.

14 A. Not that I saw.

15 Q. "He" being Mr. Oakley, correct?

16 A. Yes.

17 Q. And so you're not saying that rules out the
18 connection to benzene. You're saying that's another
19 clue?

20 A. Yes. Yes. So that was inconsistent with what
21 you would expect. If his cytopenias were related to
22 benzene, you would think that the most sensitive
23 endpoint would be the one that's changing.

24 Q. But you've already told us that a decrease in
25 lymphocytes is not -- is not necessary in order to prove

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1 a benzene-induced malignancy, correct?

2 A. It's not -- what did you call it?

3 Q. Dispositive?

4 A. Yeah, it's not that.

5 Q. Conclusive or whatever.

6 A. Yes.

7 Q. Don't be stealing my words. You don't want
8 me --

9 A. I won't be able to. I couldn't remember it.

10 Q. Okay. Let's -- you said something about the
11 cytogenics.

12 A. Uh-huh. (Witness answers affirmatively.)

13 Q. Correct?

14 A. Yes.

15 Q. He's got normal chromosomes?

16 A. Yes.

17 Q. Okay. The majority of people that have
18 benzene-induced malignancy have abnormal chromosomes,
19 correct?

20 A. Correct.

21 Q. Why?

22 A. Well, because it's probably part of the
23 mechanism by which a cytotoxic chemical like benzene can
24 transform a cell. It involves an alteration in the
25 chromosomal structure. We've known that -- you know,

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1 Forney was publishing that back in the '60s. They
2 didn't have the chromosomes identified, but they saw
3 chromosomal changes in benzene-exposed workers. So that
4 is a well-established toxic effect of benzene.

5 Q. Can an individual have a benzene-induced
6 malignancy with normal chromosomes?

7 A. I would say -- I couldn't say that
8 absolutely -- it absolutely could not happen. But I
9 would say it is in the vast, vast minority. And that is
10 also based on looking at the chemotherapy-related
11 leukemias, also cytotoxic drugs, maybe they work the
12 same way as benzene. They also, like 90 or 95 percent
13 of them, will have some type of cytogenetic change. So
14 simply based on that one fact, you would say -- what? --
15 the chances are 1 in 20, just based on that alone that
16 Mr. Oakley's has anything to do with benzene.

17 Q. If other defense experts, like Dr. Irons, have
18 testified, just recently on behalf of U.S. Steel, that
19 abnormal chromosomes are not necessary for an AML to be
20 related to benzene exposures, do you disagree with that?

21 A. I wouldn't disagree with that necessarily
22 because I don't know what he was basing that on. He
23 published a paper with myelodysplasia. And at that
24 time, when he went in and looked at their bone marrow,
25 there were no cytogenetic changes. But they didn't

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1 have -- I mean, it could very well be a progression and
2 that it hadn't developed at that point, the cytogenetic
3 changes.

4 Q. Well, how long does it take to develop?

5 A. Well, I don't think that's known. But I think
6 the chemotherapy, which is an appropriate model, is
7 pretty clear on that point. So, no, I wouldn't say it's
8 an absolute, you can't have it, but I think it's in the
9 minority.

10 Q. Minority meaning fewer of the cases have normal
11 chromosomes that are benzene-induced?

12 A. Way fewer, much more.

13 Q. Do you know Myron Mehlman, Dr. Mehlman?

14 A. I -- I was on -- at a speaking conference -- it
15 wasn't the same one we were talking about earlier -- but
16 he was part of the -- part of the panel, so I met him.
17 But, no, I don't. I don't know him.

18 Q. You didn't do any work with Dr. Mehlman when he
19 worked for Mobil?

20 A. No.

21 Q. Have you done any work for Mobil Oil Company,
22 now Exxon?

23 A. No, not them directly. Them as a constituent
24 of API.

25 Q. Let's go through your materials.

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1 A. Okay. Oh, sure.

2 Q. Getting close.

3 A. Yep.

4 Q. Let me start over here with these.

5 You've got the Stephen Petty exposure
6 report in the case.

7 A. Yes.

8 Q. You've written down some numbers.

9 A. I don't think they have anything to do with his
10 report. I might have just had it on my desk when I was
11 doodling about something else.

12 Q. So you've considered his report for your
13 opinions, correct?

14 A. I read it.

15 Q. Considered it --

16 A. Okay.

17 Q. -- true?

18 A. Well, I mean, my -- I base my quantitative
19 exposure information on John Spencer, on his report.
20 And the differences between them and what's wrong with
21 Stephen Petty, I would leave to John to discuss.

22 Q. That's not your area?

23 A. It is not my area.

24 Q. What -- tell me what that is. Is that a
25 Federal court list of the cases you've testified in?

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1 A. I have no idea. Oh, it looks like this is
2 Mr. Petty.

3 Q. This has just fallen off the report?

4 A. Yeah, I don't recognize any of those.

5 Q. Are you claiming that Mr. Oakley's asbestos
6 exposures caused his leukemia?

7 A. Am I claiming that?

8 Q. Sure.

9 A. I'm not claiming that.

10 Q. There is no support for that, is there?

11 A. Not on that piece of paper you're holding up.
12 I'm not claiming that at all, no.

13 Q. Have you worked on asbestos cases?

14 A. No. I have steadfastly refused.

15 Q. So you're not relying on any claims in an
16 asbestos case, are you?

17 A. No.

18 Q. And you've read Dr. Levy's report?

19 A. Yes.

20 MR. GRAY: Lance, he also brought a list of
21 things that he's looked at.

22 MR. LUBEL: Okay.

23 Q. (By Mr. Lubel) You're not relying on the -- on
24 the -- on the lawsuit for your opinions, are you?

25 A. No.

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1 Q. The actual designation of experts?

2 A. No.

3 Q. The initial disclosures?

4 A. No.

5 Q. The personnel file of Mr. Oakley at Air

6 Products?

7 A. No.

8 Q. Myelodysplasia Studies.

9 MR. GRAY: For the record, that's a binder
10 of studies.

11 Q. (By Mr. Lubel) You're not specifically relying
12 on any of these articles for your opinions in this case,
13 are you?

14 A. Well, I think a lot of those studies would
15 have -- would bear relevance to the cytogenetic picture.
16 And that, you know, just about every one of the
17 myelodysplasias that have been linked to therapy have
18 some form of cytogenetic change, and that the vast
19 majority of those go on to develop AML.

20 Q. Do you really need this?

21 A. Yeah.

22 Q. For this case, MDS articles?

23 A. No. I need those for -- for me personally.

24 Q. For next week?

25 A. Yes.

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1 Q. Okay. Are you really relying on every one of
2 these articles for -- for your opinions in this case?

3 A. If I say yes you're going to keep my binder.
4 Is that why you're looking at me?

5 Q. No, I'm not going to do that. I'm going to ask
6 for a copy of it, but I'm not going to keep it.

7 A. There's plenty of literature to support this
8 notion that myelodysplasias and AML have cytogenetic
9 changes. Some of it is in that binder; some of it is in
10 a binder that I have at home on therapy-related AMLs.
11 In my view, that's unequivocal.

12 Q. You need these binders for a hearing next week
13 in a benzene case, correct?

14 A. I need some of them to prepare myself, yes.

15 Q. Is this one of them?

16 A. Yes.

17 Q. Will you agree after that hearing to get these
18 binders back to your lawyer, Mr. Gray, so he can get me
19 color copies of them?

20 A. You bet. How about if I put all those on a
21 disk and mail it to you?

22 Q. That would be great, but I'd like your copies
23 because you've got highlights and marks on them.

24 A. Okay.

25 Q. Thanks. So we've covered the MDS folder,

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1 right?

2 A. Uh-huh. This we've already gone through.

3 Q. Do you need this for next week?

4 A. Let me look at it real quick. If you take it,
5 I don't have to carry it back on the plane.

6 Q. This binder of benzene regulatory documents,
7 that I most likely am going to mark as Exhibit 5 to your
8 deposition, is this information that you relied upon
9 generally for your opinions about benzene?

10 A. Sure. I mean, not specifically. But, yeah, I
11 mean, they -- they've reviewed the same literature I've
12 reviewed. So, yeah.

13 (Deposition Exhibit Nos. 4-5 marked.)

14 Q. (By Mr. Lubel) Exhibit 4, do you remember we
15 went through these articles at the very beginning?

16 A. Uh-huh. (Witness answers affirmatively.)

17 Q. These were the articles that dealt with the
18 question I posed about the nonmalignancy effects at less
19 than or equal to 1 ppm exposures, correct?

20 A. Yes.

21 Q. Okay.

22 A. Yes.

23 Q. That's Exhibit 4.

24 A. Okay. This is the binder of NCI, all of the
25 publications that I've seen to date from that series.

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1 Q. Will you see if you can find the 2001 version,
2 where they talk about the exposures? I think it's in
3 the conclusion.

4 A. 2001? Who was the author?

5 Q. I think it's Hayes.

6 A. Here's 2001. Oh, uh-huh, yes. Yep.

7 Q. Thank you. Let me read this to you: It says,
8 "The NCI CAPM" -- that's the Chinese Academy of
9 Preventive Medicine, correct?

10 A. Yes.

11 Q. -- "study extended quantitative estimates of
12 risk to lower levels of exposure below 10 ppm." Do you
13 agree with that?

14 A. Say -- that they extended --

15 Q. "Quantitative estimates of risk to lower levels
16 of exposure below 10 ppm."

17 A. Do I agree that they did that?

18 Q. Yes.

19 A. I'm assuming they did, yes.

20 Q. Do you agree with that conclusion?

21 A. I didn't hear a conclusion in what you read.

22 Q. The study, this 2001 study, extended
23 quantitative estimates of risk to lower levels of
24 exposure below 10 ppm.

25 A. Extended quantitative estimates of risk. So

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1 are they saying that there is an elevated risk below 10
2 ppm? Is that what you're saying?

3 Q. That's the way I read it.

4 A. Okay. Well, I just read that they extended the
5 quantitative analysis. But, however --

6 MS. YATES: Do you mind handing him the
7 article?

8 A. Yeah. And then we can discuss this whole
9 series, if you'd like. Yeah, I don't read that the way
10 you read it. But I think that there is support for that
11 in here.

12 Q. (By Mr. Lubel) You find it in a different spot
13 than I do?

14 A. Well, I -- I just remember --

15 Q. Probably in a table?

16 A. -- I just remember Hayes saying something about
17 that. Yeah, I didn't -- I mean, it's not -- it's not
18 evident on this. And that's -- what my read of that is
19 that it's not 10 part per million years, but they're
20 saying that there were cumulative -- that they're --
21 that they started looking at less than 10 ppm as a
22 time-weighted average air concentration. Well...

23 (Pause.)

24 Q. That's your read?

25 A. Yeah.

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1 Q. Okay. Is there another page?

2 A. There's the first and then it looks like all of
3 the rest are just -- well, I have two copies, so we can
4 each have one.

5 Q. Can you scan us a duplicate of this file
6 folder, too?

7 A. Uh-huh, yes.

8 Q. This is called the NCI China?

9 A. NCI China.

10 Q. I take it you need to keep this.

11 A. I don't know that I'll need it. But, yes.

12 Q. Just in case?

13 A. Just in case. Just stick that back there where
14 that other one was. You can stick it in the back. That
15 would be fine.

16 MR. GRAY: Did you mark that as Exhibit 6?

17 MR. LUBEL: I'm not going to mark it. Mark
18 it means the court reporter has to take it home with
19 her.

20 THE WITNESS: Thank you.

21 A. Okay. And this is -- I definitely need this,
22 but you may want it.

23 This -- these are all of the studies that I
24 know of in vitro, in vivo, epidemiology that has looked
25 at chromosomal changes in people associated with benzene

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1 exposure.

2 Q. And this notebook, this big three-ring binder
3 is titled BZ, for benzene, Cytogenetics?

4 A. Yes.

5 Q. You'll get us a copy of that?

6 A. Yes.

7 Q. Thank you. I've got a folder over here,
8 Dr. Pyatt.

9 A. Okay.

10 Q. Actually, it's a redweld. Can I mark this?

11 A. I don't know what that is. Oh, this is the
12 stuff that I sent -- although, I've never seen this, so
13 I guess Mr. Gray just printed out the whole modeling
14 thing. This is what I sent him that was my file,
15 so-called, from the chem -- from the Liquid Wrench
16 simulation study.

17 Q. I'm going to mark that as Exhibit 6; is that
18 correct?

19 A. You can have it. I guess you have to send it
20 back to me, but I don't... (Pause.)

21 (Deposition Exhibit No. 6 marked.)

22 Q. (By Mr. Lubel) What you got next?

23 A. This is the Health Watch binder. It's kind of
24 like the NCI, only a different study.

25 Q. Are you going to need this?

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1 A. Uh-huh. (Witness answers affirmatively.)

2 Q. Can you get me a copy of it?

3 A. You bet.

4 Q. What about these notes that you have in here?
5 Are those cheat sheets?

6 A. Yeah, it looks like it.

7 Q. Is that for some crafty plaintiff's lawyer?

8 A. I don't -- I don't know. I guess you can read
9 them and make of it whatever you'd like.

10 Q. Well, just put it in the copy to us.

11 A. Okay. And then this is the same deal, but it's
12 the Pliofilm.

13 Q. We can get a copy of that from you, too?

14 A. Yes. This is Benzene and Cytopenias. Some of
15 the studies that we discussed that you pulled out came
16 from this binder.

17 Q. Do we need this for your opinions in the case?

18 A. I pulled out the ones that were the most
19 relevant.

20 Q. We'll let you take that home and not get a
21 copy.

22 A. Yeah, that's fine.

23 And this we've been going through and
24 discussing in some detail all of the AML and benzene
25 papers and, yeah, that's totally germane to this -- this

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1 issue as well as all of mine. So I can make a copy if
2 you'd like.

3 Q. Okay. Benzene and AML?

4 A. Yes.

5 Q. We can get a copy of that?

6 A. Yes. Okay. I think the last one -- oh, these
7 are refinery studies, epidemiology studies of various
8 refineries.

9 Q. Did you participate in any of these?

10 A. No.

11 Q. You're seeing the same cast of characters,
12 aren't you, in these?

13 A. You'll see through the publication the main
14 toxicologist from each -- from each company that are
15 involved.

16 Q. Like who used to be at Mobil?

17 A. Jerry Robb.

18 Q. Yep. You see him a lot, right?

19 A. He's published quite a bit, yes. I think he
20 collaborated with Otto Wong on some studies.

21 Q. Schnatter, did you know that he worked at
22 Exxon?

23 A. Rob Schnatter still works at Exxon..

24 Q. He's still there. You would agree that these
25 companies have an interest in the outcome of those

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1 studies, don't they?

2 A. Well, I think they have an interest in -- I
3 think anyone would have an interest in the outcome of
4 their studies. I don't believe that interest in any way
5 influences their interpretation of the data.

6 Q. They have a financial interest in the outcome
7 of those studies, don't they?

8 A. Oh, I would -- I would make the same claim. I
9 don't know that they have a financial interest, but I in
10 no way would think that their interest would influence
11 the interpretation of their data. I know Robb Schnatter
12 pretty well. He's a very honest scientist.

13 Q. How about the rest of these guys?

14 A. I don't know them as well.

15 Q. Would it surprise you if these industry-funded
16 studies, that one of their goals was to show that low
17 level benzene exposures could not cause disease so that
18 they would not have to worry about the refineries or
19 plants being reconfigured to reduce the benzene in
20 gasoline?

21 A. Okay.

22 MS. YATES: Objection; form.

23 MR. GRAY: Yeah, same objection.

24 A. What was the question?

25 Q. (By Mr. Lubel) Would it surprise you --

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1 A. Would I be surprised?

2 Q. Yeah. If these people that are doing these
3 industry-funded studies are starting out with the goal,
4 before the study ever gets off the ground, of proving
5 that benzene exposures at low levels can't induce
6 malignancies because their clients want them to protect
7 gasoline, the Holy Grail.

8 A. I don't know whether I'd be surprised or not.
9 But, yes, I think I would.

10 Q. You would be surprised, wouldn't you?

11 A. I don't think that's true.

12 Q. Okay. You are clearly, as a scientist, not
13 supposed to start out with a study with that in your
14 mind, are you?

15 A. Well, I think every scientist, no matter what
16 study they're about to embark on, will have some
17 preconceived notions, there will be preliminary data,
18 they'll have things that they are setting out to prove.
19 That's why you do a hypothesis. The hypothesis could
20 be: Are low levels of benzene hazardous? And it's yes
21 or no based on that data.

22 Q. So do you believe it's appropriate for these
23 authors of these studies, for them to start out with the
24 mental -- the mind-set that these are going to help
25 defend these companies in lawsuits, these studies are?

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1 MR. GRAY: Objection; form.

2 MS. YATES: Same objection.

3 A. I don't know about that.

4 Q. (By Mr. Lubel) Do you think it's appropriate
5 for the authors of these studies to start out at the
6 very beginning of the study with the goal of proving, by
7 virtue of the study results, that the benzene
8 regulations should not be altered?

9 MR. GRAY: Same objection.

10 MS. YATES: Same objection.

11 A. Well, I think that's -- that's just a really
12 negative way of viewing what they're doing. I mean, if
13 what they're doing is honestly trying to figure out if
14 there's health effects associated with those levels, and
15 if they're not, then that would impact on decisions
16 regarding the regulatory position, I think that's
17 absolutely appropriate.

18 Q. (By Mr. Lubel) Do you -- it would not surprise
19 you if the authors of these studies take the position
20 before they ever start the study that one of the goals
21 is to defend the companies that are funding the studies
22 in benzene lawsuits?

23 A. I don't know how to answer that.

24 MS. YATES: Objection; form.

25 Q. (By Mr. Lubel) Do you think it's appropriate

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1 or not?

2 A. Do I think it's appropriate for them to try to
3 defend their clients in a lawsuit or to design a study
4 to do that?

5 Q. To participate in a study where one of the
6 goals of the study is to help defend those very clients
7 in benzene lawsuits.

8 A. I think the -- the pertinent question is not
9 why the study has been done, but what data comes from
10 that study. I think it would be reprehensible to change
11 the interpretation of the data for whatever reason you
12 want to say it. The data is the data and it should be
13 interpreted that way. Why somebody conducts a study, I
14 have no idea why they conduct a study.

15 Q. And don't care?

16 A. Well, again, I think what I would care about is
17 if there was any evidence that there was bias in their
18 interpretation.

19 Q. What --

20 A. My view is simply by virtue of the fact that
21 the company paid for the study is not bias in terms of
22 the interpretation.

23 Q. Were you aware, sir, that these industry-funded
24 studies for these refineries, that they were done in
25 part to defend those companies in -- in lawsuits?

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1 MR. GRAY: Objection; form.

2 MS. YATES: Same objection.

3 A. I'm not aware of that, no. And a lot of those
4 studies in there are not industry sponsored. There are
5 many university studies in there. Maybe they were being
6 funded by the university -- I mean, by the company, but
7 they weren't employees of the company.

8 Q. (By Mr. Lubel) Like Dr. Irons, do you know
9 what his Shanghai Health Study, how much money he's
10 gotten from the industry?

11 A. I don't know.

12 Q. It's in excess of \$30 million. Would that
13 surprise you?

14 A. I think that's an expensive study.

15 Q. So is that industry funded, to your knowledge,
16 or not?

17 A. If what you're telling me is true, and I know
18 they've gotten significant funding from this consortium
19 of petroleum companies, I think that's fine. They are
20 the people that have a vested interest in these issues.
21 It's very hard to get government money to study benzene.
22 Very hard. I mean, I know a few investigators that can
23 do it, but a lot of people can't.

24 Q. Who's paying for the National Cancer Institute
25 studies?

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1 A. You and I.

2 Q. The taxpayers?

3 A. Correct.

4 Q. Why don't they have to get industry to fund
5 them?

6 A. Why doesn't the NCI have to get industry to
7 fund it?

8 Q. Right.

9 A. Well, because they have their own pile of money
10 that's given to them by the granting organization.

11 Q. So they're not biased like the industry
12 studies?

13 MS. YATES: Objection; form.

14 MR. GRAY: Objection; form.

15 A. I would disagree with that interpretation.

16 Q. (By Mr. Lubel) You disagree?

17 A. I disagree with your interpretation of simply
18 by virtue -- here's what I disagree with. That you
19 think -- not you personally -- but that you can tell
20 whether a study is biased simply by virtue of where the
21 money comes from. I absolutely disagree with that.
22 I've gotten government grants; I've gotten industry
23 sponsorship. My interpretation of the data and my
24 professional integrity as a scientist is not changed.

25 Q. And when have you ever written down on a piece

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1 of paper before you did the study for any industry where
2 you said one of the goals of the study is to defend you
3 in lawsuits?

4 MS. YATES: Objection; form.

5 A. I've never done that.

6 Q. (By Mr. Lubel) Why not?

7 A. Well, because I haven't done a study that that
8 was the purpose of.

9 Q. Yeah, I bet. When have you ever been involved
10 in a study where one of the goals was to make sure that
11 the regulations didn't become more stringent on your
12 clients?

13 MS. YATES: Objection; form.

14 A. Hasn't happened.

15 Q. (By Mr. Lubel) You haven't been asked to do
16 that before?

17 A. No.

18 Q. Can we get a copy of the epi studies on benzene
19 refineries?

20 A. Actually, you can have that if you'd like it.

21 Q. I can? I mean, you need a copy back? You need
22 a copy back?

23 A. Yeah. Yeah.

24 Q. I'm going to give it to your lawyer to give me
25 a copy. Is that all right? He's got broad shoulders.

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1 A. I guess he is my lawyer, but... (Pause.)

2 Q. Your lawyer. U.S. Steel's lawyer. Do you
3 understand who you're here for? You're not here for
4 Mr. Gray, are you?

5 A. No.

6 Q. You're here for U.S. Steel and Radiator
7 Specialty Company, correct?

8 A. Right. Okay. Okay. These are all of the
9 medical records that I shipped down here.

10 Q. Wait a second. Let me get these out of your
11 way.

12 A. Okay. So here are all of the medical records
13 that were shipped to me.

14 Q. Are you relying on any of these?

15 A. Well, only to the extent that I said what I
16 said about his disease.

17 Q. What did you say?

18 A. Well --

19 Q. That he had a diagnosis of leukopenia in '92?

20 A. Right.

21 Q. I mean, what else?

22 A. That's it.

23 Q. Okay.

24 A. And then here are the ones that I felt like
25 were pretty germane to the -- you know, that he didn't

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1 have lymphocytopenias, the timing, what Shirley Riggs
2 said about his cytopenias and all of that. That's more
3 of the blood work that I pulled out of this stack.

4 MS. YATES: What number is that?

5 (Deposition Exhibit No. 7 marked.)

6 Q. (By Mr. Lubel) So Exhibit 7 are the specific
7 medical records that you are relying upon, correct?

8 A. Yes.

9 Q. I'm going to put those over here.

10 A. Okay. Here is a list of letters, E-mails, I'm
11 not relying on any of that. If you don't want it, I
12 don't -- I don't want it. You know, when they send me
13 something, there's a letter attached. I thought I was
14 supposed to keep all of that.

15 Q. This is just your correspondence?

16 A. Correct. I guess now what I'm doing is trying
17 to respond to the notice and give you what was in that
18 notice.

19 (Deposition Exhibit No. 8 marked.)

20 Q. (By Mr. Lubel) So No. 8 is your correspondence
21 with Mr. Gray and the other lawyer?

22 A. And Miss Yates, yes.

23 Q. Which one of them hired you?

24 MS. YATES: I'm relegated to "the other
25 lawyer."

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1 Q. (By Mr. Lubel) Which one of them hired you?

2 A. I think originally I talked to Miss Yates.

3 Q. Okay. Which one do you have the agreement
4 with?

5 A. What do you mean, "agreement"?

6 Q. You didn't come here for free.

7 A. You mean the paying agreement?

8 Q. Right.

9 A. Well, here's my invoice. I think I sent it
10 to -- I sent it to Mr. Gray.

11 Q. You sent it to Mr. Gray?

12 A. Yes.

13 Q. But do you understand that they're both paying
14 you?

15 A. Yes.

16 Q. So can we just get the invoices and mark them
17 as 9?

18 A. Sure. That's why I brought them.

19 Q. It's just one?

20 A. Yes.

21 Q. There will be another one after today?

22 A. You bet.

23 Q. Are you going to charge them double?

24 (Deposition Exhibit No. 9 marked.)

25 Q. (By Mr. Lubel) Do you have a resume somewhere?

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1 A. I do. Right here.

2 Q. Is it up to date?

3 A. Yes.

4 (Deposition Exhibit No. 10 marked.)

5 Q. (By Mr. Lubel) That's 10, right?

6 A. Okay. 10, yeah.

7 Q. What do you got next. List of cases?

8 A. I think I'm up to 11 now.

9 Q. Okay.

10 (Deposition Exhibit No. 11 marked.)

11 A. This is all of the stuff that I've reviewed.

12 Q. (By Mr. Lubel) We'll make that Exhibit 12.

13 (Deposition Exhibit No. 12 marked.)

14 A. I don't really know what this is -- oh, this is
15 just miscellaneous deposition testimony and --

16 Q. (By Mr. Lubel) You're not relying on any of
17 that stuff, are you?

18 A. Legal stuff.

19 Q. You're not on a fact-finding mission here, are
20 you?

21 A. What do you mean?

22 Q. In other words, you're not reading all of the
23 depositions of the witnesses on how much he was exposed
24 to whatever, things like that?

25 A. No.

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1 Q. You're not here to say those kind of things;
2 you're going to rely on experts for that?

3 A. Correct. I think that's it. I think that's
4 all I brought.

5 Q. What is this?

6 A. Oh, these are all my papers. All the ones --
7 and you can have this.

8 Q. All of your papers?

9 MR. GRAY: All of the ones --

10 A. This is a CD of all the references from my
11 report.

12 Q. (By Mr. Lubel) Oh, really? How do you do
13 that?

14 A. How do I do what? Make a CD of them?

15 Q. Yeah.

16 A. I just went through my report and went through
17 my electronic files and pulled them over. As I went
18 through the bibliography I just made sure that -- there
19 might be one or two missing, because I'm not that good
20 at it. But the vast majority of what I've referenced in
21 my report is on that CD.

22 Q. Thanks. Here's some notes --

23 A. Oh, that's -- yeah, that's just scrap stuff.

24 Q. Is that notes? That's on dermal? These are
25 all dermal notes?

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1 A. Yeah, some of them. Some of them came out
2 of -- this is -- these were just left over from -- from
3 other papers that we pulled out of the binder that I
4 need to stick back in.

5 Q. Okay. What's this one? This is a note file?

6 A. Just -- just general -- it's been flopping
7 around on a disk.

8 Q. Do you mind if I look at it?

9 A. Yeah.

10 Q. Your handwriting is better than most doctors'.
11 Why is that?

12 A. I don't know. I haven't ever heard that
13 before.

14 Q. Are you sure you went to school?

15 Okay. So we'll mark your notes as 13.

16 (Deposition Exhibit No. 13 marked.)

17 Q. (By Mr. Lubel) You don't have a written
18 agreement with U.S. Steel's lawyers or Radiator's,
19 correct?

20 A. You mean like a retainer?

21 Q. Just -- right.

22 A. No, I don't ever do that.

23 Q. Have you done work for them before?

24 A. Yes.

25 Q. What type of cases?

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1 A. What was the disease?

2 Q. What -- was it Liquid Wrench cases or something
3 else?

4 A. Yes, Liquid Wrench.

5 Q. Do you remember what cases they were?

6 MR. GRAY: Can I stop for a second and
7 think about whether or not I've retained him in cases he
8 hasn't been disclosed in?

9 MR. LUBEL: Yeah. Do you want to take a
10 break and figure it out and come back?

11 MS. YATES: Yeah.

12 MR. LUBEL: We'll leave you. How about
13 that?

14 MR. GRAY: Okay.

15 (Short recess from 2:52 to 2:55 p.m.)

16 EXAMINATION (Continuing)

17 BY MR. LUBEL:

18 Q. Were you able to answer the question?

19 A. The -- the other cases?

20 Q. Yes, sir.

21 A. There was one a while back when I actually was
22 at ChemRisk. So this was 2003, and Jeff Kemp was the
23 client and it was a Liquid Wrench case. And it was
24 Craig. So I worked on that one.

25 Q. It's a Craig case?

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1 A. Yeah. I wrote a report.

2 Q. Jeff Kemp?

3 A. Jeff Kemp, yes.

4 Q. Was he at Fulbright & Jaworski at that time,
5 working for Steve Dillard?

6 A. He's in the Dallas office, as I recall. But I
7 suspect Steve had something to do with it.

8 Go ahead. Do you have another question?

9 Q. Any other cases?

10 A. Akins. I worked on the Akins case.

11 Q. Is that over?

12 A. My understanding is, yes, it's over.

13 Q. Okay. That was a Liquid Wrench case?

14 A. Yes. And I think that's it. Stephenson.
15 Stephenson. But I wasn't designated as an expert is my
16 understanding.

17 MR. LUBEL: We got that.

18 MR. GRAY: He was designated and now he's
19 not currently designated, so I prefer you not ask him
20 about that case.

21 Q. (By Mr. Lubel) What oil companies have you
22 done work for in benzene?

23 A. Law firms?

24 Q. Oil companies.

25 A. Oil companies. I did some work on the Sugar

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1 Creek Refinery cases. And I think that was exclusively
2 BP Amoco. And it was through Maron & Marvel. Yeah,
3 that law firm.

4 I've done a little work with Larry Riff,
5 and I think he predominantly represents Chevron. Seems
6 like I had another case; that will be on there, that
7 list, because I was deposed in it.

8 Q. Tibani?

9 A. No, I've worked with Tibani. And that was
10 Exxon. And then there was a hearing that I went to in
11 Corpus Christi, a Havner hearing, and there were three
12 or four smaller companies. Sun --

13 Q. Sunco, Sun Oil Company?

14 A. Yeah, that and --

15 Q. Koch?

16 A. I've seen Koch around. And I've been around --
17 I've talked to a couple of people about some cases that
18 Koch was sort of a defendant, but it never went to
19 anything.

20 Q. You were there with Robbshaw?

21 A. Yes.

22 Q. Jim Robbshaw?

23 A. Yes. Yes. Was Koch -- was he Koch? I can't
24 remember, but... (Pause.)

25 Q. I think Koch was part of it. Flint Hills?

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1 A. Yes, Flint Hills was there, yes.

2 Q. That was a CLL case that you testified in?

3 A. NHL.

4 Q. NHL. And the Court permitted the opinion that
5 benzene can cause NHL in that case. Do you recall that?

6 A. I think what the Court said was that they would
7 allow Dr. Infante to testify to that effect, but that he
8 was going to be limited to only two papers instead of
9 the 200 or so that he had in his report.

10 Q. And did that satisfy you?

11 A. Based on the Havner, you know, however you
12 interpret that, I was -- I was fine with it.

13 Q. Is this article relevant to your opinions
14 (indicating)?

15 A. No. This -- this article discusses the -- has
16 some literature in it discussing the timing between a
17 therapy-related myelodysplasia and how long it takes for
18 AML to appear. And that's usually a very short time
19 period. We've already talked about that.

20 Q. Does the liver metabolize the chemotherapeutic
21 agents the same way it does benzene?

22 A. That's another very good question. It depends
23 a lot on the chemotherapeutic agent. Cyclophosphamide
24 as one seems to go through a metabolic pathway pretty
25 similar to benzene. In fact, we did some work back in

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1 the '90s when we were looking at cyclophosphamide as a
2 way to validate the pharmacokinetic modeling for benzene
3 because we could measure metabolites at low levels in
4 the bone marrow. So in experimental animals, how much
5 we put in and then how much of the metabolites showed up
6 in the bone marrow, which for benzene is a lot harder to
7 do. So that one appears to be, yes.

8 Things like Cisplatin, no. It's going
9 through a totally different -- totally different
10 process.

11 Q. How about topo?

12 A. I don't know that much about their metabolic
13 pathway. My understanding, sitting here right now, is
14 that the metabolism of the topo drugs deactivates them,
15 so that makes it almost exactly opposite from benzene.

16 Q. Do you know what the enzyme is?

17 A. The enzyme that deactivates topo?

18 Q. Correct.

19 A. Maybe 2D, cytochrome P450 2D. But I'm guessing
20 there. I don't know.

21 Q. Do you have the same enzymes for the -- for the
22 various chemotherapeutic agents involved in metabolism
23 that you do in benzene?

24 A. To a large extent, yes.

25 Q. Complete overlap?

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1 A. Well, there's plenty -- I mean, you don't have
2 that many metabolic enzymes, so they have a ton of
3 substrate promiscuity, if you will. They can work on a
4 lot of different chemicals. 2E1 tends to be the one
5 that benzene -- at least its primary oxidation step goes
6 through 2E1. Cyclophosphamide, to some extent, goes
7 through there. But others do its early oxidation step,
8 but then that result is the same.

9 Q. Are the -- are the metabolites the same for the
10 different chemotherapeutic agents as it is for benzene?

11 A. The metabolites?

12 Q. Right.

13 A. No. I mean they're not -- no, it's a different
14 set of drugs. But if you're looking, say, at a
15 parabenzquinone or a paraquinone, the structure for
16 some of the metabolites in these other chemotherapeutic
17 agents are the same. I mean, you might have a bigger
18 molecule, but you're going to have a functional group
19 which is the same as parabenzquinone that comes from
20 benzene. You can have hydroquinone structures on other
21 metabolites.

22 Q. Let's -- let me break this down for a second.

23 Benzene is not a chemotherapeutic agent
24 anymore, correct?

25 A. No longer, no.

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1 Q. And you're not referring -- when we refer to
2 chemotherapeutic agents today, you're not speaking about
3 benzene.

4 A. No.

5 Q. Right?

6 A. No.

7 Q. Correct?

8 A. Yes, that's correct. I'm sorry. That's
9 correct.

10 Q. If we were to look at the chemical structure of
11 the various chemotherapeutic agents, how many are we
12 talking about? How many different cocktails or drugs
13 are we referring to?

14 A. 20.

15 Q. Are any of those 20 going to have the same
16 chemical structure as benzene?

17 A. Probably not.

18 Q. Now, then they enter the body and they're both
19 metabolized by the liver, correct?

20 A. Yes.

21 Q. That part's the same, right?

22 A. Yes.

23 Q. And then what do we call what's left over after
24 the -- it's metabolized by the liver? We call that
25 metabolites?

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1 A. Yes.

2 Q. When we look at the 20 or so chemotherapeutic
3 agents and we look at their metabolites, are they
4 exactly the same as the benzene metabolites?

5 A. There will be a lot of similarities in terms of
6 the chemical reactivity. But, no, they're not going to
7 be the same.

8 Q. Has anybody done an analysis of those, the --
9 this -- the dissimilarity between those metabolites and
10 what, if any, impact that would cause?

11 A. I'm not -- not that I've seen. I don't think
12 there's ever been any -- you know, any real interest to
13 do that. They are all very different. I mean, the
14 chemotherapeutic agents are structurally very different.
15 They share some common chemical characteristics, like
16 they're all considered alkylating agents. And it's
17 believed that that alkylation and the crosslinking of
18 DNA is part of their mechanism.

19 Q. So would you think you can do -- although
20 you've acknowledged that you're going to study it more,
21 is that you can take the category of, like, alkylating
22 agents, right?

23 A. Uh-huh. (Witness answers affirmatively.)

24 Q. And study that group, right?

25 A. Okay.

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1 Q. And then you've got -- what's another category
2 of chemotherapeutic agents?

3 A. Topoisomerase inhibitors.

4 Q. Topos, right?

5 And then you can compare those to benzene
6 and you hope that your further -- your study or
7 whatever, will tell you more?

8 A. Well, what we were talking about and what I
9 think would be an interesting question is how the
10 dosing -- how the -- how the quantitative dose would
11 compare up. You know, if you take 200 part per million
12 year or 400 part per million year, whatever dose you
13 accept as potentially being leukemogenic, how does that
14 compare with the dose of --

15 Q. Is there a particular category of the
16 chemotherapeutic agents that you have a belief is more
17 like benzene, like the topos, the alkylating agents, or
18 do you have an opinion one way or the other?

19 A. Well, I think if you look at the overall
20 pathological characteristics, it seems to fall into the
21 alkylating agents more closely than the topoisomerase
22 drugs. You see the same types of cytogenetics. You see
23 the same morphologic characteristics of the disease, et
24 cetera. So I think that one is generally accepted to be
25 how -- well, not necessarily, how benzene is working,

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1 but a consistent picture of the pathology.

2 Q. Well, whether it's because it's similar to
3 benzene or whether it's just a coincidence, you're
4 seeing similarities of end results?

5 A. We are. I would be hard pressed to accept that
6 it's just a coincidence.

7 Q. Well, you don't need any studies that prove
8 it's not, do you?

9 A. I don't need any studies that --

10 MR. GRAY: Objection; form.

11 MS. YATES: Objection; form.

12 A. I don't understand that question.

13 Q. (By Mr. Lubel) You don't have any studies that
14 show it's something more than a coincidence because we
15 don't have any studies that establish the comparison of
16 the doses?

17 A. Well, I don't -- that's almost irrelevant. I
18 mean --

19 Q. The dose?

20 A. To this question. I think that would be an
21 interesting question. But what I'm talking about is
22 when you're looking at the end result, you're looking at
23 the pathological characteristics of diseases that you
24 have attributed to exposure to alkylating agents or that
25 you have attributed to exposure of benzene as well as

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1 some in vitro characterizations, responses, there are
2 similarities.

3 Q. But you're comparing a completely different
4 chemical structure, these chemotherapeutic agents, to
5 benzene, right?

6 MR. GRAY: Objection; form.

7 MS. YATES: Same objection.

8 A. I wouldn't say they are completely different,
9 but --

10 Q. (By Mr. Lubel) They are different?

11 A. They are definitely different. And the
12 topoisomerase drugs are different.

13 Q. If I showed up to have a debate with you and
14 said, You know what? Can we compare -- I'm just going
15 to make something up -- all of the aromatic chemicals
16 together? There is a bunch of them, aren't there?

17 A. Yes.

18 Q. And we're going to group them as if they're all
19 alike. You would look at me like I had a third eye,
20 wouldn't you?

21 A. Well, I would tell you that that's
22 scientifically inappropriate to do that.

23 Q. Well, that's what I'm telling you. I think it
24 seems like it's scientifically inappropriate to look at
25 chemotherapy agents as a group and compare it to benzene

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1 when the studies haven't been done to say it's apples to
2 apples.

3 A. I would disagree with that. I think you can do
4 that.

5 Q. Why?

6 A. Because the studies have been done. And that's
7 what we're talking about. We're looking at the way the
8 disease presents. We're looking at the cytogenetics
9 associated with the exposure to those chemicals. We're
10 looking at the way the disease responds to treatment.
11 We're looking at the relapse rate. All of those
12 things -- this is not me, you know; there's many people
13 who have made these same observations that benzene seems
14 to be acting as if it's an alkylating agent. The
15 diseases are very similar.

16 Q. And do we know if that's by chance?

17 A. Well, I don't think it's by chance.

18 Q. Do we know? Has it been answered?

19 A. I think I am answering it.

20 Q. Has it been answered in a study that you can
21 look at a different chemical structure, like these
22 chemotherapeutic agents, and say that it acts the same
23 way as benzene? They are alike, the same thing?

24 A. I don't think anyone would say they are the
25 same thing. And I think most people would argue that

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1 maybe they're not even acting the same way, because of
2 questions you asked earlier, that they're going through
3 different metabolic pathways, but the end result is
4 apparently the same. The end result for a lot of
5 different chemicals in humans, bone marrow toxicity will
6 respond as a leukemogenic response. And you can get AML
7 from topoisomerase drugs. You can get AML from
8 chemotherapy. You can get AML from ionizing radiation.
9 Those are all very different. So there is a similar
10 response in the bone marrow to a variety of different
11 toxic agents.

12 Q. Are you saying that they both attack the bone
13 marrow the same way?

14 A. There are some similarities, but there are
15 going to be some differences as well.

16 Q. What are the differences?

17 A. Between what?

18 Q. Benzene and ionizing radiation.

19 A. Well, ionizing radiation doesn't have to be
20 metabolized. It's likely to damage any kind of cell.

21 Whereas, benzene is going to be
22 predominantly toxic to more rapidly dividing cells.
23 There are other differences but that one.

24 Q. What's the difference between benzene and the
25 various chemotherapeutic agents, how they attack the

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1 bone marrow?

2 A. Well, I mean, I think that -- that the notion
3 that the alkylating agents are crosslinking DNA and the
4 cell dies when it divides and therein is how they're
5 using it to treat chemo or primary malignancies --
6 hydroquinone and parabenzoquinone, the metabolites of
7 benzene, have not been shown to be an alkylating agent.
8 So it's doing something different. It's not acting at
9 that same pathway. There is some in vitro data and some
10 argument that benzene and/or it's metabolites can act as
11 a topoisomerase inhibitor, but no one has shown that in
12 vivo, so it may not be acting through that mechanism
13 either. We just don't know enough to be able to answer
14 that question.

15 Q. And the chemotherapeutic drugs cause -- always
16 cause abnormal chromosomes?

17 A. 95 percent of the time.

18 Q. What's chromosome abnormalities?

19 A. That depends on the class.

20 Q. Class of what?

21 A. The class of chemotherapeutic agent that you're
22 referring to. The alkylating agents will typically
23 cause 5 or 7. The topoisomerase drugs you will get a
24 balance translocation at 11Q23, others. So it just
25 depends, really, on what class of drug you're talking

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1 about.

2 Q. But they don't always cause abnormal
3 chromosomes, correct, either class?

4 A. Within 5 percent might not. And even there --
5 I mean, there are some investigators, Pederson-Bjergaard
6 from Sweden, who's probably published the most on
7 therapy-related leukemia. I mean, he has said that if
8 it doesn't have a chromosomal change, it's probably not
9 related to chemo. It's probably just a coincidence that
10 they got leukemia.

11 Q. Is the -- what you called the etiological
12 window different for the different chemotherapeutic
13 agents?

14 A. It falls within that same category, which is
15 why I believe that it's all consistent one to the other.
16 Topoisomerase drugs, for whatever reason, tend to cause
17 their leukemias quicker.

18 Q. What time period?

19 A. Well, I've seen it as early as six months. And
20 then probably the risk peaks at about three to five
21 years.

22 Q. How about the alkylating agents?

23 A. The risks will start increasing at about two,
24 they peak at about five to seven, and then by ten years
25 out it's pretty much over.

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1 Q. And why is there a difference in these windows
2 for the different classes of chemo?

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

4 Q. Have we covered your opinions today with --
5 have you gone through the materials that form the basis
6 of them?

7 A. Yes, in detail.

8 Q. Have I been courteous to you?

9 A. You have. Thank you.

10 MR. LUBEL: Thanks for your time.

11 MR. GRAY: I've got, like, three quick
12 little questions.

13 THE WITNESS: Oh, yeah.

14 MR. LUBEL: I'll keep you here longer if he
15 does it. I'm kidding.

16 MR. GRAY: I may have misheard this.

17 EXAMINATION

18 BY MR. GRAY:

19 Q. I think in one of the questions and answers
20 there may have been a statement about pancytopenia
21 occurring in '92 or '93. The records actually said it
22 was leukopenia.

23 A. No, I didn't -- if I said that, I misspoke.
24 Yeah, it was diagnosed as leukopenia with a subtle
25 hypocellularity in the marrow. The pancytopenia didn't

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1 show up until later.

2 Q. And I flagged a couple --

3 A. I think it was in '98 is when they started
4 actually using that term.

5 Q. Okay. That was all for Exhibit 7.

6 This last article that Mr. Lubel asked you
7 about.

8 A. Yes.

9 Q. That you brought to the deposition?

10 A. Yes.

11 Q. Therapy-Related Myelodysplasia Syndrome. I'm
12 just going to mark that as Exhibit 14, if that's all
13 right.

14 (Deposition Exhibit No. 14 marked.)

15 A. Yes.

16 Q. (By Mr. Gray) There's been discussion of
17 myelodysplasia. Did Dr. Levy, in his report or in his
18 deposition, say that he believed that Mr. Oakley had
19 myelodysplasia?

20 A. Not that I saw, no.

21 Q. Okay. If Dr. Levy were to give that opinion,
22 either in an amended report or at trial, would you want
23 to have an opportunity to respond to those opinions?

24 A. Yes.

25 Q. Okay.

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1 MR. LUBEL: Why?

2 MR. GRAY: Well, I'll ask it this way.

3 Q. (By Mr. Gray) Did you specifically address
4 myelodysplasia in your -- in your report?

5 A. I did not, no.

6 MR. LUBEL: Well, let me --

7 MR. GRAY: Hang on.

8 MR. LUBEL: I want to talk to you. I'm not
9 asking a question.

10 MR. GRAY: Let's go off the record.

11 MR. LUBEL: Yeah, that's fine.

12 (Discussion off the record.)

13 EXAMINATION (Continuing)

14 BY MR. GRAY:

15 Q. The various chemotherapeutic drugs that have
16 been historically used that may cause leukemia, are
17 those drugs used at varying doses?

18 A. There are certainly clinical trials ongoing all
19 the time when they are varying the doses of the various
20 components in these cocktails and trying to figure out
21 which one works the most. They never will drop the dose
22 down below what they consider to be cytotoxic level or
23 the patient is going to die from the primary malignancy.
24 So there is some play in that. They're increasing this
25 one and decreasing this one and introducing a new one.

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1 But there is always going to be sort of a baseline where
2 they -- they believe it's going to be treating the
3 primary disease.

4 Q. Okay. But are there -- are there some
5 chemotherapeutic agents which are cytotoxic at one level
6 as where other chemotherapeutic agents are cytotoxic at
7 a higher level?

8 A. Oh, sure. The potencies are very different.

9 Q. Okay.

10 A. Potencies are different, yeah.

11 Q. And the actual amounts that are used are very
12 different in treating patients?

13 A. Yes.

14 Q. Okay.

15 A. Yes. But that's going to depend on -- they
16 base it on weight and age and types of disease. I mean,
17 we're talking as if all of the drugs treat all the same
18 diseases the same way, and that's not the case. You
19 will see AML risk associated with breast cancer and that
20 is treated with a different cocktail of drugs than if
21 the AML risk is associated with multiple myeloma
22 treatment.

23 Q. Okay. The latency that you find between
24 leukemia and chemotherapeutic agents is fairly
25 consistent even though different doses of those

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1 different chemotherapeutic agents are being used?

2 MR. LUBEL: Objection; form.

3 A. There doesn't -- there isn't much variability
4 there. I mean, it falls within the window. In fact, I
5 haven't seen a dose-dependent change in the latency,
6 ever. But that's probably because the latency is not --
7 you know, it's not 2.3 years and that's when you get
8 leukemia. It's somewhere within two to five years is
9 when the risk starts increasing. So you may not be able
10 to detect those subtle differences. But, no. The
11 answer to your question is no, the latency doesn't vary
12 with the dose.

13 MR. GRAY: No further questions.

14 FURTHER EXAMINATION

15 BY MR. LUBEL:

16 Q. Isn't chemotherapy typically injected into
17 patients?

18 A. Yes. Not all, but most.

19 Q. Does that exposure pathway differ from people
20 exposed to benzene, typically?

21 A. Well, sure. I don't think anyone is exposed to
22 a benzene IV, but --

23 Q. I hope.

24 A. Yeah, it's going to be the same. Once it gets
25 in the systemic circulation, it's going to go around

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1 once and go to the liver. So whether it's an IV -- you
2 know, these -- these drugs -- these chemotherapy drugs
3 are just really toxic.

4 Q. It goes back to your point, whether it gets
5 into your blood by virtue of breathing it, ingesting it,
6 injecting it into somebody's blood or exposing their
7 skin to it, it's going to have the same effect, in your
8 opinion?

9 A. Once it's systemically absorbed, then it should
10 all be the same.

11 MR. LUBEL: Thank you.

12 MR. GRAY: We're done.

13 THE REPORTER: Do you want to read and
14 sign?

15 THE WITNESS: Yes.

16 (Deposition concluded at 3:24 p.m.)

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CHANGES AND SIGNATURE

WITNESS NAME: DAVID PYATT, PH.D. DATE: November 7, 2008

PAGE	LINE	CHANGE	REASON

I, DAVID PYATT, PH.D., have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

DAVID PYATT, PH.D.

THE STATE OF _____)
COUNTY OF _____)

BEFORE ME, _____, on this day personally appeared DAVID PYATT, PH.D., known to me (or proved to me under oath or through _____) (description of identity card or other document) to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that they executed the same for the purposes and consideration therein expressed.

Given under my hand and seal of office this _____ day of _____, _____.

NOTARY PUBLIC IN AND FOR
THE STATE OF _____
COMMISSION EXPIRES: _____

Dr. David Pyatt
11-7-2008

1
2 IN THE UNITED STATES DISTRICT COURT
3 FOR THE EASTERN DISTRICT OF TEXAS
4 MARSHALL DIVISION

5)
6 ROBERT B. OAKLEY and)
7 IRENE OAKLEY,)
8) CIVIL ACTION
9 Plaintiffs,)
10 VS.) NO.: 2:07-CV-351
11)
12 AIR PRODUCTS AND)
13 CHEMICALS, INC.,)
14)
15 Defendants.)

16 REPORTER'S CERTIFICATION

17 ORAL DEPOSITION OF DAVID PYATT, PH.D.

18 November 7, 2008

19 I, SHAUNA L. BEACH, RDR, CRR, Certified Shorthand
20 Reporter in and for the State of Texas, hereby certify
21 that to the following:

22 That the witness, DAVID PYATT, PH.D., was duly
23 sworn by the officer and that the transcript of the oral
24 deposition is a true record of the testimony given by
25 the witness;

There was a request for examination and signature
of the witness to the deposition transcript. The
original transcript was sent to the witness for review
on .

I further certify that I am neither counsel for,
relate to, nor employed by any of the parties or

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11-7-2008

1 attorneys in the action in which this proceeding was
2 taken, and further that I am not financially or
3 otherwise interested in the outcome of the action.

4 Certified to by me this 8th day of November,
5 2008.

6
7 SHAUNA L. BEACH, RDR, CRR
8 Certified Shorthand Reporter
9 in and for the State of Texas
10 Certification No. 8408
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