SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

CENTRAL CIVIL WEST

LARRY THORPE AND JOANNE THORPE,	)	
Plaintiffs,	)	
vs.	)	No. BC348287
SAFETY-KLEEN SERVICES, INC., et al.,	)	
Defendants.	)	
AND CONSOLIDATED CASES.	)	

DEPOSITION OF

DAVID W. PYATT, Ph.D.

LONG BEACH, CALIFORNIA

DECEMBER 17, 2008

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FILE No.: A2092C4

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FOR THE COUNTY OF LOS ANGELES

CENTRAL CIVIL WEST

LARRY THORPE AND JOANNE THORPE, Plaintiffs, vs. SAFETY-KLEEN SERVICES, INC., et al., Defendants. AND CONSOLIDATED CASES.	No. BC348287

DEPOSITION of DAVID W. PYATT, Ph.D., taken on behalf of the PLAINTIFFS, at 401 East Ocean Boulevard, Suite 800, Long Beach, California, commencing at 9:05 a.m., on Wednesday, December 17, 2008, before Sheri A. Ply, CSR No. 6507, RPR.

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I N D E X

WITNESS: DAVID W. PYATT, PH.D.

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INFORMATION REQUESTED:
(NONE)

QUESTIONS WITNESS INSTRUCTED NOT TO ANSWER:
(NONE)

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LONG BEACH, CALIFORNIA, WEDNESDAY, DECEMBER 17, 2008

9:05 A.M.

* * *

09:06:27

THE VIDEOGRAPHER: I am Dan Ackley, your videographer, and I represent Atkinson-Baker Court Reporters in Glendale, California. 09:06:46 09:06:47 09:06:50

I am not financially interested in this action nor am I a relative or employee of any attorney or any of the parties. Date is December 17th, year 2008. Time is approximately 9:06 a.m. 09:06:52 09:06:54 09:06:58 09:07:01

This deposition is taking place at the Metzger Law Group, 401 East Ocean Boulevard in Long Beach, California. 09:07:03 09:07:06 09:07:10

This is case number BC348287 entitled Thorpe versus Safety-Kleen. Deponent is Dr. David Pyatt. This is Volume 1, beginning of tape 1. Your court reporter is Sheri Ply. 09:07:10 09:07:16 09:07:20 09:07:22

Would counsel present please introduce themselves. 09:07:24 09:07:26

MR. METZGER: Good morning. My name is Raphael Metzger. I represent the plaintiffs in the Safety-Kleen benzene leukemia cases. 09:07:26 09:07:28 09:07:32

MR. FOLEY: Good morning, Patrick Foley for defendant Safety-Kleen Systems, Inc. 09:07:34 09:07:36

MR. CARR: Chris Carr for Safety-Kleen. 09:07:39

MS. KELLY: Laura Kelly for BC Stocking 09:07:40

Distribution. 09:07:42

THE VIDEOGRAPHER: Please swear in the witness. 09:07:44

09:07:44

DAVID W. PYATT, Ph.D., 09:07:44

having been first duly sworn, was examined 09:07:44

and testified as follows: 09:07:44

09:07:44

EXAMINATION 09:07:44

BY MR. METZGER: 09:07:45

Q Good morning, doctor. 09:07:53

A Hi. 09:07:55

Q Would you introduce yourself, please. 09:07:56

A David Pyatt. 09:07:58

Q And what type of a doctor are you? 09:07:59

A I am a toxicologist. 09:08:01

Q Okay. You are not a medical doctor; correct? 09:08:03

A I am not. 09:08:05

Q Okay. You have been designated as an expert by 09:08:06

Safety-Kleen. And you have testified as an expert 09:08:13

witness before, have you not? 09:08:16

A I have, yes. 09:08:19

Q Okay. You are comfortable with the procedures 09:08:20

so that I don't need to review them with you? 09:08:26

A No. 09:08:28

Q You are not or you are? 09:08:29

A I'm sorry. Yes, I am comfortable with them. 09:08:31

Q All right. I will show you what has been 09:08:33
marked as Exhibit 1. 09:08:35

(Deposition Exhibit 1 was marked for 09:08:38
identification.) 09:08:38

Q BY MR. METZGER: And is that a copy of your 09:08:39
curriculum vitae? 09:08:41

A Yes. It is a little dated. There have been a 09:08:42
couple of changes; nothing substantial. 09:08:45

Q Do you have a more current curriculum vitae 09:08:48
with you? 09:08:51

A I might. 09:08:51

Q Could you retrieve that and we will mark that 09:08:52
as an exhibit? 09:08:54

A Yeah. I will have to disconnect myself. 09:08:55

Q That's fine. Sure. 09:08:57

A Can I get in behind you? 09:08:58

MR. FOLEY: Sorry. 09:09:01

THE WITNESS: If not, I probably can just tell 09:09:02
you what's different. 09:09:04

Q BY MR. METZGER: While you are going through 09:09:09
stuff, if you happen to have a case list, that would 09:09:11
also be efficient. 09:09:15

A Yes, I do. 09:09:16

Q You might just keep that folder on the table. 09:09:17

A Okay. 09:09:20

Case list. 09:09:51

Q Okay. 09:09:52

A Mr. Metzger, I think I left it at home so we can just use that one. 09:10:07 09:10:10

Q All right. 09:10:12

A I am not seeing it. 09:10:14

Q Okay. 09:10:15

A Sorry. 09:10:16

Q Well, then let's use Exhibit 1. And if you would tell me, first of all, what needs to be added to this to bring it current? 09:10:17 09:10:20 09:10:24

A Well, what I noticed was on the academic appointment, the Biometrics and Preventative Medicine, School of Medicine, that has now been rolled into the Colorado School of Public Health, which was officially inaugurated this summer and that is where my appointment has moved to, the School of Public Health. 09:10:26 09:10:28 09:10:34 09:10:39 09:10:43 09:10:47

Q Okay. 09:10:50

A And that is what made me think that this one might be a little bit -- a little bit dated. 09:10:51 09:10:54

Q Okay. 09:10:57

A And there is at least two papers that I 09:11:01

published since this one was -- this CV was generated. 09:11:06

Q What are they? 09:11:11

A There was one on interpretation of 09:11:13
biomonitoring data, kind of a follow up to this 09:11:17
"Regulatory Tox Pharma" in 2007. 09:11:24

And then there was one on formaldehyde and 09:11:28
potential modes of action for formaldehyde with regard 09:11:38
to hematopoietic malignancies that was published in 09:11:42
2008. 09:11:49

Q Were both of those articles published in 2008? 09:11:50

A Yes. 09:11:53

Q The first one regarding the interpretation of 09:11:53
biomonitoring data, in what journal was it published? 09:11:55

A I think they published it in the journal 09:12:01
"Biomonitoring." I wasn't the first author. The other 09:12:03
one was published in "Regulatory Tox and Pharm," and I 09:12:08
am the first author on that one. 09:12:11

Q Who is the first author on the biomonitoring 09:12:15
article? 09:12:17

A Pretty sure it is Sean Hays, H-a-y-s. 09:12:18

Q Does that article concern benzene at all? 09:12:27

A No. 09:12:29

Q Does it concern chromosome abnormalities in 09:12:33
secondary leukemias? 09:12:43

A No. 09:12:50

Q Okay. Is there anything else you would need to add to your CV to bring it complete and current? 09:12:50 09:12:53

A I don't -- nothing of -- nothing of substance. Yeah, it looks like the last abstract was in 2007. So I went to SOT, I went to SRA. SOT is -- you know what that is, Society of Toxicology, Society of Risk Assessment in 2008, presented abstracts, but I don't remember exactly what they were. 09:13:01 09:13:07 09:13:12 09:13:19 09:13:21 09:13:25

So yeah, this one is about six months behind. 09:13:29

Q Okay. Any of these abstracts that you presented at these meetings, did any of them concern benzene or leukemia? 09:13:33 09:13:36 09:13:40

A No. Well, it might have concerned leukemia because the formaldehyde work was trying to understand potential mechanisms by which formaldehyde could damage the bone marrow, if that happens. So it was related to kind of a chemical process in the marrow. 09:13:41 09:13:43 09:13:49 09:13:54 09:13:56

The biomonitoring data is not -- that's simply a method by which you can interpret biomonitoring data as it is being published by CDC or whoever is putting the data out. 09:14:00 09:14:06 09:14:10 09:14:15

(Deposition Exhibit 2 was marked for identification.) 09:14:15 09:14:15

Q BY MR. METZGER: Okay. And you have provided me a two-page document entitled, "Testimony Given by 09:14:15 09:14:20

Dr. Pyatt" stating: "The following is a complete list of deposition testimony." 09:14:26 09:14:30

Is that accurate? 09:14:32

A Yes. 09:14:33

Q As of this date? 09:14:33

A Yes. 09:14:34

Q Okay. 09:14:37

A You figure prominently in that list. 09:14:42

Q I do? 09:14:45

A Uh-huh. 09:14:46

MR. CARR: How do you define "prominently"? 09:14:49

THE WITNESS: Well, I think I have 10 or 11 and Mr. Metzger is two of those. 09:14:51 09:14:53

Q BY MR. METZGER: Okay. Well, let's talk about a few of them. 09:14:55 09:14:57

The first one listed is Wilkerson versus Radiator Specialty Company, testimony given on May 17th, 2006. 09:14:59 09:15:03 09:15:07

Was that at a deposition or trial? 09:15:10

A That was a deposition. 09:15:12

Q And who took your deposition in that case? 09:15:14

A Denise Clancy. 09:15:18

Q Did that case go to trial? 09:15:27

A No. 09:15:29

Q Okay. Have you testified at any trials? 09:15:37

A No. 09:15:44

Q Okay. 09:15:45

A One hearing, but I think that might be close, 09:15:50

but it wasn't a trial. 09:15:52

Q What type of hearing was that? 09:15:54

A It is at the very end of that sheet. It was 09:15:56

called the Havner hearing in Texas. 09:15:59

Q That is in the Cantu case? 09:16:01

A Yes. 09:16:03

Q Okay. There is a case listed here Henrickson 09:16:03

versus Conoco-Phillips with testimony given on April 09:16:14

17th, 2008. 09:16:20

Was that a deposition? 09:16:22

A Yes. 09:16:24

Q Who took your deposition in that case? 09:16:24

A I am sorry, I don't remember his name. It was 09:16:27

a French name, started with a "P." Brett Young was my 09:16:28

client so he could probably tell you, but I don't 09:16:37

remember the guy's name. I had never met him before. 09:16:41

Q Brett Young was the defense attorney? 09:16:45

A Yes. He is who I was working with. The guy 09:16:47

that took my deposition, I had never seen him before. 09:16:49

Q And what type of case was that? 09:17:00

A That was a gasoline case and AML. 09:17:03

Q There is a case here Saltonstall versus Dunn 09:17:17

Edwards, testimony given May 1, 2008. 09:17:24

What was that case about? 09:17:27

A I think it was an AML and a painter. 09:17:44

Q Was it a deposition that you gave in that case? 09:17:57

A Yes. 09:18:00

Q Who took your deposition? 09:18:00

A Stamos. 09:18:02

Q Greg Stamos? 09:18:04

A Yes. 09:18:05

Q Next one is Baker versus Chevron, testimony given August 13th, 2008. 09:18:16 09:18:20

What type of case was that? 09:18:23

A Baker is a closed down refinery and there were several plaintiffs kind of lumped into that group that are claiming exposures from this defunct refinery caused their diseases. 09:18:25 09:18:38 09:18:40 09:18:47

Q Are any of those hematologic diseases? 09:18:53

A They all are. 09:19:00

Q They all are? 09:19:00

A Of varying -- varying types. 09:19:00

Q About how many plaintiffs are there in that case? 09:19:00 09:19:00

A I am not sure. There were four that were lumped together in one sort of trial, I guess, or one group. So we -- in that deposition we talked about 09:19:03 09:19:05 09:19:08

four. 09:19:13

Q Okay. When you say "we," who are you referring 09:19:14

to? 09:19:16

A Me and the guy that took my deposition. 09:19:27

Q Who is that? 09:19:29

A It was by phone and I never met him so I can't 09:19:30

tell you. Jason Levin from Steptoe & Johnson, Larry 09:19:33

Riff's group was the defense attorney. And the guy 09:19:39

called in and I remember -- I mean he introduced 09:19:43

himself, but I don't remember his name. 09:19:46

Q Okay. The next one listed is Stromberg versus 09:19:57

Ashland, testimony given September 19th, 2008. 09:20:03

What was that case about? 09:20:10

A Stromberg was an idiopathic myelofibrosis 09:20:13

associated with toluene exposure. 09:20:19

Q Okay. Who took your deposition in that case? 09:20:28

A It was a law firm out of North Carolina that I 09:20:42

had never heard of before. 09:20:47

Q Who was your client in that case? 09:20:51

A Howard Jarvis. I don't know the name of his 09:20:53

firm. I think he works through Wolfe in Knoxville. 09:21:01

Q And most recently Oakley versus U.S. Steel, 09:21:07

testimony given November 7th, 2008. 09:21:15

That is about a month ago? 09:21:19

A Yes. 09:21:20

Q What type of case is that? 09:21:20

A Oakley was an AML case with alleged exposure to 09:21:33

benzene from Liquid Wrench. It was a Liquid Wrench case 09:21:39

and Lance Lubel was the plaintiff -- 09:21:44

Q Attorney? 09:21:49

A -- attorney, yes. 09:21:49

Q Okay. All right. Have you given any 09:21:50

depositions since Oakley? 09:21:56

A No. 09:21:58

Q Well, let's see. So I am counting them, 09:22:03

looking at Exhibit 2, the depositions that you gave this 09:22:05

year were in Behymer, Henrickson, Saltonstall, Baker, 09:22:10

Stromland and Oakley and now this case today; correct? 09:22:19

A Stromland? Stromberg? 09:22:29

Q Stromberg. 09:22:33

A Yes. 09:22:33

Q Oakley, and then today in the Safety-Kleen 09:22:34

cases? 09:22:36

A Correct. 09:22:37

Q And all of these cases involve a hematologic 09:22:44

malignancy; correct? 09:22:50

A Yes. 09:22:51

Q And they all involved claims that benzene 09:22:52

caused that hematologic malignancy. True? 09:22:55

A The Stromberg case -- well, I think, yeah, they 09:23:02

still -- they might be claiming it was a contaminant -- benzene contaminant in the toluene, but it was pretty much a toluene exposure. 09:23:04 09:23:07 09:23:10

Q But toluene doesn't cause AML, does it? 09:23:12

A This was a myelofibrosis case. 09:23:16

Q Let me ask more generally. 09:23:19

Toluene does not cause hematologic malignancies. True? 09:23:22 09:23:26

A Not in my view, no. 09:23:28

Q But benzene has been a contaminant of toluene? 09:23:29

A Yes. 09:23:33

Q All right. So with respect to these -- well, to the six cases in which you have given depositions this year that involved hematologic malignancies, in every one of them, did you testify that benzene did not cause the hematologic malignancy? 09:23:33 09:23:44 09:23:47 09:23:52 09:23:55

A Yes. 09:23:59

Q And is that also your opinion in the cases that you are here to testify about today? 09:23:59 09:24:03

A That the benzene exposures were insufficient to cause these diseases, yes. 09:24:05 09:24:07

Q What do you mean that the benzene exposures were insufficient? 09:24:11 09:24:14

A Well, there is benzene all over. There is benzene in fruit, there is benzene in egg, there is 09:24:15 09:24:17

benzene in background air. So it requires a sufficient dose of a sufficient duration to increase a person's risk of developing AML. 09:24:20 09:24:26 09:24:31

And AML is the only malignancy that I believe has been definitively linked with high dose chronic exposure to benzene. 09:24:33 09:24:40 09:24:44

Some of those cases were other malignancies that I don't believe are linked positively to benzene exposure. 09:24:45 09:24:49 09:24:52

Q Okay. The cases that we are here about today, do you accept that they are all acute myelogenous leukemia cases? 09:24:53 09:24:55 09:24:59

A Yes. 09:25:00

Q All right. Now, when you say that there has to be a sufficient dose of benzene of a sufficient duration, would you tell me precisely what the dose is that it has to be for it to be sufficient in your view? 09:25:00 09:25:07 09:25:09 09:25:17

A Well, I think my view is based on the quantitative scientific literature linking benzene exposure to a meaningfully increased risk of AML. And that literature seems to fall somewhere between 50 and 500 part per million years, the best exposure -- the best estimate being 200. 09:25:20 09:25:23 09:25:27 09:25:32 09:25:35 09:25:40

So in terms of the duration and the dose, they are going to have to collectively get you somewhere 09:25:48 09:25:50

around 550 or higher part per million years. 09:25:54

Q The part per million year is a cumulative dose 09:25:57

metric, is it not? 09:26:00

A Yes. 09:26:03

Q And is there any study that has established 09:26:03

that cumulative dose is the most sensitive metric for 09:26:06

assessing benzene leukemia risk? 09:26:12

A Well, in my view there are. I mean Otto Wong 09:26:16

discussed it in his '87 CMA sponsored study. In the 09:26:20

Glass paper they talk about what happens when you pull 09:26:25

cumulative exposures out of the analysis, then there is 09:26:28

no longer any kind of relationship with intensity. 09:26:31

And there has been discussion and there is 09:26:34

continual discussion about the most appropriate metric 09:26:36

of exposure to correlate with risk. But I do believe 09:26:39

that the cumulative exposure is still valuable and is 09:26:44

still usable in terms of attributing risk for AML. 09:26:48

Q Is there any study that has conducted an 09:26:53

analysis of different dose metrics and specifically 09:26:57

determined that the dose metric that is most sensitive 09:27:02

for predicting leukemia risk is cumulative dose? 09:27:06

A I am not sure that anyone has gone about 09:27:11

addressing the question the way you posed it. 09:27:13

Q Okay. You told me that the benzene exposure 09:27:16

has to be of a sufficient dose and of a sufficient 09:27:31

duration. 09:27:34

Are you able to tell me what the dose component 09:27:36

of that has to be as separate from the duration? 09:27:40

A Well, I am not sure that you could really do 09:27:47

that. 09:27:49

Q Can you tell me what the duration part has to 09:27:49

be separate from the dose component? 09:27:51

A That is really the flip side of the same 09:27:53

question. I mean -- 09:27:55

Q What is your answer though? 09:27:56

A I think that is the debate as to whether or 09:28:00

not -- if you simply look at cumulative exposure, that 09:28:01

does not give you all the information that you probably 09:28:04

need to understand the risk and the biology. 09:28:07

I think that would be generally accepted by 09:28:10

everyone in this field, so both components are 09:28:13

important. 09:28:16

Acute exposures do not increase one's risk of 09:28:17

leukemia, so there has to be some kind of prolonged 09:28:21

exposure in order to start seeing an increased risk. 09:28:24

Really low levels of benzene, no matter how 09:28:28

long you are exposed to them, does not increase one's 09:28:30

risk of developing AML. So there you have to have some 09:28:34

degree of intensity. 09:28:38

When you combine those, you need to get 09:28:39

somewhere around 50 to 500 part per million years before the scientific literature will support that you have an increased risk. 09:28:41 09:28:44 09:28:48

Q You mentioned acute exposures. 09:28:52

What -- could you quantify that for me, what you mean by that? 09:28:55 09:28:59

A Well, toxicologically usually acute exposure is something short of less than 24 hours. 09:29:00 09:29:04

Q But in terms of when you said acute exposures with respect to benzene, what do you mean? What is an acute exposure for benzene? 09:29:07 09:29:10 09:29:16

A Something less than 24 hours. 09:29:18

Q Of any dose? 09:29:19

A Correct. 09:29:21

Q I see. 09:29:21

And how many acute exposures of any dose does it take to cause leukemia -- to cause AML? 09:29:22 09:29:26

A Acute exposures don't cause leukemia. 09:29:30

Q Not at all? 09:29:32

A Not in my view. 09:29:34

Now, if you start doing it more than once, then it's no longer acute. Now you are starting to talk about intermittent peak exposures over some prolonged period of time. 09:29:35 09:29:38 09:29:40 09:29:45

And there you can quantitate what those 09:29:45

exposures would end up being and you are going to be comparing those to the quantitative epi. literature. 09:29:48 09:29:52

If you are just looking at intensity, pure intensity, there are studies that have tried to associate risk of leukemia with intensity of exposure and those are consistent with the cumulative. They have to be pretty high. 09:29:55 09:29:58 09:30:00 09:30:03 09:30:07

Q What are the -- how many peak and intermittent exposures to benzene does it take to cause AML? 09:30:08 09:30:12

A I guess it depends on what you mean by peak and how high they are. The only time that has ever been quantitated in the epidemiologic literature that I'm aware of is the study on the Dow workers published by Ireland where they saw an increased risk, but it was not statistically significant. 09:30:17 09:30:20 09:30:23 09:30:27 09:30:31 09:30:34

But nonetheless they saw an increased risk with peaks over 100 part per million the numbers of days that they had a peak over 100 part per million correlated with AML risk. 09:30:35 09:30:36 09:30:39 09:30:43

Q Now, you mentioned the terms "peak" and "intermittent," so what do you mean by them? 09:30:45 09:30:47

A Well, I think peak is peak. Peak is how high it got to within a certain exposure duration. Intermittent means it happens intermittently. It comes -- it is not a continuous metric. It is not a 09:30:51 09:30:54 09:31:00 09:31:04

continuous exposure. 09:31:08

Q Do you consider peak and intermittent benzene 09:31:10
exposures to be important in assessing leukemia risk? 09:31:12

A Well, as we have been talking about, I feel 09:31:18
like the epidemiological literature is the clearest with 09:31:21
regard to cumulative exposure. And those cumulative 09:31:25
exposures, however they have been calculated, will 09:31:28
contain peaks and intermittents because that is the way 09:31:32
people are exposed in the workplace. 09:31:36

Q I will move to strike the response as 09:31:38
nonresponsive. 09:31:40

Do you consider peak and intermittent benzene 09:31:41
exposures to be important in assessing leukemia risk? 09:31:43

A In terms of the -- how they contribute to the 09:31:47
cumulative exposure, yes. 09:31:52

Q But not otherwise? 09:31:57

A Well, how can they not contribute to the 09:31:59
cumulative exposure? 09:32:01

Q I am just asking, you don't consider peak and 09:32:03
intermittent benzene exposures important in assessing 09:32:05
leukemia risk in and of themselves; is that true? 09:32:09

A I don't know how you could do that. I don't 09:32:13
know how you could assess them in and of themselves 09:32:14
except for how they contribute to the cumulative overall 09:32:17
exposure with the exception of the Ireland paper. 09:32:21

Q Now, does continuous low level benzene exposure or intermittent low level benzene exposure present a greater leukemia risk? 09:32:24 09:32:41 09:32:48

A I'm sorry, could you ask that again. You mean between those two? 09:32:49 09:32:51

Q Between those two, continuous low level or intermittent low level benzene exposure. 09:32:54 09:32:56

A Well, depending on what you mean by low level, I don't think either one of them would contribute to the risk. 09:32:59 09:33:01 09:33:04

So you'd have to quantitate low level and you'd have to quantitate what you are referring to when you -- what do you mean by intermittent and what are the peaks. 09:33:04 09:33:07 09:33:08

Q Well, you have told us what intermittent means to you; correct? 09:33:14 09:33:18

A Yeah, not very articulately, but yes. 09:33:19

Q Okay. Is intermittent or continuous exposure to a particular dose, which of those presents the greater risk of leukemia? 09:33:22 09:33:25 09:33:30

A Oh, I see what you are saying. 09:33:32

Probably the only way that they have been compared in a way to allow an answer to that is in the animal studies. And in the animal studies that literature is pretty conflicted. 09:33:34 09:33:37 09:33:42 09:33:45

There are some studies that indicate that high 09:33:47

dose intermittent exposure damages the marrow more than that same dose over a continual basis. 09:33:49 09:33:54

But then there are other studies by Cronkite that are exactly the opposite, so I don't think that is totally known. 09:33:57 09:34:00 09:34:03

Q What case by Cronkite is totally the opposite? 09:34:04

A I don't remember the date. It was some of his animal work. 09:34:07 09:34:09

Q Specifically for benzene? 09:34:11

A Yes. 09:34:12

Q And what do you recall him finding? 09:34:15

A What do I recall him what? 09:34:18

Q Finding in the study that you can't identify. 09:34:19

A I think he published it in '87, but I am not positive, that continuous exposure was more damaging to the marrow than intermittent exposure on a per gram, per milligram basis. 09:34:24 09:34:28 09:34:33 09:34:39

Q Okay. When you say damaging to the marrow, are you talking about a hematotoxic effect or a carcinogenic effect? 09:34:41 09:34:44 09:34:48

A In that case it was a hematotoxic effect. 09:34:49

Q Are there any studies in animals that you can identify that have shown that continuous exposure is more carcinogenic than intermittent exposures? 09:34:54 09:34:56 09:35:03

A Not sitting here now, but I'm pretty sure that 09:35:13

is true. 09:35:15

Q What is true? 09:35:16

A That continuous exposure, that there is data in animals looking at the carcinogenicity of benzene that would support that same position. The Cronkite one, as I recall, was 300 part per million year and it was a hematotoxic effect. 09:35:17 09:35:21 09:35:27 09:35:29 09:35:33

But there are others where they have looked at zymbal gland formation and find the lymphomas in various rodents and have come to the same conclusion. 09:35:35 09:35:37 09:35:41

Q Can you identify any of the authors? 09:35:44

A I think one was Huff, H-u-f-f. I just didn't -- I didn't prepare to talk about the animal studies today. 09:35:46 09:35:49 09:35:51

Q Okay. Well, let me ask you, have you written out your opinions for this case at all? 09:35:52 09:36:10

A No. 09:36:12

Q How many opinions have you formed for this case? 09:36:13 09:36:15

A Well, kind of thought they would just naturally flow from our discussion, but I have three or four. 09:36:19 09:36:22

Q Okay. Why don't you tell me what they are? 09:36:26

A So I guess the first one would be that this is really a mineral spirits, Stoddard solvent case and that is what Safety-Kleen produced. 09:36:33 09:36:38 09:36:43

Maybe you could extend it to that it is a gasoline case, but it isn't a benzene exposure case, at least not it my view since those things, mineral spirits and Stoddard solvent and gasoline have been looked at. So that would be one. 09:36:48 09:36:52 09:36:57 09:37:01 09:37:07

The second one would be that based on the quantitative exposure assessments that I have seen from both sides, from Dr. Nicas as well as from Dr. Sheehan, none of the three plaintiffs had sufficient exposures to benzene to meaningfully increase their risk of AML. So that would be another one. 09:37:09 09:37:13 09:37:18 09:37:26 09:37:31 09:37:40

The third one, that Mr. Hazlehurst's AML doesn't really fit with what we would think a chemically induced leukemia might look like. The others, to varying degrees, have some characteristics that are consistent. 09:37:48 09:37:55 09:38:01 09:38:06 09:38:11

I disagree with the notion that cytogenetic changes in the absence of sufficient dose or cumulative exposure to benzene is all you need to be able to establish etiology; that the dose has to reach a level that you would associate with an increased risk of AML. 09:38:20 09:38:27 09:38:34 09:38:39 09:38:47

And if that has been achieved, then the cytogenetic changes will give you some insight as to whether or not that disease was caused by those exposures, but not in -- the reverse is not true, in my 09:38:53 09:38:57 09:39:02 09:39:05

opinion. 09:39:09

So I disagree with plaintiff experts that take the position that having a deletion on chromosome 5 is sufficient evidence in and of itself to link it to benzene exposure. 09:39:10 09:39:19 09:39:23 09:39:26

Q Okay. Let's talk about some of these things. 09:39:30

MR. CARR: Raphael, could I put something on the record real quick to hopefully save time and incapsulate. 09:39:38 09:39:40 09:39:43

We are also going to ask him to discuss generally what hematotoxicity is, what genotoxicity is molded into his opinions regarding mineral spirits and gasoline, the inhibitory effects in certain mixtures. 09:39:44 09:39:46 09:39:51 09:39:54

You have already discussed some of the metrics in broad terms that he has looked at, including intermittent and lifetime cumulative dose and we are going to ask him to discuss those. 09:40:02 09:40:05 09:40:08 09:40:13

MR. METZGER: Okay. 09:40:17

MR. CARR: And to the extent that plaintiffs' experts have taken the position that certain of these leukemias are benzene induced or that benzene was a substantial contributing factor, we are going to ask Dr. Pyatt to talk about that as well. 09:40:17 09:40:20 09:40:23 09:40:26 09:40:30

MR. METZGER: What, substantial factor? I am not following you. 09:40:35 09:40:38

MR. CARR: Both plaintiffs' opinions on substantial factor and substantial factor with regard to this chemical, meaning the comparison of the dose of this chemical to the other doses. 09:40:39 09:40:41 09:40:44 09:40:49

MR. METZGER: I don't understand that at all, but I am sure Dr. Pyatt will explain it if he can, if he can understand it. 09:40:54 09:40:54 09:41:01

Okay. Thank you for that framework. 09:41:02

Q So this is a mineral spirits case and not a benzene case? 09:41:11 09:41:13

A That's my view, yes. 09:41:16

Q Okay. Was there benzene in Safety-Kleen 105 solvent? 09:41:18 09:41:26

A Very low levels, yes. 09:41:28

Q What levels were there that you say are very low? 09:41:31 09:41:34

A You'd have to -- I'm probably not the best person to be asked that, but low part per million in the liquid. 09:41:36 09:41:41 09:41:46

Q Do you have an understanding as to what the benzene concentration of Safety-Kleen 105 solvent was in California say, for example, in 1990? 09:41:47 09:41:49 09:41:56

A I can find that information. 09:42:01

Q Let me just ask you, when you say low part per million, what range do you put on that? 09:42:10 09:42:12

A 10, 20 part per million. 09:42:14

Q Okay. Now, why do you call this a mineral 09:42:18

spirits case or a Stoddard solvent case or perhaps a 09:42:29

gasoline case but not a benzene case? 09:42:32

A Well, because Safety-Kleen solvent was mineral 09:42:36

spirits with -- or basically Stoddard solvent with 09:42:43

really low levels of benzene in them. 09:42:47

But the ATSDR, OSHA, there is no regulatory 09:42:49

body out there that considers mineral spirits or 09:42:55

Stoddard solvent that has these levels of benzene in 09:42:58

them to be carcinogenic. 09:43:01

When you look at the animal data, any of the 09:43:04

data, there is no evidence that these materials cause 09:43:06

cancer in people. 09:43:10

And I would argue that is the same with 09:43:12

gasoline where it has 100, perhaps 1,000 times the 09:43:14

concentration of benzene than the mineral spirits do. 09:43:20

Q Well, Safety-Kleen 105 solvent is a product, is 09:43:24

it not? 09:43:28

A I guess, yes. 09:43:30

Q And it's not the same thing as mineral spirits, 09:43:32

is it? 09:43:36

A Well, I think chemically it's very close. 09:43:36

Q It's not the same, is it? 09:43:39

A I would have to compare the actual chemical 09:43:41

compositions, but I think it is very close. 09:43:43

Q Does mineral spirits contain 12 known human 09:43:47
carcinogens in it? 09:43:53

A I don't know the answer to that. My guess is 09:43:58
no. 09:43:59

Q Does mineral spirits contain six known human 09:44:00
carcinogens in it? 09:44:04

A I would have to look at the examples, but no, I 09:44:06
don't think that is true. 09:44:09

Q How many known human carcinogens are you aware 09:44:11
of that are in mineral spirits? 09:44:14

A I am not aware of any with the exception of the 09:44:27
low levels of benzene. 09:44:29

Q Okay. So if this product contained several 09:44:31
known human carcinogens, it would not be properly 09:44:36
characterized as mineral spirits, would it? 09:44:39

A I don't know how to answer that question. I 09:44:50
don't -- 09:44:52

Q If it contained several known human 09:44:52
carcinogens, would it be properly characterized as 09:44:55
Stoddard solvent? 09:44:58

A In my opinion, from what I have seen the 09:44:59
Safety-Kleen solvent is very similar to Stoddard 09:45:02
solvent. 09:45:05

Q Move to strike as non responsive. 09:45:06

If the product contained several carcinogens, would it be properly classified as Stoddard solvent? 09:45:07 09:45:11

MR. FOLEY: Object. It lacks foundation. Calls for speculation. Beyond the scope of what this expert is here for. 09:45:15 09:45:16 09:45:19

You could go ahead and answer though, doctor. 09:45:21

THE WITNESS: I stand by my original answer. As far as I can tell, it is very similar to Stoddard solvent. 09:45:24 09:45:25 09:45:29

Q BY MR. METZGER: You have told me that twice now, but I am asking you a different question, Dr. Pyatt. I am asking you a hypothetical question. 09:45:29 09:45:32 09:45:33

I'd like you to assume that the Safety-Kleen 105 solvent contains several carcinogens. If that is true, would that product be properly classified as Stoddard solvent? 09:45:36 09:45:38 09:45:42 09:45:46

MR. FOLEY: Object that it's an incomplete hypothetical. Lacks foundation. Calls for speculation. 09:45:48 09:45:49

THE WITNESS: I haven't seen any evidence that that is true. 09:45:52 09:45:54

Q BY MR. METZGER: I am not asking you that. 09:45:54

Do you understand what a hypothetical question is? 09:45:56 09:45:58

MR. FOLEY: Objection. Argumentative. 09:45:59

THE WITNESS: That we are just making it up. 09:46:01

Q BY MR. METZGER: No. A hypothetical question is not made up. I am asking you to make an assumption that I believe I can prove at trial. I wouldn't just make up something for a hypothetical -- 09:46:02 09:46:05 09:46:10 09:46:16

A Okay. 09:46:19

Q -- unlike some people. 09:46:19

So I am asking you to assume that I have evidence that there are several carcinogens in Safety-Kleen 105 solvent. 09:46:23 09:46:26 09:46:29

Assuming that to be true, would you classify that product as Stoddard solvent? 09:46:33 09:46:36

A I don't think it is Stoddard solvent, but I think it is chemically very similar to Stoddard solvent. 09:46:42 09:46:44

Q Okay. Now, let me ask you why would you classify the product as Stoddard solvent at all when Stoddard solvent isn't even used to make it? 09:46:48 09:46:50 09:46:54

A Well, just because of the chemical composition of these materials, they seem to me to be pretty similar. 09:46:57 09:47:00 09:47:02

Q What seems to be pretty similar to Stoddard solvent? 09:47:03 09:47:06

A Mineral spirits and the Safety-Kleen products. 09:47:07

Q You understand that the starting material so to speak for Safety-Kleen 105 is mineral spirits; correct? 09:47:14 09:47:18

A That wouldn't surprise me, but no, I didn't 09:47:23

know that. 09:47:25

Q Okay. There is a difference between mineral 09:47:26

spirits and Stoddard solvent; isn't there? 09:47:28

A They are relatively subtle from a toxicological 09:47:31

point of view. 09:47:35

Q There is a difference; isn't there? 09:47:36

A There might be a small difference chemically 09:47:37

between percentages of this and that, but from a 09:47:40

toxicological point of view, I don't think they matter. 09:47:42

Q Stoddard solvent has a higher initial boiling 09:47:45

point than mineral spirits, does it not? 09:47:48

A I didn't know that. 09:47:50

Q And because Stoddard solvent has a higher 09:47:52

initial boiling point than mineral spirits, it would 09:47:56

have a lesser benzene concentration, would it not? 09:47:59

A That seems true. 09:48:03

Q Okay. So why do you say that this is a -- 09:48:05

well, let me ask you, are you aware that Safety-Kleen 09:48:11

105 solvent is hazardous waste? 09:48:14

MR. FOLEY: Objection. Argumentative. Lacks 09:48:18

foundation. 09:48:19

THE WITNESS: No. 09:48:21

Q BY MR. METZGER: Okay. If Safety-Kleen 105 09:48:22

solvent were hazardous waste, would you say that this is 09:48:26

a hazardous waste case rather than a mineral spirits 09:48:30

case? 09:48:33

MR. FOLEY: Same objection. And incomplete 09:48:34
hypothetical. 09:48:35

THE WITNESS: I don't even understand what you 09:48:37
mean. 09:48:38

Q BY MR. METZGER: Well, you have told me that 09:48:39
this is not a benzene case; it's a mineral spirits case. 09:48:40

MR. FOLEY: Objection. Argumentative. 09:48:44

Q BY MR. METZGER: But if in fact Safety-Kleen 09:48:45
105 solvent wasn't mineral spirits but it's hazardous 09:48:47
waste, would you then say that this is actually a 09:48:50
hazardous waste case? 09:48:54

A Well, you can classify lots of things as 09:48:56
hazardous waste. I mean there are all kinds of things 09:48:59
that would be considered hazardous waste, heavy metal 09:49:02
contamination, all kinds of things. It would have no 09:49:07
relationship whatsoever to this product. 09:49:09

Whereas I think the relationship with mineral 09:49:12
spirits and Stoddard solvent is pretty clear. They are 09:49:14
very closely related. 09:49:17

Q Okay. All right. 09:49:19

Now, your second opinion you told me was that 09:49:22
none of the three plaintiffs had sufficient exposure to 09:49:25
benzene to meaningfully increase their risk of AML. 09:49:29

What do you mean by the word "meaningfully"? 09:49:34

A Well, I mean from an epidemiologic point of view, I mean that it is a statistically significant finding and that it is likely reproducible with other studies. 09:49:43 09:49:47 09:49:50 09:49:53

If you look at how EPA regulates benzene and it's a linear extrapolation with their modeling, then any dose has a quantifiable risk, but that is a public policy type decision that I don't think matches with the biology. 09:49:54 09:50:01 09:50:05 09:50:14 09:50:17

So a meaningful increased risk is going to be something where you see statistically significant elevations in more than a single study. 09:50:18 09:50:22 09:50:26

Q In more than a single epidemiologic study? 09:50:30

A Correct. 09:50:33

Q Okay. Well, isn't epidemiology too insensitive an analytical technique to determine increased risks for low level exposures? 09:50:34 09:50:40 09:50:49

A Well, I have heard that argument. 09:50:52

Q Isn't it true? 09:50:54

A I'm not sure whether it's true or not. 09:50:55

Q Okay. Now, your third opinion, you told me that Mr. Hazlehurst's AML doesn't fit with what a chemically induced leukemia -- I actually didn't get it all down here. 09:50:57 09:51:13 09:51:18 09:51:23

A I was probably rambling. 09:51:25

Q But could you restate that since I didn't write it all quickly enough. 09:51:28 09:51:31

A Well, Mr. Hazlehurst's AML was cytogenetically normal which, in my view, is not consistent with what we would think a chemically induced leukemia would look like. 09:51:32 09:51:37 09:51:41 09:51:45

Q Okay. What does a chemically induced leukemia look like? 09:51:45 09:51:49

A Well, that depends really on the chemical. 09:51:51

Q Well, what does a benzene induced leukemia look like? 09:51:53 09:51:56

A Well, we've learned the most, in my view, about benzene from looking at the chemotherapy and I think it seems to match with the alkylating chemotherapy the best. 09:51:57 09:52:01 09:52:04 09:52:08

And in those cases you would have potentially a hypocellular marrow. You would have a preceding pancytopenia, perhaps a preceding diagnosis of myelodysplasia and there would be evidence of cytogenetic changes. 09:52:08 09:52:11 09:52:18 09:52:23 09:52:26

Q What cytogenetic changes? 09:52:28

A The alkylating chemotherapy, you would see potentially monosome 7 or deletions in chromosome 5 as well as others, but those would be -- seem to correlate the best. 09:52:30 09:52:33 09:52:40 09:52:42

Q And those are exactly the chromosomal abnormalities that Mr. Pearl and Mr. Machado had. True? 09:52:45 09:52:48

A I have not seen any evidence that Mr. Pearl had monosome 7, but he did have a deletion in chromosome 5. There are deletions in chromosome 5 in lots of AML cases that have no evidence of exposure. 09:52:52 09:52:54 09:52:59 09:53:04

So I don't believe that in and of itself will allow someone to say this was definitively a benzene case or a benzene case of AML. 09:53:07 09:53:10 09:53:14

Mr. Machado did have those cytogenetic changes but was also positive for the Philadelphia chromosome, which I have never seen anyone associate that with benzene exposure or show that the 922 translocation can be caused by benzene or its metabolites. 09:53:17 09:53:22 09:53:26 09:53:30 09:53:34

Q All right. Let's talk about each of these. 09:53:42

Is it true that Mr. -- both Mr. Pearl and Mr. Machado had deletions of chromosome 5? 09:53:50 09:53:54

A That is my understanding, yes. 09:54:00

Q Is it true that benzene causes deletions of chromosome 5? 09:54:02 09:54:07

A There is evidence that it can do that. It generally is thought to require very high doses. 09:54:10 09:54:13

Q Is it true that Mr. Pearl and Mr. Machado both had abnormalities of chromosome 7? 09:54:21 09:54:26

A That I don't have a record of. I did not see 09:54:30

chromosome 7 in Mr. Pearl, but I might have missed it. 09:54:34
I did see that in Mr. Machado. 09:54:38
Q Would it be -- okay. 09:54:41
Is it true that benzene causes abnormalities of 09:54:45
chromosome 7? 09:54:50
A There is evidence that it can do that. 09:54:51
Q And is there evidence that benzene causes the 09:54:55
type of abnormality of chromosome 7 that Mr. Machado 09:54:58
had? 09:55:02
A The monosome 7, that seems to be the case under 09:55:03
the appropriate exposure conditions. Lots of people 09:55:08
have monosome 7 with no benzene exposure. 09:55:11
Q And you have reviewed the cytogenetic records 09:55:20
for Mr. Pearl and you have found no evidence of any 09:55:24
abnormality of chromosome 7? 09:55:27
A I didn't -- I didn't see it. 09:55:29
And yes, I did look at all of the biopsy 09:55:31
results and the cytogenetic changes. It is possible I 09:55:33
missed it. 09:55:37
Q Okay. That would be an important thing to 09:55:38
miss, would it not? 09:55:40
A Well, it's an important finding, but it 09:55:42
wouldn't change my overall opinion because he had 09:55:45
deletion in chromosome 5. 09:55:49
Q Why would that be a -- why is that an important 09:55:51

finding? 09:55:55

A Well, because of the subject that we are now 09:55:56
discussing, is that high dose exposure to benzene has 09:55:59
been linked to changes in those chromosomes. 09:56:03

Q Specifically chromosomes 5 and 7? 09:56:08

A Correct. 09:56:10

Q In AML? 09:56:11

A That is a harder question to answer. They 09:56:14
have -- they have seen those chromosome changes 09:56:17
associated with chemotherapeutic agents that act as 09:56:21
alkylating agents and that seems to follow the same 09:56:27
pattern as benzene. 09:56:29

They have shown that benzene can cause these 09:56:31
cytogenetic changes in a petri dish in vitro. It is 09:56:33
very difficult to relate those exposures to what an 09:56:38
occupational exposure might be. 09:56:41

And there have been some studies where they 09:56:43
have shown that under high dose exposure to benzene, 09:56:45
they do see increases in those findings in peripheral 09:56:48
lymphocytes. 09:56:53

Q But the abnormalities of chromosome 5 and 7 are 09:56:55
typically abnormalities that are seen in AML, are they 09:56:59
not? They are not seen in other diseases? 09:57:04

A There is a thing called 5Q syndrome, which is 09:57:08
not per say AML. It is more of a myelodysplastic type 09:57:12

process that doesn't always go to AML. And you can see monosome 7 in other diseases, but by and large I think what you are saying is correct. 09:57:16 09:57:20 09:57:25

Q Okay. Now, Mr. Pearl and Mr. Machado both had complex karyotypes, did they not? 09:57:26 09:57:30

A Mr. Pearl and Mr. Machado, yes. Machado, for sure. And Mr. -- yes. 09:57:33 09:57:41

Q And that is entirely consistent with a benzene induced leukemia, is it not? 09:57:43 09:57:48

A As far as that goes, that is consistent with most cases of a chemically induced leukemia. 09:57:55 09:57:58

Q Okay. 09:58:01

A But there are also many examples of complex cytogenetic changes where there was no exposure history at all. So you can't separate out the quantitative exposure from that single finding. 09:58:01 09:58:05 09:58:12 09:58:13

Q Can you tell me what chemicals are known to be capable of causing the abnormalities of chromosome 5 and 7 that Mr. Pearl and Mr. Machado have? 09:58:17 09:58:29 09:58:35

A I haven't seen any evidence of any. 09:58:57

Q Other than benzene, you mean? 09:58:59

A Well, I don't believe their exposures to benzene were sufficient to do it either. 09:59:01 09:59:03

Q I am not asking -- we are not tracking, doctor. I am not asking you particularly about what caused Mr. 09:59:05 09:59:08

Machado's or Mr. Pearl's AML. What I am asking you is to focus on the types of chromosome abnormalities that they had in chromosomes 5 and 7, those abnormalities. 09:59:11 09:59:17 09:59:23

What I am asking you is what chemicals are you aware of that are capable of producing those abnormalities? 09:59:31 09:59:34 09:59:38

A That they would have been exposed to? 09:59:42

Q No. 09:59:45

A In general. 09:59:46

Q In general. 09:59:46

A Cyclophosphamide, Melphlan. There is a variety of other -- the nitrogen ureas, the nitrogen mustards. Melphlan has several cousins that are all alkylating agents. Those are all leukemogenic chemotherapeutic agents that potentially could cause these changes. 09:59:47 09:59:52 10:00:00 10:00:06 10:00:12

Q What else? 10:00:15

A Radiation, ionizing radiation potentially. 10:00:18

Q What else? 10:00:24

A Some squirrely evidence, but there is probably a little bit of evidence about cigarette smoking, although it doesn't tend to track with those changes too often. And then high dose benzene exposure. 10:00:26 10:00:31 10:00:34 10:00:38

Q Okay. So let's consider these. There is a whole bunch of drugs that you mentioned which are alkylating agents; correct? 10:00:43 10:00:52 10:00:54

A Yes. 10:00:56

Q Did Mr. Pearl have any exposure to those drugs before he developed AML? 10:00:58 10:01:02

A Not to my knowledge, no. 10:01:05

Q Did Mr. Machado have any exposure to those drugs before he developed AML? 10:01:07 10:01:12

A Not that I am aware of, no. 10:01:13

Q Did Mr. Pearl have any exposure to those drugs before he developed the abnormalities of his chromosome 5 and chromosome 7? 10:01:17 10:01:23 10:01:26

A Not that I am aware of. 10:01:29

Q Did Mr. Machado have any exposure to alkylating agents before he developed the abnormalities to chromosomes 5 and 7? 10:01:31 10:01:35 10:01:42

A Machado, no, not that I know of. 10:01:44

Q Did Mr. Pearl have any exposure to ionizing radiation before he developed AML or his abnormalities of chromosome 5 or 7? 10:01:50 10:01:53 10:01:58

A Not that I am aware of. 10:02:00

Q Did Mr. Machado have any exposure to ionizing radiation before he developed AML or the abnormalities to chromosome 5 and 7? 10:02:01 10:02:04 10:02:09

A Not that I am aware of. 10:02:12

Q You mentioned some squirrely evidence of cigarette smoking; correct? 10:02:16 10:02:19

A Yes. 10:02:20

Q Do I take it then that you do not consider cigarette smoking to be an established cause of chromosome 5 or 7 abnormalities? 10:02:20 10:02:23 10:02:26

A I think the literature supports that cigarette smoking is an established cause of AML, but the cytogenetic changes associated with that have not been very well characterized in my view. 10:02:30 10:02:32 10:02:37 10:02:41

Q So can you answer my question? 10:02:44

A So I don't think 5 or 7 have very good support for being associated with a cigarette smoking induced AML, but there might be a single study that has said maybe these are the ones. 10:02:46 10:02:49 10:02:52 10:02:56

Q I am trying to ask a simple question, and that is do you consider cigarette smoking to be an established cause of abnormalities of chromosome 5 and 7? 10:03:00 10:03:03 10:03:08 10:03:13

A I don't have a definitive answer for that. I don't think the evidence is very strong. 10:03:17 10:03:19

Q Do you think that the evidence is less than probable at this point? At this point it is a possibility, but it has not been established to be a probability? 10:03:25 10:03:27 10:03:32 10:03:34

A I guess that would depend on your definition of probability, but I think that is fine. It is a 10:03:35 10:03:38

possibility. And I think there are some studies that suggest perhaps, but then there are others that say no. And then there is a lot of evidence that they basically don't know. 10:03:40 10:03:43 10:03:46 10:03:49

So that would be my position, that we just do not know the cytogenetic changes that are likely associated with cigarette smoking induced AML. 10:03:49 10:03:52 10:03:55

Q And when you say, "we don't know," you mean that you don't know; correct? 10:03:58 10:04:00

A I mean the scientific and medical community does not know. 10:04:02 10:04:05

Q Well, with due respect, doctor, you haven't been appointed by any medical or scientific body to testify as an expert in this case; correct? 10:04:06 10:04:08 10:04:11

A No one has appointed me -- I think I am following your question. 10:04:15 10:04:18

Q Right. 10:04:20

You haven't been appointed by any medical body or scientific body to be an expert in this case; correct? 10:04:20 10:04:23 10:04:26

A Oh, I think I see where you are going. 10:04:27

Well, no, but I have read all of the studies that have linked cigarette smoking to AML and those that have discussed the cytogenetic changes. 10:04:30 10:04:32 10:04:35

And I feel like I am qualified to tell you that 10:04:37

they do not show a consistent pattern of cytogenetic changes. 10:04:43 10:04:44

Q And that is your opinion? 10:04:44

A That is what the scientific literature supports. 10:04:45 10:04:48

Q In your opinion? 10:04:48

A Yes. 10:04:49

Q Okay. I would ask you if you would be kind enough to confine your testimony to your opinions as opposed to those of the scientific and medical communities because you haven't been appointed by them to testify as an expert in this case. 10:04:49 10:04:52 10:04:54 10:04:57 10:04:59

Okay? 10:05:01

A Well, that is a semantical argument and I will do what I can. I feel you can point it out if you are not happy with my answer. 10:05:03 10:05:07 10:05:10

THE VIDEOGRAPHER: Counsel, there is two minutes left on the tape. 10:05:12 10:05:13

MR. METZGER: All right. 10:05:15

Q In any event, you would agree, would you not, that cigarette smoking did not cause the chromosome 5 or 7 abnormalities of either Mr. Machado or Mr. Pearl; correct? 10:05:17 10:05:21 10:05:29 10:05:32

A I would agree with that, yes. 10:05:33

Q Okay. And so it is true, is it not, that the 10:05:35

only chemical to which they were exposed which are known to cause the chromosome 5 and chromosome 7 abnormalities in AML is benzene? 10:05:40 10:05:44 10:05:48

MR. FOLEY: Objection. Lacks foundation. Calls for speculation. 10:05:51 10:05:52

THE WITNESS: It -- the basis of that question ignores the reality that these cytogenetic changes happen all the time with no chemical etiology. 10:05:55 10:05:58 10:06:02

Q BY MR. METZGER: What is the answer to my question? 10:06:07 10:06:08

Is it true that the only chemical to which they were exposed which is known to cause chromosome 5 and 7 abnormalities in AML is benzene? 10:06:09 10:06:10 10:06:13

MR. FOLEY: Same objections. 10:06:18

THE WITNESS: The evidence in terms of a quantitation, you know -- one molecule of Melphlan isn't going to do this. 10:06:20 10:06:22 10:06:28

So while the answer to your question -- the simple answer is yes, you also have to include what their exposures were. 10:06:29 10:06:31 10:06:35

And there is no evidence in any literature that these individuals' exposures can cause those cytogenetic changes to benzene. Their exposures to benzene, in my view, are not sufficient to cause these changes. 10:06:36 10:06:39 10:06:44 10:06:48

MR. METZGER: Okay. We need to take a tape 10:06:52

change so why don't we take a break. 10:06:53

THE VIDEOGRAPHER: Now going off the record. 10:06:55

Time is 10:06 a.m. This is the end of Volume 1, tape 10:06:56

No. 1. 10:07:04

(Brief recess.) 10:07:05

THE VIDEOGRAPHER: Now back on the record. 10:18:08

Time is 10:58 a.m. This is the beginning of Volume 1, 10:18:18

tape No. 2 in the deposition of Dr. David Pyatt. 10:18:21

Counsel? 10:18:26

Q BY MR. METZGER: Dr. Pyatt, before we took a 10:18:27

tape break and a break, we were talking about the 10:18:30

various things that are known to cause abnormalities of 10:18:36

chromosome 5 and 7 and their relevance to Mr. Pearl and 10:18:42

Mr. Machado. 10:18:47

I want to ask you a broader question now, and 10:18:48

that is, what are the chemicals or other things that in 10:18:52

your opinion are known to cause AML? 10:18:59

A Well, the majority of AML cases have no known 10:19:08

etiology. So 80, 85 percent of them we don't know what 10:19:11

caused them. 10:19:17

Q You don't know? 10:19:18

A They are unknown. 10:19:20

Q To you? 10:19:21

A To the scientific community. 10:19:21

Q Doctor, please don't speak on behalf of the 10:19:24

scientific community. You can speak -- you can tell us your own opinions. 10:19:26 10:19:30

Let me try this: You understand that you are testifying as an expert today; correct? 10:19:31 10:19:34

A Yes. 10:19:36

Q You understand that different experts can have different opinions; correct? 10:19:36 10:19:41

A Yes. 10:19:42

Q And when you say, "the scientific community," I assume that you have not actually done a scientific survey of the scientific community to assess the opinions of all of the members of the scientific community on particular issues; is that correct? 10:19:43 10:19:46 10:19:49 10:19:53 10:19:56

A Well, on particular issues, that is true. But with regard to what we are discussing right now, there are a limited number of publications where people have attributed a percentage of AML cases to known factors and to unknown factors. 10:20:00 10:20:03 10:20:05 10:20:09 10:20:14

And I have read all of those papers and they are all very consistent that say about 80 or 85 percent of them. 10:20:16 10:20:18 10:20:22

Q Okay. And based on that, you can express your opinion? 10:20:23 10:20:25

A That is what I did. 10:20:26

Q Right. 10:20:27

And I would ask you please not to purport to express opinions on behalf of the scientific community because they have not been designated as experts in this case and you have not done a statistical survey or published any article regarding their opinions. 10:20:27 10:20:30 10:20:33 10:20:38 10:20:42

All right? 10:20:46

A All right. 10:20:48

How about this: It is my opinion that 80 or 85 percent of them are unknown, which is consistent with all of the publications that exist on this issue. 10:20:48 10:20:51 10:20:54

Q That's fine. 10:20:56

Now, I am glad that we have that squared away, but that wasn't what I asked you -- 10:20:58 10:21:05

A Right. 10:21:06

Q -- at all. 10:21:07

A Right. 10:21:07

Q What I asked you was, please tell me what are those chemicals or things that, in your opinion, are known to cause AML? 10:21:08 10:21:10 10:21:13

A So then when you get past the 80 or 85 percent that we don't know what causes them, then you have got the majority of the remainder are chemotherapeutic agents, either alkylating agents or topoisomerase inhibitors, two classes of chemotherapeutic agents that are established leukemogenic chemicals. 10:21:15 10:21:20 10:21:23 10:21:29 10:21:31 10:21:35

Then there is ionizing radiation. There is high dose chronic exposure to benzene. There is cigarette smoking. There are some viruses associated with some types of leukemia but not AML. 10:21:41 10:21:46 10:21:49 10:21:52

So those -- that would be the list. 10:21:56

Q Okay. So is it true that Mr. -- neither Mr. Pearl nor Mr. Machado nor Mr. Hazlehurst had exposure to any chemotherapeutic agents prior to their AML diagnosis? 10:21:59 10:22:04 10:22:14 10:22:20

A Not that I have seen, no. That is true. 10:22:22

Q Okay. And that includes both alkylating agents and topoisomerase inhibitors, they had no exposure to those classes of drugs before they were diagnosed with AML; correct? 10:22:25 10:22:28 10:22:33 10:22:36

A They have had no exposure to the chemotherapeutic agents that act like topoisomerase inhibitors. There is literature that we are all exposed to drugs or chemicals in our diet that may be topoisomerase inhibitors, but I don't believe that is what caused their leukemia either. 10:22:38 10:22:40 10:22:45 10:22:48 10:22:53 10:22:57

Q Okay. And is it true that Mr. -- none of the plaintiffs in these cases had exposure to ionizing radiation before they developed their AML's? 10:22:59 10:23:02 10:23:05

A Not to my knowledge, no. 10:23:08

Q That's true, what I said? 10:23:10

A I'm sorry. Yes, that is true. 10:23:11

Q Okay. And is it true that cigarette smoking, 10:23:13

in your opinion, did not cause Mr. Pearl's and Mr. 10:23:17

Machado's leukemias? 10:23:21

A That is true. 10:23:24

Q Okay. Do you have an opinion as to whether 10:23:25

cigarette smoking caused the AML of Mr. Hazlehurst? 10:23:27

A I don't know whether it caused Mr. Hazlehurst's 10:23:33

AML or not, but I can say that cigarette smoking is an 10:23:36

established etiological risk factor for AML and I don't 10:23:41

think you can completely rule it out. 10:23:45

Q All right. And why can't you completely rule 10:23:53

it out? 10:24:00

A Because it's an established risk factor for AML 10:24:03

and he had a sufficient exposure history of smoking that 10:24:08

would put him at an elevated risk. 10:24:15

Q Benzene is an established risk factor for AML, 10:24:18

is it not? 10:24:22

A Certain exposure scenarios, yes. 10:24:23

Q But you are able to rule out benzene but not 10:24:30

cigarette smoking as the cause of Mr. Hazelhurst's AML? 10:24:32

A I am. 10:24:38

The benzene has been quantitated so his benzene 10:24:41

exposure has numbers associated with it. The cigarette 10:24:44

smoking, less so. 10:24:48

And the epidemiologic literature, in my view, is better in terms of the quantitation of benzene required to give someone AML. 10:24:48 10:24:51 10:24:56

Cytogenetics for Mr. Hazlehurst are not consistent with a chemical etiology, which would also, in my view, limit the probability that cigarette smoking caused his AML. I would think that those are probably going to be cytogenetically abnormal as well. 10:24:59 10:25:02 10:25:06 10:25:11 10:25:16

But I don't think you can completely rule it out because there is quite a bit of discussion, argument, variability, uncertainty around the cytogenetic changes associated with smoking. 10:25:19 10:25:21 10:25:25 10:25:28

Q Okay. Now, you mentioned earlier something about a hypocellular marrow. 10:25:35 10:25:41

First of all, what is that? 10:25:47

A That when you go in and look in a person's bone marrow with biopsies, there is a cellularity component, how many cells are in there and the rest of the space is filled up with fat. 10:25:52 10:25:55 10:26:02 10:26:05

And you can have a hypercellular, too many cells in the marrow where you go in and look and there is not as much fat as you would expect and there is more cells. 10:26:07 10:26:10 10:26:14 10:26:19

Or you could have a hypocellular, which is too few cells in the marrow that you would expect based on 10:26:19 10:26:22

1	the age and the person.	10:26:26
2	Q And what -- when you mentioned a hypocellular	10:26:29
3	marrow, what was the significance of that to you?	10:26:35
4	A That in alkylating chemotherapy and some	10:26:38
5	literature on benzene, high exposure to benzene results	10:26:44
6	in a hypocellular marrow.	10:26:48
7	And that is frequently seen in myelodysplasias	10:26:51
8	that precede the development of AML following exposure	10:26:55
9	to alkylating chemotherapy.	10:26:59
10	So this bone marrow damage that is manifest as	10:27:02
11	a peripheral pancytopenia and seen in the marrow as a	10:27:05
12	hypocellular marrow is consistent with severe chemical	10:27:10
13	insult.	10:27:14
14	Q And what is the relevance of that to these	10:27:17
15	cases that we have here?	10:27:19
16	A Well, I don't think it has much relevance to	10:27:25
17	these cases because none of these patients or	10:27:28
18	individuals had the levels of benzene that would cause	10:27:32
19	any kind of bone marrow damage.	10:27:32
20	Q Well, I am not quite understanding.	10:27:35
21	Are you suggesting that Mr. Pearl, Mr.	10:27:37
22	Hazlehurst and Mr. Machado did not have a hypocellular	10:27:41
23	bone marrow?	10:27:47
24	A You asked me about what I thought an alkylating	10:27:50
25	agent AML would look like, and that is where I listed	10:27:55

the hypocellular marrow as a characteristic associated with that type of chemical exposure. 10:28:00 10:28:04

Q I see. 10:28:07

So I think you are absolutely right. I couldn't remember the context. 10:28:07 10:28:11

So you mentioned that a hypocellular marrow, a preceding pancytopenia and myelodysplastic syndrome were all characteristic of an alkylating induced AML? 10:28:13 10:28:17 10:28:24

A That is correct. And to the extent that it has been evaluated, it is also somewhat consistent with a benzene induced process. 10:28:29 10:28:31 10:28:36

Q What do you mean it is somewhat consistent with a benzene induced process? 10:28:37 10:28:39

A Well, we just don't know as much. We just don't know as much about and benzene its ability to cause AML and what those diseases look like. 10:28:41 10:28:44 10:28:48

There are studies going on now that potentially will answer some of those questions, but we know a whole lot more about people that get AML in the clinic and we can follow them and look at their cytogenetics and all these other aspects that we are not really able to do with benzene. 10:28:51 10:28:53 10:28:57 10:29:00 10:29:05 10:29:07

Q There have been enumerable case reports over the years, over nearly a century of benzene induced hematologic disease. True? 10:29:09 10:29:14 10:29:18

A Well, I think it was just a figure of speech. 10:29:23
I don't know what "enumerable" means, but there have 10:29:25
been many. 10:29:28
Q There has been a whole lot? 10:29:29
A Yes, there has been a whole lot. 10:29:31
Q And you have reviewed those, have you not? 10:29:32
A I have reviewed them, probably not all of them. 10:29:34
I mean some were published in the teens, the twenties, 10:29:36
the thirties. And I couldn't get all of them, but I 10:29:38
have reviewed many of them. 10:29:42
Q You made an effort to do that as part of your 10:29:43
research; correct? 10:29:47
A Yes, yes. 10:29:48
Q And the marrows in those -- they are typically 10:29:52
workers, are they not? 10:29:55
A Yes. 10:29:56
Q The marrows in those workers who are exposed to 10:29:56
benzene are sometimes hypocellular; correct? 10:30:02
A Sometimes hypocellular? 10:30:06
Q Yes. 10:30:08
A Yes. 10:30:09
Q And they are sometimes hypercellular; correct? 10:30:09
A There are definitely studies that would suggest 10:30:12
that, yes. 10:30:14
Q Benzene can cause both types of damage, 10:30:15

hypocellularity and hypercellularity. True? 10:30:19

A Well, that is not totally known. I mean just 10:30:25
because there is a case report where they report a 10:30:28
hypercellular marrow, well, maybe that had something to 10:30:31
do with their benzene and maybe it did not. And you 10:30:34
could make the same argument with the hypocellular. 10:30:37

In my view, a hypocellular marrow is a more 10:30:40
consistent pathological picture for a benzene or an 10:30:44
alkylating agent induced AML. 10:30:47

Q Well, have you actually looked at those case 10:30:50
reports where workers heavily exposed to benzene, pure 10:30:53
benzene, had developed hematologic malignancy and their 10:31:00
marrows were evaluated? 10:31:07

Have you looked at those to determine what 10:31:08
percentage of them had hypercellular marrows and what 10:31:10
percentage of them had hypocellular marrows? 10:31:15

A I haven't ever done, you know, a five to three 10:31:20
comparison. Aksoy has published several review papers 10:31:23
where that is exactly what he did. 10:31:28

And you are correct, there are examples of 10:31:30
where, in his view, they were attributable to benzene. 10:31:33
And there is some that are hyper and there is some that 10:31:36
are hypo. I think the hypo are in a greater quantity, 10:31:39
but that doesn't mean that the hyper cannot happen. 10:31:45

Rich Irons published a paper in 2005 out of 10:31:48

China and that is probably the most recent analysis of what the bone marrow looks like in highly exposed workers and in that case the majority were hypocellular. 10:31:52 10:31:57 10:32:01

Q And none of those, the subjects of Dr. Irons' study, actually had AML. True? 10:32:07 10:32:12

A They did not have AML. 10:32:15

Q Okay. You also mentioned a preceding pancytopenia. 10:32:17 10:32:23

Did either Mr. Pearl or Mr. Hazlehurst or Mr. Machado have that? 10:32:26 10:32:29

A Not that I recall. There was one medical report of Mr. Machado -- I think it is Mr. Machado -- where in '89 he had surgery and they saw some blood abnormalities that, if it were persistent, they might classify it as pancytopenia, but all that I saw record of was lymphocytopenia at that one snapshot. 10:32:37 10:32:44 10:32:50 10:32:53 10:32:58 10:33:05

When a person develops AML, there is frequently going to be pancytopenia at the time of diagnosis. I am not sure that that really tells you anything about the etiology. 10:33:07 10:33:10 10:33:14 10:33:16

So this would be six months, a year previous to the diagnosis where they were seeing persistent refractory type changes in the blood. And I did not see evidence of that in any of these three people. 10:33:17 10:33:18 10:33:23 10:33:28

Q Incidentally, regarding that cytopenia that you 10:33:30

observed in the medical records regarding Mr. Machado when he was -- that was in connection with a surgery that he had, was it not? 10:33:34 10:33:37 10:33:40

A It was prior to the surgery, getting ready for it, that is my understanding, yes. 10:33:41 10:33:44

Q And you don't -- do you attribute any significance to that finding to the etiology, to the cause or progression of his AML? 10:33:45 10:33:48 10:33:50

A I think that it would be really hard to say. I mean it was a single snapshot in time. It did indicate at least at that one point there was something going on with his blood. 10:33:55 10:33:57 10:34:00 10:34:03

Q Are you a hematologist, by the way? 10:34:05

A No. 10:34:07

Q Okay. Are you prepared to tell the jury in this case that he had his disease back at the time he had that surgery? 10:34:07 10:34:10 10:34:13

A "His disease," being his AML? 10:34:16

Q Yes. 10:34:18

A No, no. 10:34:19

Q Okay. Now, in your opinion is a preceding pancytopenia consistent with a benzene induced AML? 10:34:19 10:34:31

A I think there is evidence to support that, yes. 10:34:45

Q In your opinion, is the absence of a preceding pancytopenia consistent with a benzene induced AML? 10:34:48 10:34:55

A It is less so. I don't think -- it's not a black or white issue. You can't say definitively that if you have it or you don't have it that that is the be all and end all, and therefore you can totally rule out etiology. 10:35:01 10:35:04 10:35:08 10:35:11 10:35:15

But I do think that pancytopenia or evidence of blood changes associated with marrow damage is consistent with the pathology of benzene induced AML. 10:35:15 10:35:20 10:35:22

Q When you say that a -- that the absence of preceding pancytopenia is consistent with a benzene induced leukemia but less so, are you able to quantify the less so for me? 10:35:28 10:35:31 10:35:39 10:35:43

A You mean how much less consistent it is? No, I couldn't put a number on it. 10:35:47 10:35:50

Q Okay. 10:35:52

A But I do believe that it is inconsistent with our understanding. 10:35:53 10:35:56

Q With your understanding? 10:35:57

A With -- right, my understanding and the published literature on this topic. 10:35:58 10:36:02

Q Okay. Now, you also mentioned myelodysplastic syndrome in connection with -- that a preceding myelodysplastic syndrome is consistent with a chemotherapy induced AML. 10:36:05 10:36:12 10:36:17 10:36:21

Did I get that right? 10:36:24

A You did. 10:36:26

I mean I don't want to quibble, but there are 10:36:26

two big classes of chemotherapeutic agents and the 10:36:30

topoisomerase drugs will rarely have a preceding 10:36:33

myelodysplasia. 10:36:39

Q Okay. With a preceding alkylating induced AML? 10:36:42

A Yes, yes. 10:36:46

Q Did any of the three plaintiffs in these cases 10:36:49

have a preceding myelodysplastic syndrome? 10:36:52

A Not that I recall, no. 10:36:56

Q Have you read the depositions of the 10:37:02

plaintiffs' hematologist in this case? 10:37:04

A Who is that, Dr. Gale? 10:37:08

Q Yes. 10:37:09

A I tried. It was a very difficult read. 10:37:12

Q What do you mean it was a difficult read? 10:37:16

A I mean it was just a very difficult read. It 10:37:18

was hard to follow. There was so much semantical 10:37:20

argument, there was very few questions that were 10:37:25

answered directly. 10:37:28

It was hard for me to get at what the defense 10:37:29

attorney was asking and what his answer was. Sometimes 10:37:33

it would go on for pages before he would ever answer it. 10:37:36

And then I had forgotten what the original question was. 10:37:39

I am just telling you for me, it was a hard 10:37:42

thing to read. 10:37:44

Q Okay. Do you recall what Dr. Gale's opinion 10:37:45

was as to whether Mr. Machado had a preceding 10:37:51

myelodysplastic syndrome? 10:37:55

A I do not. 10:37:57

Q Okay. Have you read the deposition of 10:37:58

Dr. Bennett? 10:38:00

A No. My understanding is that it is still going 10:38:01

on. 10:38:04

Q Okay. Is a preceding myelodysplastic syndrome 10:38:06

consistent with induced AML? 10:38:15

A With the caveat that we know less about what a 10:38:18

benzene induced AML is going to look like, I think that 10:38:21

it is, yes. 10:38:25

Q Is the absence of a preceding myelodysplastic 10:38:25

syndrome consistent with a benzene induced AML? 10:38:27

A With the caveat that I just described, that we 10:38:33

know less about the pathology of these diseases, I would 10:38:35

say that that is inconsistent with a benzene induced 10:38:38

AML, the absence of a preceding myelodysplasia. 10:38:42

Q Have you read the literature regarding that 10:38:51

particular issue? 10:38:55

A I have. 10:38:55

Q And what does -- you have read Aksoy about 10:38:57

that? 10:39:03

A Uh-huh. 10:39:03

Q And what does Aksoy say about that? 10:39:04

A When Aksoy was writing, I am not even sure he was using the terminology myelodysplasia. 10:39:07 10:39:10

Q Isn't it true that Aksoy -- well, Muzaffer Aksoy was a Turkish hematologist who saw a lot of benzene induced leukemias and other diseases from Turkish shoe workers who were exposed to benzene; right? 10:39:13 10:39:15 10:39:20 10:39:25

A I wouldn't agree with that per say. I would agree that he was a Turkish hematologist that reported on diseases that he believed might have been associated with the benzene exposure. 10:39:29 10:39:32 10:39:37 10:39:39

Q Okay. And isn't it true that he concluded that you didn't have to have a preceding myelodysplastic syndrome for benzene to cause leukemia? 10:39:41 10:39:46 10:39:50

A I think he concluded that many of the cases that he attributed to benzene had a pre-leukemic phase, which is what he called it. He was not using the terminology that we are using today. 10:39:53 10:39:56 10:40:00 10:40:03

Q Okay. 10:40:05

A And I think the pre-leukemic phase, based on current hematological diagnostic criteria would probably be myelodysplasia, but it didn't have to be there, no. 10:40:06 10:40:09 10:40:14

Q That is what he concluded? 10:40:18

A Yes. 10:40:19

Q All right. And are there other researchers who have concluded likewise with Dr. Aksoy that you don't have to have a preceding pre-leukemic or myelodysplastic phase to have a benzene induced leukemia? 10:40:19 10:40:26 10:40:29 10:40:34

A My appreciation of the literature is that that is more commonly seen than not. 10:40:39 10:40:42

Q Move to strike as non responsive. 10:40:44

What is the answer to my question, doctor? 10:40:46

A I thought I answered your question. 10:40:48

Q No. I will ask the court reporter to read it back and if you could answer that question. 10:40:51 10:40:53

A Okay. 10:40:54

(Record read.) 10:40:54

THE WITNESS: Well, all I can answer is what other researchers have published and what I have been able to glean from their publications. 10:41:22 10:41:23 10:41:26

Q BY MR. METZGER: I am not asking you to interpret their publications. I am asking you whether you are aware of any other researchers like Dr. Aksoy who have concluded that you don't have to have a preceding pre-leukemic or myelodysplastic phase to have a benzene induced leukemia? 10:41:29 10:41:31 10:41:34 10:41:37 10:41:41 10:41:48

A I can't put my finger on one, but I wouldn't argue that they are out there. 10:41:50 10:41:53

Q Okay. Regarding the six -- I think it was six 10:41:54

cases in which you have given depositions this year regarding benzene and AML or other hematologic diseases, in every one of those cases was it your opinion that the plaintiff was not exposed to enough benzene to cause the disease? 10:42:11 10:42:15 10:42:25 10:42:28 10:42:31

A No. Sometimes my opinion was that the exposure was irrelevant because no dose of benzene can cause that disease. 10:42:32 10:42:35 10:42:37

Q Okay. So let me ask that then with respect to the AML cases in which you gave depositions this year, is it true that in every one of those cases, you have testified that the benzene exposure that the plaintiff had was insufficient to cause the plaintiff's AML? 10:42:38 10:42:43 10:42:49 10:42:56 10:42:59

A In the cases where that has been discussed, that has been my conclusion, yes, that none of these individuals had sufficient benzene exposure to bring them up to where I would consider their risk to be significantly elevated. 10:43:07 10:43:09 10:43:12 10:43:16 10:43:19

Q What do you mean by significantly elevated? 10:43:23

A We have already discussed that. It is a statistically significantly elevated finding in the quantitative literature that is consistent among different studies. 10:43:26 10:43:29 10:43:32 10:43:36

Q Okay. Now, expanding the question beyond just this year, in all the cases in which you have testified 10:43:37 10:43:42

where the plaintiff had AML, was it your opinion that 10:43:47
the plaintiff had not been exposed to enough benzene to 10:43:50
cause his AML? 10:43:54
A Yes. 10:43:55
Q And in all of the cases where the plaintiff did 10:43:57
not have AML, was it your opinion that benzene didn't 10:43:59
cause the disease because it doesn't cause the disease 10:44:02
in your opinion? 10:44:05
A Out of the ones that I have been deposed in, 10:44:10
yes, that is true. 10:44:12
Q What percentage of the cases that you consult 10:44:24
on are you actually deposed in? 10:44:27
A From 2008 it was pretty high, which is why I 10:44:32
have given five or six depositions. 2007 I probably did 10:44:35
more litigation type work, but I only gave one or two 10:44:42
depositions. They just didn't go very far. So it's 10:44:45
hard to say. 10:44:47
Q Okay. 10:44:49
A I have been very busy this year so I really 10:44:50
have not done as much litigation work as I have in other 10:44:53
years. 10:44:57
Q Well, how much work do you do in other years? 10:44:59
A Oh, I try to keep it about 25 or 30 percent of 10:45:03
my professional time spent. And that is going to vary 10:45:07
depending on the duration of time that you are looking 10:45:11

at. It gets busy, then it goes away, and then it gets busy again. 10:45:17 10:45:19

Q Okay. 10:45:19

A But I think over the course of my career since I started testifying, I think that is a pretty fair guess. 10:45:20 10:45:22 10:45:25

Q And this litigation work that you do which compromises about 25 -- you try to keep about 25 to 30 percent of your time, what percentage of your income does that work generate? 10:45:26 10:45:28 10:45:32 10:45:35

A That is a good question. Probably more. 10:45:40

Q More than what? 10:45:44

A Probably would be more than 25 or 30 percent because a lot of my time -- like this past fall I spent quite a bit of time teaching a toxicology class, but the university pays me very little. 10:45:46 10:45:48 10:45:52 10:45:57

So if you average it all out, then the litigation might be 40 or 50 percent of my, you know, total income. 10:45:58 10:46:00 10:46:03

Q Okay. It's a good gig? 10:46:04

A What is that, the teaching? I like teaching. 10:46:09

Q Okay. Let's see. Let's go back to your opinions. 10:46:14 10:46:24

You mentioned at one point in your testimony Glass -- I think you said the Glass study. 10:46:29 10:46:34

What study were you referring to? 10:46:38

A The publication from 2003 that she published 10:46:41
from the Health Watch Series. 10:46:47

Q Is that that article in the "Journal of 10:46:49
Epidemiology" -- 10:46:52

A Yes. 10:46:54

Q -- entitled something like "Low Dose Exposure 10:46:55
to Benzene and Leukemia," something like that? 10:46:58

A That sounds right. I have it around here, but 10:47:02
yes, that sounds -- 10:47:05

Q You brought that with you? 10:47:05

A Uh-huh. 10:47:07

Q Yes? 10:47:08

A Yes. 10:47:08

Q And is that something that the attorneys gave 10:47:14
you or something that you were familiar with before this 10:47:16
case? 10:47:18

A I was familiar with it before. 10:47:19

Q Okay. There is also a very large report that 10:47:22
was published. It's the case control study of the 10:47:28
Australian cohort. 10:47:37

Do you know what report I am talking about? 10:47:39

A No. 10:47:41

Q You are aware that there are the annual -- not 10:47:45
necessarily annual but periodic reports, Health Watch 10:47:51

10, 11, 12 out of Australia? 10:47:55

A Yes. 10:47:58

Q Are you aware that there was also a large 10:47:58

report which is a case control study entitled, "Lympho 10:48:01

Hematopoietic Cancer in the Australian Cohort," 10:48:04

something of that effect? 10:48:08

A I am very familiar with the technical report 10:48:10

that has that title. 10:48:13

Q That report. 10:48:14

A Okay. 10:48:15

Q Okay. So you have read that report? 10:48:15

A Yes. 10:48:17

Q And is that something that the attorneys gave 10:48:17

you to read for this case? 10:48:19

A No. 10:48:20

Q You were familiar with that before? 10:48:20

A I was. 10:48:21

Q Okay. I want to ask you some questions about 10:48:23

the subjects that Mr. Carr mentioned he might be asking 10:49:09

you about. One of those was -- I think he mentioned 10:49:17

lifetime cumulative dose of benzene. 10:49:23

Do you have an opinion -- well, I think you 10:49:31

mentioned earlier that benzene is in some food items and 10:49:34

it's in the air that we breathe; correct? 10:49:38

A Yes. 10:49:41

Q Do you have an opinion whether the cumulative dose of benzene that we all receive from inhaling the air wherever we live is a cause of leukemia? 10:49:41 10:49:46 10:49:50

A It is my opinion that it is not a cause of leukemia. 10:50:02 10:50:05

Q And why do you consider that not to be a cause of leukemia? 10:50:06 10:50:09

A Because it takes a lot more benzene to give a person leukemia than what you are going to be exposed to from just background living in an urban environment. 10:50:11 10:50:14 10:50:17

Q Do you know what a lifetime cumulative dose of benzene is from inhaling the air in California? 10:50:23 10:50:26

A I don't off the top of my head. It wouldn't be that difficult to figure out, but no, I don't. 10:50:31 10:50:34

Q Okay. In any event, it is your opinion that the -- even though we are constantly exposed to benzene by inhaling it in the air, that that low level exposure to benzene continuously throughout our life doesn't significantly increase the risk of leukemia? 10:50:36 10:50:41 10:50:44 10:50:49 10:50:54

A That is my opinion, yes. 10:50:59

Q Okay. Mr. Carr said that he was going to ask you to testify about hematotoxicity and genotoxicity. 10:51:29 10:51:33

What are your opinions about that? 10:51:40

A I don't know. It would depend on what you are asking. I mean I don't -- ask a question and I will try 10:51:44 10:51:47

to answer it, but I am not sure what -- that's just very vague. 10:51:52 10:51:58

Q I agree, it is, and that is what he said so... 10:51:58

A Right. 10:52:01

Q So as you sit here right now, are there any particular opinions regarding the hematotoxicity of benzene that you have formed for this case? 10:52:02 10:52:05 10:52:07

A Well, I mean I think part of it might be that low levels of benzene or, you know -- that is debatable what low is, but levels of benzene have been shown to cause cytopenia in some exposed workers; and that that does not necessarily imply that those individuals are going to have an increased risk of AML. 10:52:11 10:52:18 10:52:23 10:52:26 10:52:31 10:52:34

And you cannot extrapolate and say since you see a subtle change in their peripheral lymphocytes, that also herego means that that person is at an elevated risk of AML; that those are different. 10:52:37 10:52:40 10:52:44 10:52:48

With the genotoxicity, I am assuming we were talking about the doses of benzene that have been shown to cause cytogenetic damage and those are pretty high. 10:52:52 10:52:55 10:53:00

Q How high? 10:53:05

A Well, as far as the in vitro studies, it is really difficult to say. I mean it would be difficult to say how much benzene you would have to be exposed to to result in in vitro concentrations of these 10:53:07 10:53:10 10:53:13 10:53:16

metabolites in the bone marrow, so that is a really hard extrapolation. 10:53:20 10:53:25

When they have looked at air concentrations, Martin Smith's group has published two or three papers out of China and it was 31 part per million that they -- above and below that they made comparisons with and found a statistically significant difference. 10:53:26 10:53:29 10:53:33 10:53:35 10:53:39

Q That shows a dose response relationship, doesn't it? 10:53:43 10:53:45

A It shows that they saw an increased risk of these cytogenetic changes in individuals that had a time weighted average of something greater -- eight-hour time weighted average of greater than 31 part per million. 10:53:46 10:53:48 10:53:50 10:53:52

Q Those studies did not show that there was no risk below 31 parts per million, did they? 10:53:56 10:54:00

A The group that they compare the upper with was the lower and it was a statistically significant difference. They didn't use the lower and compare that to someone that had even lower exposures than that. 10:54:05 10:54:08 10:54:12 10:54:14

Q Do you interpret that study to mean that the authors concluded that exposures below 31 parts per million do not cause cytogenetic damage? 10:54:19 10:54:22 10:54:28

A I do not think that that is what they concluded. 10:54:31 10:54:33

Q Okay. 10:54:34

A I think what they concluded was that there was a statistically significant increase above 31 PPM. 10:54:34 10:54:36

Q And in the Australian -- well, Dr. Glass' paper which you have mentioned, she compared different exposure levels of workers in the cohort with the risk of leukemia, did she not? 10:54:41 10:54:50 10:54:57 10:55:03

A Sure. 10:55:07

Q Right. 10:55:08

A Both -- both on an intensity level as well as a cumulative part per million basis. 10:55:09 10:55:13

Q Right. 10:55:15

And let's talk about the intensity. 10:55:16

What that showed was that -- that analysis showed that exposures to benzene of an intensity of just .1 or .2 parts per million significantly increased the risk of leukemia, did they not? 10:55:19 10:55:22 10:55:31 10:55:36

A I think that would be greatly over-interpreting that finding. 10:55:38 10:55:41

Q There was that finding in that data, was there not? 10:55:43 10:55:46

A That number was there, yes. 10:55:47

Q Okay. And they also showed that the rate of leukemia increased with increasing intensities of exposure, did they not? 10:55:48 10:55:56 10:56:02

A That is not true. That would be why I am 10:56:04

saying your -- that question is over-interpreting the data. And in the next group up, .2 to .4, it went down and was not statistically significant. 10:56:07 10:56:11 10:56:16

Then .4 to .8 on a time weighted average was back up. And then .8 to 1.6, it was lower than all of the three previous intensity measurements. That is a very quirky dose response. 10:56:19 10:56:23 10:56:27 10:56:31

Q Was there a statistically significant trend? 10:56:34

A I don't recall that being done. And the other point for those measurements is that that was total leukemias; not AML. 10:56:38 10:56:42 10:56:47

She also did a cumulative exposure for ANLL -- AML, we use them interchangeably -- but did not have the intensity data for that. 10:56:51 10:56:56 10:57:02

Q Are you aware of any epidemiologic studies outside of the workplace that have evaluated the risk of leukemia from benzene exposure? 10:57:10 10:57:18 10:57:26

A Well, I am aware of a lot of studies that have attempted to make associations to environmental levels of benzene and childhood leukemia, other diseases. 10:57:35 10:57:39 10:57:43

They all suffer significantly from having very poor to no exposure quantification at all. So to take that data and try to, you know, make firm conclusions on this environmental study, it is impossible to do. 10:57:53 10:57:57 10:58:01 10:58:06

Q Okay. Dr. Pyatt, as you sit here today, do you 10:58:13

know what caused the chromosome 5 and 7 abnormalities of Mr. Pearl? 10:58:17 10:58:23

A I do not know. It was just part of the basic pathology that lead to his AML. 10:58:24 10:58:31

Q Do you know what caused the chromosome 5 and 7 abnormalities of Mr. Machado? 10:58:33 10:58:36

A And as you ask about 7 in Mr. Pearl, I am still not convinced that it was there, but I will take your word for it. I don't know for Mr. Machado if -- 10:58:39 10:58:43 10:58:46

Q There was a marker 7. 10:58:49

A There was, okay. 10:58:50

Q Do you agree that the 5Q minus abnormality which both Mr. Pearl and Mr. Machado had was significant in the development of their leukemias? 10:58:55 10:59:01 10:59:11

A Good question. I think speaking on behalf of my understanding of the scientific and medical literature, those are non random cytogenetic changes. So yes, they played some role in the development, the pathogenesis of their AML. 10:59:18 10:59:23 10:59:27 10:59:31 10:59:37

Janet Rowley published some papers in the late '80s and it was followed up by Michelle Le Beau and Larson and others in the '90s where they tried to clone that piece of the chromosome to try to understand what was going on there that would lead people that had that deletion to develop AML with the caveat that the 5Q 10:59:38 10:59:41 10:59:45 10:59:48 10:59:50 10:59:54

minus syndrome doesn't do that. 10:59:58

But they found hematopoietic growth factors, 11:00:00
hematopoietic growth factor receptors, lots of things 11:00:05
that at least peripherally make sense in terms of the 11:00:08
pathogenesis of a hematopoietic disease. 11:00:12

So yes, I think that it's likely that that 11:00:17
played a role in the development of their disease. It 11:00:19
wasn't just a random cytogenetic change. 11:00:21

Q And do you agree that the monosome 7 that Mr. 11:00:24
Machado had also played a role in the development of his 11:00:28
AML? 11:00:33

A I would make the same argument that yes, most 11:00:35
people would think -- my interpretation of the 11:00:38
scientific literature is that most authors think that it 11:00:40
might. 11:00:44

But monosome 7 is also frequently seen as an 11:00:44
evolutionary event, so it may not be causative. It may 11:00:48
have arised -- it may arise in the leukemia through its 11:00:53
development and evolution and may not have had anything 11:00:57
to do with the original transformation. So the data is 11:01:01
less clear on that one. 11:01:03

They have also cloned out the whole chromosome 11:01:05
and there is nothing on there that really makes sense, 11:01:09
so they don't know why chromosome 7 -- my interpretation 11:01:11
of the scientific literature is that no one can really 11:01:14

define what it is on chromosome 7 that is associated with AML. 11:01:17 11:01:20

Q Now, let's talk more generally about chemically induced AML, which would include the chemotherapeutic drugs which you mentioned. 11:01:23 11:01:46 11:01:51

Okay? 11:01:53

A Okay. 11:01:53

Q And perhaps I will ask it generally as to chemically induced AML, but if you feel to answer the question you need to specifically refer to alkylating induced AML, feel free. 11:01:55 11:02:00 11:02:05 11:02:08

A Okay. 11:02:11

Q But here is the question: Can you approximate what percentage of people or patients who have AML with abnormalities of chromosome 5 and chromosome 7 have a history of exposure to chemicals? 11:02:12 11:02:16 11:02:29 11:02:37

A I could not do that sitting here. 11:02:52

Q Okay. Then let's talk specifically about alkylating induced -- alkylating agent induced AML. 11:02:55 11:03:00

A Okay. 11:03:06

Q What does the literature indicate as far as what percentage of patients who have abnormalities of chromosome 5 and 7 in AML have prior alkylating agent therapy? 11:03:07 11:03:10 11:03:18 11:03:24

A Well, I think the literature would support that 11:03:28

those are frequently seen cytogenetic changes in AML associated with chemotherapy. 11:03:33 11:03:38

If you looked at all of the AML's across the country that have a 5 or a 7 or both -- I don't know what that percentage is -- how many of those you could then track back and say these diseases are related to their chemotherapy. 11:03:40 11:03:44 11:03:48 11:03:52 11:03:55

Q Okay. 11:03:57

A I wouldn't mind taking a quick break, if that is all right. 11:04:10 11:04:13

Q Yeah, let's do that. 11:04:14

THE VIDEOGRAPHER: Want me to change out tapes, counsel? 11:04:16 11:04:18

THE WITNESS: Are we done? 11:04:19

MR. CARR: Yes. You are off the record. 11:04:21

MR. METZGER: Yes, change out the tape, sure. 11:04:23

THE VIDEOGRAPHER: Now going off the record. Time is 11:04 a.m. This is the end of Volume 1, tape No. 2. 11:04:24 11:04:26 11:04:31

(Brief recess.) 11:04:32

(Mr. Carr left the proceedings.) 11:11:31

THE VIDEOGRAPHER: We are now back on the record. Time is 11:11 a.m. This is the beginning of Volume 1, tape No. 3 in the deposition of Dr. David Pyatt. 11:11:55 11:11:55 11:11:58 11:12:02

Counsel? 11:12:03

(Deposition Exhibit 3 was marked for identification.) 11:12:03

Q BY MR. METZGER: Dr. Pyatt, I am showing you what has been marked as Exhibit 3. 11:12:03

Would you tell me what that is? 11:12:09

A This is a list of topics that I thought might be important and that I provided quite a bit of literature that they sent to you sometime, this weekend or whenever, but they would all be -- would fit under one of these categories, the literature that I submitted. 11:12:11

Q Okay. Do you have a list of that literature? 11:12:35

A I don't. It is pretty expansive. 11:12:38

Q Well, okay. I did receive a CD, I believe, and maybe two. All right. 11:12:40

We will mark as Exhibit 4 this document, which is a letter from the Jones Carr firm to me enclosing a DVD of materials upon which Dr. -- it says Dr. Pyatt is relying on. We will find out about that. 11:12:58

(Deposition Exhibit 4 was marked for identification.) 11:13:01

Q BY MR. METZGER: Would you take a look at this and tell me if some of the literature that you are indicating is on here? 11:13:04

A On this first page is -- well, actually this is all stuff that they sent me. These were Becky exhibits. I don't know what those are. 11:13:39 11:13:43 11:14:01

Q Well, let me tell you what -- first of all, what this is. When I received the disk, I asked some technical person to merely print out lists of directories of what was on the disk and that is what this is. 11:14:04 11:14:07 11:14:10 11:14:13 11:14:16

And what I'd like to know is the literature that you are referring to on Exhibit 3, is that -- does that appear to you to be included here? 11:14:18 11:14:20 11:14:26

A No. 11:14:30

Q Oh. 11:14:31

MR. FOLEY: I will represent that there are two different productions on two different disks sent to your office, one on the 12th and one on the 13th. 11:14:32 11:14:34 11:14:36

I think the stuff on the 13th were the articles that the doctor was just testifying about or referring to. 11:14:39 11:14:42 11:14:45

MR. METZGER: Are you saying that there is another disk that I don't have here? 11:14:45 11:14:46

MR. FOLEY: Correct. 11:14:49

MR. METZGER: And I haven't reviewed for this deposition. I only was aware of one disk. 11:14:49 11:14:50

MR. FOLEY: Okay. 11:15:00

MR. METZGER: Do you have a copy of the second disk? 11:15:02 11:15:03

MR. FOLEY: It was -- 11:15:04

THE WITNESS: I put it on a thumb drive because my hard drive -- my CD burner broke last week and I couldn't -- I mean I was trying to send it to their firm and I couldn't. 11:15:05 11:15:07 11:15:11 11:15:15

So finally on Friday I put it onto a thumb drive. 11:15:16 11:15:20

Q BY MR. METZGER: Do you have that with you? 11:15:20

A Uh-huh. They gave it back to me. 11:15:21

MR. FOLEY: Actually I think I have a copy of the disk. It was sent by FedEx to your office on December 13th. 11:15:23 11:15:25 11:15:30

MR. METZGER: Well, I do need it. The question is how do I -- 11:15:31 11:15:33

THE WITNESS: You can have the thumb drive. Just send it back to me when you are done. 11:15:36 11:15:38

Q BY MR. METZGER: I appreciate that. I mean whether it is on a disk or a thumb drive, that -- either one is fine. 11:15:41 11:15:42 11:15:45

I am just trying to figure out how I could ask you about it because I don't know what is on the disk or thumb drive. 11:15:47 11:15:50 11:15:55

MR. FOLEY: Let me do this: Let me see what's 11:15:55

on the -- let's go off the record for a minute. Is that okay or do you want to stay on? 11:15:56 11:15:57

MR. METZGER: Sure, let's go off. 11:15:58

THE VIDEOGRAPHER: Now going off the record. The time is 11:15 a.m. 11:16:00 11:16:00

(A discussion was held off the record.) 11:16:02

THE VIDEOGRAPHER: We are now back on the record. Time is 11:19 a.m. 11:19:51 11:19:52

Counsel? 11:19:54

Q BY MR. METZGER: Okay. Dr. Pyatt, referring to Exhibit 3, this is a list that you prepared; right? 11:19:54 11:20:01

A Yes. 11:20:05

Q And at the top it says, "Literature List," and then in parenthesis "Sent." 11:20:05 11:20:08

Does that mean that the attorney sent this to you or you sent it to them or what does that mean? 11:20:11 11:20:14

A That means that I sent it to them to then forward on to you. 11:20:17 11:20:19

Q Okay. And the topics here are gasoline literature, mineral spirits/Stoddard solvent, benzene in solvents, AML literature, benzene and AML, competitive inhibition, Health Watch, plio. 11:20:20 11:20:29 11:20:34 11:20:39

Is that the pliofilm cohort? 11:20:43

A Yes. 11:20:47

Q China, is that the Chinese cohort studies? 11:20:47

A Correct. 11:20:49

Q What is the next one? 11:20:50

A Refineries. 11:20:51

Q Refineries studies? 11:20:52

A Yes. 11:20:54

Q Then mechanics, then organic solvents, and then benzene and cytogenetics? 11:20:54 11:20:57

A Correct. 11:21:00

Q So is this list an indication of literature that you have gathered, studied and compiled on these different topics? 11:21:00 11:21:05 11:21:12

A Correct. 11:21:13

Q And the thumb drive that you gave me, does it have directories which -- on it which match the subjects on Exhibit 3? 11:21:15 11:21:19 11:21:26

A I doubt it. It probably would have if I could take the folder because this matches the folders that I have on my hard drive. But then I opened up those folders and pulled out the studies and put them all on a hard drive, so it probably will not match up to this. 11:21:27 11:21:30 11:21:34 11:21:37 11:21:40

Q So are there any folders or directories on the thumb drive or is it just all of the studies themselves? 11:21:44 11:21:49

A I think it is just all of the studies. 11:21:54

Q Okay. 11:21:57

A And if it alphabetized them, it is going to be 11:21:58

a mess. Probably would have been better to keep them in 11:22:04
the folders, but it wasn't working when I was trying to 11:22:07
pull them over so ... 11:22:10
Q Do you have your computer here now? 11:22:11
A I don't. 11:22:13
Q All right. Well, I have asked my technical 11:22:13
person here to print out -- to copy that thumb drive on 11:22:17
to a disk and to print out the directory so we will see 11:22:23
what we actually have. 11:22:27
A Yeah. 11:22:28
Q I am gathering that you are intending to 11:22:30
testify about each of the topics that are on Exhibit 3? 11:22:33
A Not necessarily. These were just the topics 11:22:38
that I felt like were relevant and important to this 11:22:41
case. And my reliance on any of the literature and 11:22:44
those topics would be dependent on your questions and 11:22:51
where we went. 11:22:56
If we are going to talk about the pliofilm 11:22:57
cohort, then, you know, I wanted to have all of the 11:22:59
studies so that I could rely on all of the studies that 11:23:02
are associated with that cohort. 11:23:06
Q Okay. Well, are you relying on the studies 11:23:10
that are on the thumb drive in support of your opinions 11:23:16
in these cases? 11:23:19
A Yes. 11:23:20

Q So everything on the thumb drive you have read, reviewed, considered and are relying upon in support of your opinions in these cases? 11:23:22 11:23:26 11:23:29

A I think that is fair, yes. 11:23:31

Q Okay. I'm going to try to approach this kind of in a macro approach initially. And then when I get the list of articles, we might get a little more specific. 11:23:33 11:23:43 11:23:47 11:23:50

But let me ask you generally then, what is it about the gasoline literature that is on the thumb drive that supports your opinions in these cases? 11:23:51 11:23:53 11:23:59

A That one actually I brought a hard copy of that folder, so I have all of those studies. And I also have the benzene and AML studies as a hard copy binder because those seem to me to be the most pertinent and germane. 11:24:03 11:24:07 11:24:12 11:24:16 11:24:19

The gasoline, to answer your question, since that figured prominently in Dr. Nicas' exposure assessments, at least for Hazlehurst and Pearl, then I think it's instructive to the jury or whoever is thinking about this case, "Well, what does the gasoline literature say? What do those studies say that people who are highly exposed to gasoline in the workplace, do they have an increased risk of AML?" 11:24:20 11:24:23 11:24:29 11:24:32 11:24:36 11:24:40 11:24:42 11:24:46

And I think the answer is no, which is why the 11:24:48

ATSDR and OSHA and other regulatory agencies, none of them classify gasoline as a human carcinogen, even though it has one to two percent benzene in it. 11:24:51 11:24:57 11:25:00

So I do think that literature is germane to this issue. 11:25:05 11:25:09

Q Okay. Do you have that binder here on the gasoline literature? 11:25:10 11:25:13

A I do. 11:25:14

Q Let me see it, please. 11:25:15

I was hoping to find in this binder an index. There is none? 11:25:43 11:25:48

A I am not that organized, Mr. Metzger. Sorry. 11:25:49

Q You should have the attorneys do that for you. I am sure they would be willing to. 11:25:52 11:25:54

Okay. I am just going to take a quick look through this binder and ask you some questions without marking it as an exhibit because hopefully it's all going to be on the CD that is being burned. 11:25:59 11:26:02 11:26:05 11:26:08

A Yes, it is. 11:26:11

Q Okay. There is a tab here for "Bernardini 2005 Case Report" -- oh, I see. And you have the abstract of that? 11:26:15 11:26:24 11:26:29

A Yes. 11:26:30

Q Okay. 11:26:31

A And I haven't -- for whatever reason, didn't 11:26:33

track down and find the whole study. Then I put the abstract in there, but usually I try to get the whole study. 11:26:35 11:26:37 11:26:40

Q Have you read that whole study by Bernardini? 11:26:41

A I don't think I have, no. Since it was a case report, I wasn't as interested in it. 11:26:45 11:26:46

Q You have in here the Glass 2003 study, "Leukemia Risk Associated with Low Level Benzene Exposure"; correct? 11:27:10 11:27:13 11:27:15

A Yes. 11:27:17

Q That is the study that we were talking about before that you have read and considered before this case and for purposes of this case; correct? 11:27:17 11:27:19 11:27:22

A Correct. 11:27:25

Q May I? 11:27:59

A Sure. 11:27:59

Q I will keep it all in order. 11:28:00

A That is fine. I will figure it out. 11:28:02

Q Just do a few things. 11:28:05

Okay. So included in here you have some papers from the Tranguch gasoline spill, Hazelton study; correct? 11:29:51 11:29:57 11:30:02

A Yes. That should -- that is probably under the tab for Patel 2004. 11:30:03 11:30:05

Q So you are familiar with her article which was 11:30:07

published I think in -- what was it -- "Archives of Epidemiology" or -- 11:30:10 11:30:12

A Yeah, I don't remember the journal, but yes, I have read that study. 11:30:14 11:30:17

Q Archives of something, I think. 11:30:18

A Uh-huh. I think that's right. 11:30:20

Q Okay. Now, regarding the gasoline literature, what workers in the gasoline industry, if I can call it that, are most heavily exposed to benzene? 11:30:27 11:30:38 11:30:49

A I don't know the answer to that. I mean they have looked at service station attendants and their exposures are measurable. They are not really high, but they have gasoline exposure. 11:31:01 11:31:03 11:31:10 11:31:15

They have looked at mechanics. I would say probably the individuals that are likely to have the highest exposure to gasoline would be tank truck drivers when they were doing bottom loading type activities. 11:31:17 11:31:20 11:31:23 11:31:27

Q Okay. And how many -- 11:31:32

A I am sorry, top loading, top activities. 11:31:33

Q And regarding those particular workers, have there been epidemiologic studies that have evaluated their risk of leukemia? 11:31:37 11:31:41 11:31:45

A Yes. 11:31:46

Q How many? 11:31:47

A Rushton did it, Snyder did it. There is 11:31:50

some -- when you look at the different workers in the Elk Lodge, they looked at distribution, marketing workers. Calling them tanker drivers, I know Rushton did -- Leslie Rushton did, Rob Snyder did and Otto Wong has done it, so there is at least three big tank driver cohorts. 11:31:54 11:31:57 11:32:02 11:32:08 11:32:12 11:32:15

Q And you are familiar with all of those studies? 11:32:17

A I am. 11:32:19

Q And those studies showed an increased risk of leukemia, did they not? 11:32:19 11:32:21

A I disagree with that. 11:32:23

Q Okay. What about the Patel study, did that study show an increased risk of leukemia from environmental exposure to gasoline? 11:32:25 11:32:38 11:32:43

A I don't think the Patel study showed anything that -- it is hard to rely on that study for anything at all. The exposures were very low. 11:32:44 11:32:45 11:32:49

There was only two AML cases. There were no correlation between where they lived in the plume and what their exposures could have been. They were both in their 60's. 11:32:52 11:32:57 11:32:59 11:33:02

There were two smokers out of the four leukemias. She doesn't say which of the two were smokers. I mean that study really doesn't stand for much of anything in my view. 11:33:02 11:33:05 11:33:08 11:33:12

Q Were you involved in that case at all? 11:33:14

A No. 11:33:15

In that case, you mean the study? 11:33:15

Q The lawsuit. 11:33:17

A I didn't know there was a lawsuit. 11:33:27

Q Okay. Until we get that in here, you said you had some other binders. 11:33:28 11:33:34

What other binders do you have here? 11:33:35

A I brought one other one. I am sure you have seen all these, but this is the benzene and AML, so these are all of the quantitative epidemiology studies that I am aware of that have really looked quantitatively at benzene exposures and associated risks of either AML or leukemia, depending on the study. 11:33:37 11:33:40 11:33:44 11:33:49 11:33:52 11:33:56

Q Okay. And what other binders do you have? 11:34:04

A That is -- those are the only two that I was willing to drag out here. 11:34:09 11:34:12

Q Okay. Regarding -- looking at Exhibit 3, the second topic you have here is mineral spirits/Stoddard solvent. 11:34:13 11:34:18 11:34:21

Do you have any studies on that topic which address hematotoxicity or leukemia? 11:34:27 11:34:31

A Well, what I sent and what I have looked at is the ATSDR Talks Profile on Stoddard solvent and the general -- some database sort of collections of 11:34:38 11:34:42 11:34:49

literature on mineral spirits and Stoddard solvent. 11:34:56

The hazardous substance database has an entry 11:35:00

for those. There is some regulatory literature out of 11:35:06

EPA. They don't have an Iris entry but some -- so that 11:35:09

is what I have reviewed. They are -- 11:35:13

Q Well, are there any case reports included in 11:35:17

that material regarding workers who used mineral spirits 11:35:21

or Stoddard solvent and developed hematologic disease? 11:35:25

A There would not be a specific study included at 11:35:30

all. There would be just what was discussed within that 11:35:34

regulatory document. 11:35:37

Q So have you actually reviewed the case reports 11:35:39

of workers who used mineral spirits or Stoddard solvent 11:35:41

and developed hematologic disease? 11:35:44

A I can't think of one, no. 11:35:48

Q Okay. Are there any particular documents in 11:35:49

that collection on mineral spirits or Stoddard solvent 11:35:55

which address the historical benzene concentrations of 11:35:59

those products? 11:36:02

A I don't know the answer to that. I think they 11:36:09

do, but I am not positive. 11:36:11

Q All right. The third topic listed on Exhibit 3 11:36:14

is benzene in solvents. 11:36:19

What is that about? 11:36:21

A I don't have many because this is sort of a 11:36:23

little outside of my interest and area of expertise, but I had 10 or so studies that have evaluated the amount of benzene that is in various solvents; for example, what is in hydrotreated toluene now versus what might have been toluene -- what might have been in toluene 30 or 40 years ago, and that kind of thing. 11:36:26 11:36:31 11:36:35 11:36:40 11:36:45 11:36:49

Q Okay. Are any of the studies in that group studies regarding benzene in mineral spirits? 11:36:50 11:36:57

A I think so. I think probably that would have been where Joe Fedoruk's study would have been and perhaps some of the simulation studies for Liquid Wrench. So I think there would be some in there, yes. 11:37:03 11:37:06 11:37:11 11:37:16

Q Okay. The fourth topic on Exhibit 3 is the AML literature. This is what, studies about acute myelogenous leukemia generally? 11:37:22 11:37:25 11:37:31

A Yeah. This would be the one that would mess up probably our ability to go through and say, "This is this," because there is so many of them. 11:37:33 11:37:36 11:37:39

This is the literature that I have read and reviewed through my professional career that have to do with the development of AML and what people are thinking with regard to the pathogenesis of that disease. 11:37:42 11:37:44 11:37:47 11:37:50

Q The next topic, benzene and AML, that is this binder here? 11:37:53 11:37:57

A It is. 11:37:58

Q All right. The next topic is competitive inhibition. 11:37:58 11:38:03

Are you intending to render any opinions at trial in this case regarding competitive inhibition? 11:38:05 11:38:08

A I might be asked to explain the concept to the jury, in which case I would be happy to do that. I haven't seen any exposures from either expert of benzene and then the corresponding exposures that would come from toluene or other things in the solvents that would make me think that competitive inhibition was likely to be an issue. 11:38:13 11:38:17 11:38:21 11:38:27 11:38:31 11:38:34 11:38:40

Q Okay. 11:38:41

A But if -- if you are going to try to take -- not you, but if you try to take benzene in low levels in Stoddard solvent or Safety-Kleen or even gasoline up to where they are going to give a person an increased risk of developing AML, then you have to account for the other solvents that are going to go along with those. 11:38:41 11:38:43 11:38:49 11:38:52 11:38:55 11:38:59

At those levels of exposure, I do believe that competitive inhibition is an issue. 11:39:02 11:39:05

Q Well, let's see if we can determine whether this whole topic is an issue in this case. 11:39:08 11:39:10

You have read Dr. Nicas' reports regarding the exposures of these plaintiffs; correct? 11:39:12 11:39:17

A I have, yes. 11:39:22

Q At the doses reported in Dr. Nicas' exposure assessments, do you believe that competitive inhibition of the toxicity of benzene is at play? 11:39:23 11:39:28 11:39:36

A I don't know the answer, whether there is any interaction effect on enzymatic metabolism. But even if it's not, I don't believe those exposures are sufficient. 11:39:41 11:39:43 11:39:50 11:39:54

So I don't think that you have to decrease the toxicity of those exposures with competitive inhibition to render them safer. 11:39:55 11:39:57 11:40:02

So I don't know whether at those levels you see it or not, but I don't think it matters. 11:40:05 11:40:07

Q Well, that is what I am trying to get at. I mean assuming that the only evidence that the plaintiffs are going to introduce regarding the dose of these individuals at trial is the information contained in Dr. Nicas' reports. 11:40:13 11:40:14 11:40:19 11:40:24 11:40:29

For those doses, do you believe that competitive inhibition of the toxicity of the benzene by other organics is a relevant issue or not? 11:40:31 11:40:35 11:40:39

A Definitely it would not be a relevant issue for Machado. Probably would not be a relevant issue for Mr. Hazlehurst. If Mr. Pearl had exposures of .5 part per million benzene, then there would be accordingly higher exposures to these other solvents. It is possible that 11:40:47 11:40:50 11:40:55 11:41:07 11:41:14

there could be some interaction effects. 11:41:18

Q Okay. Anything is possible. 11:41:20

Are you prepared to tell the jury that it is 11:41:22
scientifically probable that there was a competitive 11:41:24
inhibition of the toxicity of benzene through other 11:41:28
organics in the solvent? 11:41:31

A I would not say that it is probable, no. 11:41:34

Q Would the same be true for Mr. Pearl's exposure 11:41:37
to benzene from gasoline? 11:41:41

Well, let me break that down. 11:41:47

Would the same be true for Mr. Pearl's exposure 11:41:49
to Safety-Kleen 105 solvent which contained gasoline 11:41:51
from carburetors that he cleaned in the parts washer 11:41:58
machine? 11:42:03

A I think I understand your question. I don't 11:42:03
think it matters. I mean I think from a -- 11:42:05

Q That is fair enough. That is all I need. 11:42:08

A Okay. 11:42:10

Q Now, there has been testimony that Mr. Pearl 11:42:11
also on occasion cleaned parts with gasoline in a 11:42:14
bucket, with a bucket of gasoline. 11:42:20

Do you have an opinion as to whether the 11:42:26
benzene to which he was exposed in that circumstance is 11:42:29
such that its toxicity would have been competitively 11:42:34
inhibited by other organics in the gasoline? 11:42:43

A I would -- I would say the -- answer it the same way, that it's possible, but I wouldn't say that it's probable. 11:42:51 11:42:56 11:42:59

Q Okay. Is there any other exposure scenario for any of the plaintiffs that you are aware of where you would actually say that competitive inhibition of the toxicity of benzene by other organics is actually scientifically probable? 11:43:01 11:43:05 11:43:07 11:43:12 11:43:15

A Not based on what I have seen at this point, no. 11:43:18 11:43:20

MR. METZGER: Well, if you see something else and you are going to change your opinion on that, counsel, will you let me know because otherwise I don't see any reason to ask him about it? 11:43:23 11:43:24 11:43:26 11:43:29

MR. FOLEY: I think that is our obligation. 11:43:31

MR. METZGER: Okay. So we can consider that topic closed out. 11:43:33 11:43:35

Q Health Watch? 11:43:39

A I have collection of the one that you mentioned as well as some others, the 9th Report, 11th Report, 13th Report, Technical Report, Glass' study, letters to the editor, Gun, and that came out later. 11:43:44 11:43:47 11:43:52 11:43:57

I have all of the cumulative literature that I have been able to find that have to do with the Australian Petroleum Institute's Health Watch Program. 11:44:00 11:44:03 11:44:06

Q And what relevance -- strike that. 11:44:11

What are you -- regarding that body of 11:44:14

literature, what is it that you are relying on from that 11:44:17

for your opinions in this case? 11:44:22

A The quantitative assessment between benzene 11:44:24

exposure and AML risk. 11:44:27

Q And which of those documents primarily address 11:44:30

that? 11:44:33

A Well, the technical report has a lot of 11:44:34

quantitative information in it, but I think it is 11:44:36

adequately summarized in Deborah Glass' work from 2003. 11:44:39

Q The pliofilm cohort, for what purpose are you 11:44:45

relying on the pliofilm cohort studies for your opinions 11:44:49

in this case? 11:44:55

A Same -- same answer. It is the quantitative -- 11:44:55

quantitative benzene exposures associated with risks of 11:44:59

AML, both from a cumulative PPM year basis as well as an 11:45:02

intensity part per million basis. 11:45:08

Q What is it about the pliofilm cohort that -- 11:45:15

what is it about the intensity of the doses in the 11:45:20

pliofilm cohort upon which you base your opinion? 11:45:23

A Well, Rob Snyder published a paper in 1996 11:45:27

where he went in and looked at what the time weighted 11:45:30

average exposures needed to be before there was an 11:45:35

elevation in total leukemia risk and in AML risk. 11:45:39

And there was no elevation at anything below about 20 part per million years -- I am sorry, 20 part per million. So his analysis of that cohort supports that you have to have exposures close to 20 part per million before you are going to see an increased risk of AML. 11:45:43 11:45:46 11:45:50 11:45:54 11:46:00 11:46:03

Q You when you say 20 part per million, are you saying intensity or PPM years? 11:46:03 11:46:07

A No, 20 part per million over eight-hour time weighted average. 11:46:10 11:46:13

Q I see. 11:46:14

A So there is both. There is PPM years in that analysis, but there is also intensity. 11:46:14 11:46:17

Q Were there any workers in the pliofilm cohort who had average intensity benzene exposures less than 20 part per million? 11:46:19 11:46:22 11:46:29

A Oh, sure, there were. 11:46:30

Q Really. 11:46:31

Who? 11:46:32

A Well, all of the dry side workers were far lower than that. 11:46:33 11:46:36

Q Dry side, were they even exposed? 11:46:37

A Well, you asked if there were workers in the cohort that had less than 20. I am telling you there were a large amount of workers that had less than 20. 11:46:40 11:46:42 11:46:44

Q Okay. The dry side workers have been used as a comparison group? 11:46:47 11:46:51

A Well, they are still part of the cohort and they still had exposures less than 20. 11:46:53 11:46:55

Q Fair enough. 11:46:58

Were there any benzene -- strike that. 11:46:59

Were there any workers occupationally exposed to benzene from pliofilm in the pliofilm cohort who had average intensity exposures less than 20 parts per million? 11:47:03 11:47:05 11:47:12 11:47:16

A Yes. 11:47:16

Q Who were they? 11:47:17

A I would have to go back and look at that study, but he -- in his analysis there are data points looking at groups of workers that had exposures less than 20 where there was no elevated risk. And then at 20 is where it went up. 11:47:18 11:47:20 11:47:26 11:47:29 11:47:32

And if their exposures were one part per million or half of a part per million, it doesn't matter whether it is the control or dry side worker. I mean if there is no elevated risk -- well, it wouldn't matter if it was the control, but that is not what they did. 11:47:33 11:47:36 11:47:41 11:47:44 11:47:48

Q So other than the Snyder study -- is this the Snyder 2005? 11:47:51 11:47:54

A 1996. 11:47:55

Q Okay. What other studies in that pliofilm body of literature are you relying on? 11:47:57 11:48:04

A Well, when you asked me earlier in this deposition what levels of benzene do I think result in an elevated statistically significant risk of AML, I think that is in large part driven by the pliofilm cohort and various exposure assessments and analysis of that cohort. 11:48:08 11:48:10 11:48:16 11:48:21 11:48:26 11:48:30

Q Okay. The China studies, those are -- what is that referring to? 11:48:34 11:48:42

A This is the collection of studies started in the late '80s -- might have even started earlier than that -- and went up to current for NCI in collaboration with Chinese Academy of Preventative Medicine and they published 15 or 20 papers looking at benzene workers in various industries in China. 11:48:44 11:48:48 11:48:53 11:48:58 11:49:02 11:49:05

Q Okay. Are there any papers in that collection other than the NCI Chinese Academy of Medicine papers? 11:49:08 11:49:12

A Well, the earlier ones didn't have NCI in them. 11:49:26

Q Okay. 11:49:30

A And then there is at least one fairly argumentative paper regarding the potential quantification of benzene that took place in that cohort by Budinski is the guy's name, you know, pointing out the uncertainties and the inconsistencies in how they 11:49:31 11:49:35 11:49:40 11:49:43 11:49:48

quantified benzene exposure, and he wasn't associated with either one so... 11:49:53 11:49:56

Q And what are you -- generally what are you relying on those studies for for your opinions in this case? 11:49:58 11:50:01 11:50:04

A The same as the first two, that there is quantification of a cumulative dose of benzene that is required to increase the risk of AML. 11:50:04 11:50:06 11:50:09

And there is evidence about the intensity or the time -- part per million in the air that is required in order to see an increased risk of AML. 11:50:12 11:50:15 11:50:19

Q You are familiar, of course, with the Hays 1997 study? 11:50:28 11:50:31

A Yes. 11:50:32

Q And that is one of these studies that you are referring to; correct? 11:50:32 11:50:35

A It is. 11:50:35

Q And are you aware that the California Department of -- the California Environmental Protection Agency did an analysis to actually refine what the exposures were to the -- that were reported in the Hays study? 11:50:36 11:50:37 11:50:44 11:50:50 11:50:58

A I don't know what CalEPA has done. 11:50:59

Q Are you familiar with the California -- CalEPA Public Health Goal document for benzene? 11:51:02 11:51:06

A I seem to have -- I recall reading it several years ago. 11:51:09 11:51:11

Q Okay. And do you recall reading the information in that document regarding what the average exposures of the workers reported in Hays 1997 as less than 10 parts per million actually were? 11:51:13 11:51:15 11:51:21 11:51:28

A No. 11:51:30

Q The refinery studies, what are you relying on those for? 11:51:43 11:51:45

A Well, we have already kind of talked about it. This is, you know, the Snyder, the Rushton, the Wong. Those are all sort of refinery petroleum worker studies. There is a lot of others that -- 11:51:48 11:51:51 11:51:54 11:51:59

Q Well, those were mentioned in the context of gasoline? 11:52:02 11:52:04

A Right. But they would be -- if I pulled out a binder for refinery workers, they would be in there too. 11:52:05 11:52:07

Q Okay. 11:52:12

A And this is -- and it's not -- this one is not complete. I mean there is so many of those. I don't have all of them, but I have a lot of them looking at what the risks of leukemia are within the refinery worker population. 11:52:12 11:52:13 11:52:15 11:52:19 11:52:22

Some of them are germane if they have -- well, I consider them germane if they have quantitated the 11:52:23 11:52:27

exposures within those cohorts. Sometimes they do; 11:52:30
sometimes they don't. 11:52:33
Q Okay. The next topic is mechanics. 11:52:34
What literature is that comprised of? 11:52:37
A 20 or so studies, cohort, small studies looking 11:52:42
at mechanics as a profession and their risks of 11:52:47
hematopoietic malignancies generally, AML specifically, 11:52:55
associated with being a mechanic. 11:53:00
Q So these are all epidemiology studies? 11:53:03
A Yes. There is one good review paper by Holtz 11:53:06
that would also be in that pile. 11:53:09
Q Okay. So they are all epidemiology studies 11:53:11
that you are talking about here? 11:53:18
A In mechanics, yes. 11:53:19
Q Okay. Have you reviewed the studies regarding 11:53:20
specifically benzene exposures of mechanics? 11:53:28
A I did not review those for this case, no, but I 11:53:34
have seen them through the years. 11:53:36
Q Which ones have you seen? 11:53:38
A I remember some by Latinon -- Latinon. 11:53:42
Q Any others? 11:53:49
A I can't think of them. 11:53:53
Q Have you reviewed the studies regarding 11:53:55
exposures to mechanics of benzene as determined by the 11:54:01
presence and quantification of benzene metabolites? 11:54:09

A It sounds familiar, but I can't put my finger on it right now. 11:54:15 11:54:19

Q Okay. Have you read the papers regarding -- have you read the papers which have evaluated genotoxicity in mechanics? 11:54:20 11:54:28 11:54:30

A I have seen a few. They are not in there. 11:54:33

Q Which ones are you aware of? 11:54:37

A I can't think of the authors, but I have seen a couple. 11:54:41 11:54:43

Q Have you -- okay. 11:54:51

Have you ever compiled a list of studies regarding -- epidemiology studies which are case control studies regarding solvents and AML? 11:54:56 11:55:00 11:55:07

A What do you mean by solvents? 11:55:18

Q I am talking about studies -- case control studies of AML patients which report the risks of leukemia or AML in relation to prior solvent exposure? 11:55:20 11:55:25 11:55:31

A There are studies that have done that. 11:55:39

Q Can you identify any of them? 11:55:41

A Not without looking at my list. 11:55:42

Q What list? 11:55:45

A Whatever the list you are going to provide me, what was in this organic solvent pile. 11:55:46 11:55:47

Q Oh, I see. Okay. 11:55:51

So if we move on then to the organic solvent 11:55:55

pile or the category, is that a collection of literature of organic solvents and hematologic disease? 11:55:57 11:56:02

A Yes. 11:56:08

Q And what are you relying on that for? 11:56:09

A Well, one is that it is difficult to -- to evaluate that literature because the term "organic solvent" can mean a lot of different things. 11:56:11 11:56:15 11:56:18

So sometimes there is chlorinated solvents; sometimes there are is not. Carbon disulfide, there is just all kinds of things that would be considered an organic solvent, so it is hard to use that literature. 11:56:20 11:56:22 11:56:29 11:56:31

But there are some where they discussed the risk associated with petroleum based solvents and I think those have some relevance to this case. 11:56:36 11:56:39 11:56:42

Q Which ones? 11:56:44

A We'd have to look at the list. 11:56:46

Q All right. Last category, benzene and cytogenetics, what does that comprise? 11:56:47 11:56:50

A That is every study that I know of that has looked at either chromosomal damage associated with benzene exposure historically or more recently specific chromosomal changes associated with benzene and/or its metabolites. 11:56:54 11:56:57 11:57:04 11:57:13 11:57:14

That includes workers, healthy individuals that have been exposed to benzene and looking at peripheral 11:57:16 11:57:20

lymphocytes. It includes the in vitro mechanistic work, all of it. 11:57:24 11:57:30

Q Okay. Are you familiar with studies which have evaluated genetic polymorphisms of susceptibility for benzene induced AML? 11:57:32 11:57:34 11:57:39

A Yes. 11:57:41

Q And have there been genetic polymorphisms identified which confer an increased susceptibility to development of AML from exposure to benzene? 11:57:42 11:57:48 11:57:52

A That last part, the answer is no, but I think that by extension it will probably be true. We have shown -- the scientific community has shown that an NQ01 polymorphism increases susceptibility to benzene induced hematotoxicity and increases susceptibility to alkylating agent induced AML. 11:57:55 11:57:58 11:58:03 11:58:08 11:58:13 11:58:19

So I believe that it would probably also induce increased susceptibility to AML, but that last piece has not been shown that I have seen. 11:58:21 11:58:24 11:58:30

Q How would you go about showing it? What kind of study would you have to do? 11:58:32 11:58:36

A Well, I think they would be -- I think that's probably something that either the NCI or the Shanghai Health Watch study in China would be looking at would be the proportion of patients with AML that have this polymorphism in the population versus those that don't 11:58:38 11:58:39 11:58:42 11:58:46 11:58:51

have disease. 11:58:54

Q Okay. And that is just one polymorphism, the 11:58:55

NQ01; correct? 11:59:05

A That's correct. 11:59:08

Q The other polymorphisms of susceptibility have 11:59:08

also been evaluated; correct? 11:59:12

A They have looked at various glutathione 11:59:14

transferases, epoxide hydrolase, myeloperoxidase. They 11:59:16

have looked at several. None of these really paint as 11:59:22

consistent a picture as the NQO1 does. 11:59:25

Q Now, if a person has the type of NQ01 11:59:29

polymorphism which confers susceptibility to benzene 11:59:43

induced disease, would that person develop or could that 11:59:50

person develop AML from benzene at a lesser dose than 12:00:01

someone who did not have that susceptibility? 12:00:06

A The mutations that confer the susceptibility of 12:00:16

mutation in the enzymatic activity of NQ01 is present in 12:00:22

five to 10 percent of the U.S. population; something 12:00:28

like 15 to 20 percent in the Chinese population. 12:00:30

So no, I think the answer to your question is 12:00:33

there is no evidence that that's true; that when we 12:00:37

looked at pliofilm and other cohorts that developed -- 12:00:41

where individuals developed AML from a certain exposure 12:00:44

to benzene, it is, in my view, likely that those 12:00:47

individuals were the ones that had this susceptibility 12:00:50

gene because they are the ones that got AML. Same likely with China. 12:00:53 12:00:56

Q That wasn't studied in those cohorts, was it? 12:00:58

A No. 12:01:01

Q So you don't know whether those -- it's actually possible that none of the pliofilm cohort workers had that susceptibility polymorphism, is it? 12:01:01 12:01:03 12:01:09

A I guess it's possible, but if five to 10 percent of the U.S. population have it, then you would figure 5, 10 -- I mean it's not new. 12:01:15 12:01:17 12:01:19

People in the fifties had it just like people in the 2000's have it. It is just that now we can identify it and look for it and back then we couldn't. 12:01:22 12:01:24 12:01:28

Q As you sit here today, you don't actually know whether any one of the pliofilm cohort workers who developed AML had that NQ01 genetic susceptibility polymorphism. True? 12:01:31 12:01:32 12:01:35 12:01:40

A No, they did not look. 12:01:42

Q You don't know? 12:01:43

A I don't know. 12:01:43

Q And that hasn't been evaluated in any cohort of benzene exposed workers, to your knowledge; correct? 12:01:44 12:01:48

A No, that is not correct. I mean they have looked at NQ01. In the Lan study they looked at NQ01 polymorphism. 12:01:53 12:01:55 12:02:01

Q I misspoke. 12:02:02

That has not been evaluated in any cohort 12:02:03

studies in connection with AML; correct? 12:02:07

A To my knowledge, that is true. That is why 12:02:09

that last piece is still an uncertainty. 12:02:11

Q Okay. All right. I think we have now the CD 12:02:15

and a list and it does appear to be just an alphabetical 12:02:23

list without directories -- well, actually not true. 12:02:29

We will mark this list as Exhibit 5. 12:02:47

(Deposition Exhibit 5 was marked for 12:02:49

identification.) 12:02:49

Q BY MR. METZGER: I will ask you to identify it 12:02:50

for me, please. 12:02:52

A This appears to be an alphabetized list of 12:03:23

studies that I put onto a thumb drive to send to you. 12:03:28

Q Okay. 12:03:33

A And what this last page is, I don't know what 12:03:34

this is. I don't recognize it and I don't recognize 12:03:38

this. This might be something that is just part of the 12:03:46

thumb drive, kind of its internal workings maybe. 12:03:48

Q Well, let's just leave it altogether and you 12:03:53

have identified it. Let's put a clip on it. 12:03:58

Okay. Do you have any upcoming depositions 12:04:06

this year or scheduled for early next year? 12:04:17

A No. 12:04:22

Q Any trials coming up? 12:04:23

A Well, allegedly the Stromberg case might be going to trial in March. I think this one is scheduled to potentially go to trial in April. 12:04:27 12:04:32 12:04:37

Q Okay. 12:04:40

A And the Baker deposition, that might happen sometime in '09 too, but that is all I can think of. 12:04:42 12:04:45

Q Okay. Do you have any papers that you have submitted for publication which have not yet been accepted for publication? 12:04:49 12:04:57 12:05:00

A No. 12:05:02

Q Okay. Are you -- have you done any work regarding the Chinese -- strike that. 12:05:04 12:05:10

Have you done any work regarding the Shanghai study sponsored by the consortium of petroleum companies? 12:05:16 12:05:19 12:05:23

A No, no. 12:05:24

Q You haven't been involved in that at all? 12:05:24

A Well, I was in Rich Irons' lab when it first came about, so I was around when they were discussing the feasibility. 12:05:28 12:05:31 12:05:35

And when Dr. Irons went over there to kind of get the lay of the land and look at the factories that would participate, as I recall I taught an immunology class on his behalf so that he could take the time off. 12:05:36 12:05:39 12:05:41 12:05:45

But as far as the scientific participation, no. 12:05:48

Q Okay. Have you seen any of the data from those 12:05:50

studies? 12:05:53

A No. Just only what has been in the published 12:05:54

literature. 12:05:56

Q Okay. Are you currently doing any consulting 12:05:57

work for industry outside of the litigation context? 12:06:05

A For any industry? 12:06:11

Q Well, for the petroleum industry, petrochemical 12:06:13

industry. 12:06:18

A I would say sitting here right now, no, I am 12:06:20

not. 12:06:24

Q Do you currently have any funding or grants 12:06:28

from any petroleum industry? 12:06:31

A No, not active. 12:06:35

Q What is the last project that you did for the 12:06:37

petroleum industry? 12:06:40

A We looked at the -- there have been some really 12:06:42

interesting analyses that have come out of UNC and David 12:06:50

Richardson looking at the pliofilm cohort and he has 12:06:56

done some really interesting things about time to tumor, 12:07:00

age of exposure. 12:07:03

And even before that came out, there was 12:07:05

interest within the industry, and I believe that it was 12:07:08

useful to do all along, and that is to take Pamela 12:07:13

Williams -- the Williams exposure estimates that she published in 2003 and see whether that -- that new, more rigorous exposure data for the workers in pliofilm, whether that changes the cancer potency or whether it changes really the dose response relationship. 12:07:18 12:07:21 12:07:28 12:07:32 12:07:36

And so we did have a small grant from API to take that data and to put it into a format whereby epidemiological analysis can be done. 12:07:38 12:07:42 12:07:48

And that was all we did was put it into spreadsheets from the Williams publication that then you can conduct epidemiological analysis on that data. 12:07:51 12:07:53 12:07:57

They have asked us to submit a proposal to then conduct that analysis on that data, which we are in the process of doing. 12:08:02 12:08:06 12:08:10

Q Who is the "we" you are referring to? 12:08:12

A The "we" -- the "we" would be Sean Hays, my partner, Lisa Howard, the three of us that represent Summit Toxicology. 12:08:14 12:08:17 12:08:23

And then this one is a little over our head as far as epidemiologic analysis concern so we have collaborated with David Garabrant at the University of Michigan and potentially Michael Goodman at Emory University. 12:08:25 12:08:28 12:08:31 12:08:34 12:08:39

Q Okay. And you have spoken about two projects, one which you have done and one which you are seeking to 12:08:39 12:08:44

do? 12:08:48

A Right. 12:08:49

Q For the project that you have done, what was the payment from the American Petroleum Institute for that work? 12:08:50 12:08:51 12:08:57

A Oh, what we charged them? What they paid us for it? 12:08:59 12:09:02

Okay, I got it. 12:09:03

Q I assume they were the same? 12:09:04

A Yeah. I understand. 12:09:06

Q If they were different, I will take both. 12:09:06

A No, I was thinking about something like pat on the head kind of payment. 12:09:08 12:09:10

We charged them $13,000, I think, to do that. 12:09:12

Q Okay. And you have submitted the proposal to API for the next phase? 12:09:15 12:09:19

A We haven't yet, but it's on my desk to do as soon as I am finished here. 12:09:21 12:09:24

Q Okay. And what is the estimated duration of that study that you are proposing? 12:09:25 12:09:27

A Well, it is kind of dependent upon our epidemiologic people, but I would say we would probably have some preliminary results in six or eight months. 12:09:32 12:09:35 12:09:39

So I am guessing a year -- it would take us a year to get from start to where we could have something 12:09:42 12:09:44

ready for publication. 12:09:47

Q What is the amount of funding that you are 12:09:49

seeking from API for that? 12:09:50

A I think the total budget is about 70,000. 12:09:52

Q Okay. What -- other than this project and the 12:09:57

past phase and the future phase which you are hopeful 12:10:07

will occur, what other work have you done for the API 12:10:09

since 2000? 12:10:14

A Since 2000? I guess the only other funded 12:10:18

project that came from API, and it was in conjunction 12:10:29

with this thing called the Benzene Task Force, which was 12:10:33

part of ACC, a lot of the same companies, but they 12:10:37

weren't all petroleum companies. They were chemical 12:10:42

companies that also had an interest in benzene. 12:10:45

We did a child specific risk assessment in the 12:10:48

VCEPT program -- you and I have talked about that 12:10:52

before -- that we presented to EPA a year ago. 12:10:55

Q And how much were you paid for that? 12:10:58

A I was paid -- personally was paid $20,000 for 12:11:01

my work on looking at the susceptibility of children to 12:11:08

chemically induced leukemias, which I published a couple 12:11:13

papers on. 12:11:17

I think Summit Toxicology probably had twice 12:11:18

that. Maybe 50 or 60,000. The whole project was very 12:11:22

expensive and I don't know what the whole budget was. 12:11:25

The exposure piece was very expensive. 12:11:28

Q Okay. Your affiliation with Summit Toxicology 12:11:31

is what? 12:11:37

A I am a principal, co-owner. I don't think I 12:11:38

have given myself a title, but Sean is the president so 12:11:43

I guess I can be the vice president. 12:11:47

Q Other than -- you mentioned the ACC. 12:11:51

Is that the American Chemistry Council? 12:11:54

A Yes. 12:11:56

Q Formally known as the Chemical Manufacturers 12:11:56

Association? 12:11:59

A Right. 12:12:00

Q And formerly known as the Manufacturing 12:12:00

Chemists Association? 12:12:03

A I think that is all true, yes. 12:12:04

Q All right. Have you done any other work for 12:12:06

that organization and any of its iterations, its 12:12:10

different names? 12:12:17

A The work that I have been doing on 12:12:18

formaldehyde, it was for the Formaldehyde Council, which 12:12:20

is kind of its own little business entity, but it is 12:12:24

part of -- well, I certainly think of it as part of ACC. 12:12:28

I have gone to and participated in scientific 12:12:32

meetings from the Olefins Panel, which is part of ACC 12:12:38

that have a long-standing interest in butadiene. 12:12:44

So I have been involved in looking at the new Czech studies that they are doing, the Houston Ship Channel study, just analysis of the data that is out there regarding butidiene. 12:12:47 12:12:52 12:12:56 12:13:00

THE VIDEOGRAPHER: You have three minutes, counsel, on the tape. 12:13:02 12:13:03

Q BY MR. METZGER: What funding have you received from Formaldehyde Institute or ACC for that? 12:13:04 12:13:06

A The Formaldehyde, they paid for that paper which I split with co-author. I think I probably got 25,000 for my work in there. And since I am a poor writer, it was not a very good paycheck for me. 12:13:10 12:13:17 12:13:22 12:13:27

For the Olefins group, really not much. They paid for my travel and my time to sit in these meetings and discuss what I know about butadiene. 12:13:34 12:13:39 12:13:44

Q Do you plan on attending the Benzene Conference in Munich this coming September? 12:13:48 12:13:51

A I do. I was part of the original planning committee for that. 12:13:53 12:13:55

Q Is anyone paying for your travel for that? 12:13:57

A I doubt it. 12:13:59

Q Okay. Do you have any other notes here that I haven't seen? 12:14:00 12:14:06

A No, just miscellaneous junk. 12:14:07

Q I think we need to take a tape change. Let me 12:14:10

just take a look at that while we are changing tape. 12:14:13

THE VIDEOGRAPHER: Now going off the record. 12:14:15

Time is 12:14 p.m. This is the end of Volume 1, tape 12:14:16

No. 3. 12:14:20

(At the hour of 12:15 p.m. the luncheon 12:14:20

recess was taken, the proceedings to be 12:14:20

resumed at 1:00 p.m.) 12:14:20

(At the hour of 1:05 p.m. the following proceedings were had at the same place with the same persons present.)

THE VIDEOGRAPHER: Now back on the record. Time is 1:03 p.m. This is the beginning of Volume 1, tape No. 4 in the deposition of Dr. David Pyatt. Counsel?

(Deposition Exhibits 6-15 were marked for identification.)

Q BY MR. METZGER: Are you ready to proceed, Dr. Pyatt?

A Sure, thanks.

Q Reviewing my notes from this morning --

THE VIDEOGRAPHER: Counsel, your microphone, I think it's on the floor there actually.

MR. METZGER: Yeah, thank you.

Q Reviewing my notes from this morning, you mentioned something I didn't ask you about, and I think that was that Mr. Machado had the Philadelphia chromosome abnormality?

A Correct.

Q Is that significant to you in your evaluation of causation in this case?

A To some extent. I mean there is no evidence that I have ever seen that benzene exposure has been associated with the 922 translocation. And I don't believe benzene exposure at any dose causes CML, which is likely to have the 922 translocation. 01:04:21 01:04:23 01:04:26 01:04:29 01:04:32

So it just makes Mr. Machado's presentation and his disease a little inconsistent with what a benzene or a chemically induced AML might look like. 01:04:35 01:04:39 01:04:45

Q Because he had the 922 translocation? 01:04:51

A Correct. 01:04:54

Q Is that the same thing as the Philadelphia translocation? 01:04:54 01:04:57

A Yes. 01:04:58

Q Okay. I guess while we are on this topic, were there any other hematologic, morphologic, immunophenotypic or cytogenetic abnormalities or features of Mr. Machado's AML that you considered to be inconsistent with benzene induced AML other than what we have already discussed? 01:04:59 01:05:17 01:05:27 01:05:30 01:05:34 01:05:40

A I think we have discussed it, lack of preceding myelodysplasia, but we have discussed that in some detail. 01:05:41 01:05:45 01:05:48

Q Same question for Mr. Pearl? 01:05:48

A Yes. The only characteristic of Mr. Pearl would be that the evidence -- my interpretation of the 01:05:56 01:05:59

scientific literature is that chemically induced leukemias are notoriously difficult to treat. 01:06:04 01:06:07

And his was treatable, but I am not positive if that is a totally fair comparison because a lot of the older studies they didn't have a well matched bone marrow transplant or a stem cell transplant that they could use as therapy. 01:06:11 01:06:15 01:06:18 01:06:22 01:06:25

So had they had that option available to them, then those leukemias -- those chemically induced leukemias might have been as treatable as Mr. Pearl. 01:06:27 01:06:32 01:06:35

So I don't believe that is necessarily that fair of a comparison. 01:06:38 01:06:41

Q Okay. All right. 01:06:43

You consider yourself to be an expert in benzene induced leukemia; right? 01:06:48 01:06:51

A Yes. 01:06:57

Q But since you are not a medical doctor, you don't see patients who have those diseases or treat them; correct? 01:06:58 01:07:00 01:07:03

A I do not, no. 01:07:04

Q Okay. And have you personally done -- been involved in any epidemiologic studies of benzene exposed work forces to assess leukemia in those work forces? 01:07:08 01:07:14 01:07:19

A Well, the answer is no, I haven't. 01:07:24

Q Okay. So have you ever seen a benzene induced 01:07:26

leukemia? 01:07:38

A There have been cases that people brought to me 01:07:42

where there was substantial exposure or the possibility 01:07:48

of substantial exposure and there were cytogenetics and 01:07:54

characteristics that I was unable to rule out. 01:08:00

Q But have you ever actually seen a benzene 01:08:07

induced leukemia and said, "Yes, this is a worker who 01:08:10

actually has it. I'm convinced"? 01:08:13

A I think those cases would be the closest. 01:08:16

Q But you never actually came out and said that, 01:08:20

"This worker has a benzene induced leukemia"; right? 01:08:22

A Well, I don't know what you mean by "came out 01:08:27

and said." I told the people that were talking to me 01:08:29

about this that there wasn't anything that I could do to 01:08:33

help them in this case. They are going to have to do 01:08:36

something else because I can't help them. 01:08:40

Q So this was a litigation matter? 01:08:41

A Yes. 01:08:43

Q How many times has that occurred? 01:08:44

A I can think of two. 01:08:48

Q And tell me what the presentations were of 01:08:51

these two workers. 01:08:56

Where you told -- you were speaking to the 01:08:59

defense lawyers; right? 01:09:02

A Yes. 01:09:04

Q Okay. And what were the presentations of these two workers that you told those defense lawyers that you didn't think that you could help them? 01:09:05 01:09:08 01:09:12

A That they both had ample opportunity for high dose exposure to benzene. They both used benzene in the workplace. They had sufficient exposures within the appropriate time window. 01:09:14 01:09:18 01:09:21 01:09:26

They had preceding myelodysplasias. They had cytogenetic changes that were consistent. Both were refractory to treatment. So those characteristics. 01:09:29 01:09:35 01:09:39

Q What were the cytogenetic changes that they had? 01:09:45 01:09:48

A One had monosome 7. And -- they both did. They both had monosome 7. 01:09:49 01:09:54

Q And so the loss of one of the chromosome 7's? 01:09:57

A That is correct. 01:10:03

Q Okay. All right. Let's take a look please at Exhibit 6. 01:10:13 01:11:09

And are these two pages typewritten notes that you made regarding Mr. Pearl? 01:11:09 01:11:09

A Yes. 01:11:34

Q And take a look at Exhibit 7. 01:11:38

Are those two typewritten pages notes that you made regarding Mr. Hazlehurst? 01:11:41 01:11:44

A Yes. 01:11:46

Q Did you also make some typewritten notes regarding Mr. Machado? 01:11:47 01:11:49

A Sure seems like I did. I don't know where they are. 01:11:51 01:11:54

Q Would you see if you can find those because I didn't see them in your materials there. 01:11:56 01:11:58

A It was mainly just so that I could -- well, this is the exposure stuff. And if they are not -- it's not on the table somewhere, it must not have made it into my bag coming out here. That one over there doesn't have anything in it. 01:12:01 01:12:09 01:12:34 01:12:36 01:12:41

Q Well, why don't we do this -- 01:12:45

A I can certainly send it to you, provide it to you. 01:12:47 01:12:49

Q Yeah. Why don't we do this: Why don't you send it to me. And if I feel that I need to ask you some questions about it, we will just have a short telephonic resumption of the deposition. 01:12:49 01:12:52 01:12:54 01:12:57

A Sounds fair. 01:13:00

Q Okay. All right. 01:13:00

So let's start now with Exhibit No. 6. You have here, "John Pearl, born in 1960." 01:13:10 01:13:19

Is there any significance of that to your analysis? 01:13:23 01:13:25

A No. I mean he was a relatively young person to 01:13:31

develop AML so I made note of that. 01:13:36

Q And does that, the fact that he was young, tend 01:13:40

to -- is that consistent with a chemically induced 01:13:46

leukemia as opposed to one that is not? 01:13:50

A Well, AML is a disease that the incident rate 01:13:57

increases precipitously with age. And so the incident 01:14:01

rate at people at John's -- at John Pearl's age have a 01:14:05

lower incident rate of leukemia than someone say 70, 65 01:14:13

or 70. 01:14:17

Q And does that tend to indicate that it would be 01:14:19

more consistent with chemically induced leukemia than an 01:14:25

age related leukemia? 01:14:29

A Yeah. The way you worded that question, I 01:14:35

think the answer is yes. 01:14:39

Q Okay. You also wrote that he was a nonsmoker. 01:14:40

What was the significance of that? 01:14:43

A That cigarette smoking is an established cause 01:14:45

of -- etiological risk factor for AML. 01:14:48

Q And "healthy guy," what is the significance of 01:14:54

that? 01:14:57

A Not -- there is no significance. I think he 01:15:00

said he was a weight lifter and just things that I 01:15:02

picked up from reading his medical records. 01:15:05

Q Okay. The fourth bullet point you have is, 01:15:07

"His original cytogenetic study was complex, including 01:15:21

minus 5 deletion (q1.1-33.)" 01:15:25

What was the significance that his original 01:15:32

cytogenetic study was complex? 01:15:35

A It was just taking note of what I saw in the 01:15:40

medical records. It wasn't necessarily significant or 01:15:43

not significant. It just was -- these were the things 01:15:46

that I picked up from reading his medical records. 01:15:50

Q Does a complex cytogenetic study or karyotype, 01:15:54

is that more consistent with a chemically induced 01:15:58

leukemia than one that is not? 01:16:01

A We talked about that. I think that when you 01:16:06

look at chemically induced leukemias, you will 01:16:08

frequently see complex cytogenetic picture, but there 01:16:12

are plenty of cases of AML where there are complex 01:16:16

cytogenetic changes where there is no known documented 01:16:20

exposure to anything. 01:16:24

So whether it is more or less consistent, I 01:16:25

couldn't say definitively. 01:16:27

Q We have already spoken about the minus 5. That 01:16:30

is the chromosome abnormality on chromosome 5 that we 01:16:33

have been talking about; correct? 01:16:37

A Yes. 01:16:38

Q Do you attribute any significance to the change 01:16:50

in his cytogenetic picture following chemotherapy? 01:16:53

A In relation to this case, no. I mean I think 01:17:01

that that is either -- it's either A, an evolutionary event in the development of his disease or it had something to do with his myeloablative therapy that they gave him for the stem cell transplant. 01:17:03 01:17:07 01:17:10 01:17:14

Q The last bullet point you have, "His benzene exposures per Dr. Nicas were driven by the introduction of gasoline into the parts washer while carburetors were being cleaned." 01:17:25 01:17:28 01:17:31 01:17:35

What is the significance of that? 01:17:35

A Just to remind me that that is what the -- Dr. Nicas' exposure was based on. 01:17:37 01:17:41

Q Was there any significance to the fact that Dr. Nicas, as you interpret it, found that the exposures were driven by gasoline in the parts washer? 01:17:48 01:17:49 01:17:55

A Well, yes, to some extent. I mean the gasoline literature that we have discussed, in my view, doesn't support a gasoline exposure increases one's risk of AML. 01:17:59 01:18:03 01:18:07

I don't believe there that there is enough benzene mineral spirits or Safety-Kleen or Stoddard solvent to increase one's risk of AML. 01:18:12 01:18:16 01:18:19

So you take a substance that has benzene in it, not enough, and you pour it into the parts washer, it just doesn't make sense to me. 01:18:22 01:18:24 01:18:29

Q Okay. Anything beyond that? 01:18:31

A No. 01:18:32

Q All right. Now, you have written some opinions here. Let's just take a look at those. 01:18:35 01:18:38

Your second opinion, "Mr. Pearl's exposures to benzene from SK 105 and parts washing are lower than his background exposures to benzene," did I read that correctly? 01:18:46 01:18:48 01:18:54 01:18:56

A Yes. 01:18:57

Q What are you referring to as the background exposures? 01:18:57 01:19:00

A I am referring to what Pat Sheehan calculated for his exposure quantification, working in the shop, just had background per Dr. Sheehan. 01:19:01 01:19:05 01:19:11

Q So you mean the benzene in the air in the shop as opposed to the benzene in the -- just in the environment? 01:19:17 01:19:20 01:19:26

A Well, I mean whatever Dr. Sheehan means. The exposure from the shop, exposure from washing parts in gasoline in a bucket, exposure to gasoline in a part -- well, so I guess it would be exposure to benzene in the shop per his calculations. 01:19:27 01:19:34 01:19:38 01:19:42 01:19:46

Q He also has benzene in the air, does he not, the ambient air? 01:19:48 01:19:50

A Yes. 01:19:53

Q So it would be those two? 01:19:54

A Sure. 01:20:00

Q Do you think it's reasonable that Mr. Pearl's exposure to benzene from the use of Safety-Kleen 105 solvent would be less than his exposure to benzene in the air that we all breathe? 01:20:04 01:20:10 01:20:12 01:20:20

A From the solvent, yes. That seems -- that seems plausible to me. 01:20:26 01:20:29

Q Why? 01:20:31

A Because there is not that much benzene in Safety-Kleen solvent. 01:20:31 01:20:34

Q Well, whatever amount there is, wouldn't that be additive to what's in the air? 01:20:36 01:20:39

A Well, if you want to look at his total cumulative exposure, I guess that's right. But I think what Dr. Sheehan did was try to separate that out and say what was his exposures to background, what was the additional contribution that his use of Safety-Kleen solvent might have made to his total overall exposures. 01:20:51 01:20:53 01:20:56 01:20:59 01:21:02 01:21:06

Q Oh, so -- 01:21:10

A All I am saying is that when you compare those, it is not totally out of line to think that his exposures to solvents in the parts washer was lower than his background levels. 01:21:12 01:21:15 01:21:18 01:21:21

Q Well, let's not talk cumulatively for a moment. Let's talk about real time. 01:21:22 01:21:25

When Mr. Pearl was washing parts in the parts 01:21:27

washer machine using Safety-Kleen 105 solvent, according to Dr. Sheehan, the concentration of benzene that he inhaled was less than the concentration of benzene in the air that we all breathe, wasn't it? 01:21:30 01:21:35 01:21:41 01:21:44

A I don't think that's the proper interpretation of these numbers. I think he did what I am saying, that he tried to calculate what the individual contributions would be from using the parts washer, using the parts washer with gasoline added, washing parts in a bucket with gasoline and other potential sources. 01:21:48 01:21:50 01:21:55 01:21:59 01:22:02 01:22:06

Q You have had a conversation with Dr. Sheehan, have you not? 01:22:09 01:22:11

A I have. 01:22:12

Q About an hour? 01:22:13

A Seems about right, yes. 01:22:14

Q And did you ask him about that during that conversation? 01:22:15 01:22:18

A No. 01:22:19

Q We will talk more about the conversation, but let's go on for now. 01:22:20 01:22:23

Your fourth opinion you write, "There were some features of Mr. Machado's -- wait a second. We are looking at the summary regarding Mr. Pearl and the fourth one, it says, "Mr. Machado." 01:22:39 01:22:42 01:22:50 01:22:53

A Oh, yeah. 01:22:55

Q Which one did you mean there? 01:22:56

A Yeah, this one was -- this one is for 01:23:02

Mr. Pearl. 01:23:05

Q So it should say "Mr. Pearl." That is what you 01:23:06

intended to say? 01:23:09

A No, because Mr. Pearl -- yes, yes, yes. 01:23:10

Q So correcting that, it says, "There were some 01:23:12

features of Mr. Pearl's AML that were consistent with 01:23:15

the chemically induced AML, but others that were not. 01:23:19

The cytogenetics is what one might expect. On the other 01:23:23

hand, chemically induced AML are notoriously difficult 01:23:29

to treat, although SCT was not available for many." 01:23:31

That's -- 01:23:36

A Stem cell transplant. 01:23:37

Q Okay. "Also, there was no evidence of a 01:23:39

preceding MDS. Based on these characteristics alone, it 01:23:41

would be too close to call." 01:23:46

Did I read that correctly? 01:23:49

A Yes. 01:23:50

Q What do you mean it would be too close to call? 01:23:51

A That if I didn't know anything about the case 01:23:53

other than the presentation of the disease, I would not 01:23:56

be able to say yes, this looks like a chemically induced 01:23:59

leukemia versus no, this does not look like a chemically 01:24:03

induced leukemia. It has characteristics of both. 01:24:06

When you factor in what his cumulative exposures were, then that totally pushes it toward the de novo and that benzene didn't cause it. That is what I meant. 01:24:09 01:24:11 01:24:17 01:24:20

Q Okay. In reaching your conclusion that benzene did not cause Mr. Pearl's AML, what standard are you applying? 01:24:23 01:24:51 01:24:55

A I don't understand that question. 01:24:56

Q Are you -- are you reaching that opinion to a scientific certainty? 01:24:57 01:25:00

A Yes. 01:25:04

Q Okay. Let's take a look at Exhibit 7 regarding Mr. Hazlehurst. You have that he was born 1948. 01:25:23 01:25:26

Any significance to that? 01:25:38

A Same that we talked about, just what his age was at time of diagnosis. 01:25:39 01:25:42

Q You note that, "For at least five years prior to his diagnosis, he routinely visited a doctor for pain medication and sleep medicine for chronic low back pain," is there any significance to your opinion? 01:25:45 01:25:47 01:25:52 01:25:55

A No. 01:25:58

Q Or the next sentence that says, "There was no evidence of nausea, headaches or other systemic symptoms during this time"? 01:26:01 01:26:03 01:26:08

A No. I mean the levels of exposure that both 01:26:09

experts -- exposure experts have talked about, I wouldn't expect there to be any systemic manifestations of toxicity to those. 01:26:11 01:26:14 01:26:21

Q Your next bullet point mentions that he was diagnosed in 2005 and then that he was experiencing shortness of breath following a tooth extraction and had an elevated WBC. 01:26:29 01:26:39 01:26:46 01:26:49

That's a white blood count; correct? 01:26:51

A As I recall, that was how it originally presented. 01:26:56 01:26:57

Q Is there any significance of that initial presentation for your opinion? 01:26:57 01:26:59

A Not the shortness of breath and the elevated WBC, that is what you would expect. We have already talked about the normal cytogenetics. 01:27:02 01:27:04 01:27:08

Q Okay. You noted he had no preceding MDS. 01:27:11

And then that he had a 20 pack-year history of smoking. What is the significance of that? 01:27:15 01:27:17

A As we discussed earlier, cigarette smoking is an established etiological risk factor for AML. 01:27:19 01:27:22

Q Okay. You note that in October 2006, there was one cell that was plus 13 and minus 19. And that by November 2006, there were two cells with trisome 13. 01:27:29 01:27:32 01:27:37

What is the significance of that? 01:27:41

A That there was evolution going on in his 01:27:43

disease or that there were additional cytogenetic changes associated with his induction therapy. 01:27:47 01:27:50

Q Which do you believe is responsible for those changes? 01:27:57 01:28:00

A I don't think you can definitively say, but I don't think you can rule out -- well, I know that you cannot rule out the possibility that it was related to the induction therapy. 01:28:01 01:28:03 01:28:06 01:28:10

Q Okay. So it could be either-or, either one or the other? 01:28:11 01:28:15

A I think that is fair. 01:28:16

Q Okay. Now -- 01:28:17

A I also pointed out that most cytogeneticists do not consider a single cell -- findings in a single cell to be evidence of a clonal lesion. 01:28:22 01:28:28 01:28:32

So the trisome 13 and the monosome 19 was observed in one cell as I recall, so that really isn't considered a positive finding. 01:28:34 01:28:37 01:28:41

Q In October, but then a month later they found the trisome 13 in two more cells? 01:28:42 01:28:45

A Right. So there probably most cytogeneticists would think, okay, there probably is something going on, but I think the diagnostic criteria is still three cells. They need three cells that have the same cytogenetic change to say that it was clonal. 01:28:48 01:28:50 01:28:54 01:28:58 01:29:01

Q Okay. Now, the purpose of chemotherapy is to destroy cancer cells; correct? 01:29:04 01:29:10

A Yes. 01:29:14

Q And some chemotherapeutic agents are specifically designed to knock out chromosome abnormalities. True? 01:29:15 01:29:20 01:29:27

A Some are specifically designed to target abnormal proteins that are associated with chromosomal abnormalities, yes. 01:29:35 01:29:38 01:29:41

Q Okay. And that has the effect of killing the cells that have those abnormalities so that you don't see those chromosomal abnormalities; right? 01:29:44 01:29:47 01:29:51

A Right, preventing those cells from proliferating and passing on that genetic instability. 01:29:55 01:29:57

Q So is it true that following chemotherapy -- well, in practice, a repeat cytogenetic study is done to see if chemotherapy is working; in other words, if those cytogenetic abnormalities that were there initially remain there? 01:30:02 01:30:04 01:30:14 01:30:19 01:30:23

A Yes. 01:30:24

Q So the -- at least the short-term effect of the chemotherapy is to reduce or eliminate the cytogenetic abnormalities? 01:30:24 01:30:29 01:30:37

A Well, I think that is a -- I think that is a side effect. That is a peripheral -- I mean what they 01:30:40 01:30:42

are trying to do is reduce the tumor burden of leukemic cells that are positive for that cytogenetic change. 01:30:45 01:30:49

Q Right. 01:30:52

And as a result, hematologists are pleased when they give chemotherapy to a patient and a subsequent cytogenetic analysis shows that prior chromosome abnormalities are no longer observed; right? 01:30:53 01:31:02 01:31:06 01:31:12

A That is an indication that they were successful in their original treatment, yes. 01:31:16 01:31:18

Q Right. 01:31:20

So isn't it true that the effect of the chemotherapy is actually to eliminate or reduce the chromosomal abnormalities rather than to produce them, at least short term? 01:31:21 01:31:26 01:31:32 01:31:35

A Well, I don't think that any hematologist or pharmacologist would say that they are trying to design drugs to cause chromosomal changes, but that doesn't change the fact that they do. 01:31:37 01:31:41 01:31:44 01:31:48

Q I understand. 01:31:49

What I am getting at is when an oncologist or hematologist gives a patient chemotherapy drugs, an AML patient, what they hope to see is that, if there were previously observed chromosomal abnormalities, those are no longer observed after chemotherapy? That is what they are looking for and hoping; right? 01:31:50 01:31:54 01:31:58 01:32:07 01:32:12 01:32:14

A Yes. 01:32:16

Q All right. So at what point in the process can 01:32:16

you say that the chemotherapy induced the chromosome 01:32:26

abnormality that is observed rather than eliminating it? 01:32:32

A Well, I think the point in the process will be 01:32:36

when the disease relapses, what it looks like. And if 01:32:40

it is different than the original presentation and now 01:32:45

there are new cytogenetic changes that you did not see 01:32:48

prior to the chemotherapy, you cannot rule out the 01:32:51

probability that the chemotherapy is what caused that 01:32:54

additional cytogenetic change. 01:33:01

Q How long does it take for those additional 01:33:02

cytogenetic changes to occur as a result of chemotherapy 01:33:04

where they are observed in patients? 01:33:09

A I'm probably not the best person to ask. I 01:33:11

mean I think these diseases can relapse as quickly as 01:33:15

three months. 01:33:19

Q Okay. Is there any significance to you of the 01:33:20

trisome 13 abnormality that Mr. Hazlehurst had? 01:33:36

A No. 01:33:40

Q It is a type of chromosomal abnormality that is 01:33:45

classified as aneuploidy; correct? 01:33:51

A Yes. 01:33:54

Q And benzene does cause aneuploidy, does it not? 01:33:54

A I would agree that under appropriate exposure 01:33:59

conditions, you will see changes in chromosome numbers, yes. 01:34:01 01:34:05

Q And the minus 19 is also a form of aneuploidy. It's the loss of a chromosome; correct? 01:34:07 01:34:10

A Yes. 01:34:14

Q And benzene also does that, does it not? 01:34:14

A I have never seen any evidence that benzene causes a monosome in 19 -- causes a loss of 19. 01:34:18 01:34:20

Q No, I am sorry. I meant more generally, benzene does -- has the ability to knock out chromosomes producing monosomes? 01:34:25 01:34:27 01:34:33

A Particularly with monosome 7, that seems to be true. 01:34:36 01:34:39

Q Okay. All right. So you next write that, "Cumulative exposures per Dr. Sheehan from SK 105 and gasoline was 0.6 PPM years"; correct? 01:34:39 01:34:52 01:34:57

A Right, yes. 01:35:01

Q And by that do you mean that the cumulative exposures that Mr. Hazlehurst had from Safety-Kleen solvent and gasoline in a Safety-Kleen parts washer operation was .06 PPM years? 01:35:02 01:35:04 01:35:11 01:35:16

A That seems right. I think that is what he said. 01:35:19 01:35:22

Q And isn't it true that 0.6 PPM years is sufficient to cause AML? 01:35:24 01:35:27

A That is not true. 01:35:31

Q Okay. Your first opinion for Mr. Hazlehurst is 01:35:39

that "Mr. Hazlehurst was not exposed to sufficient 01:35:54

quantities of benzene to meaningfully increase his risks 01:35:58

for forming AML." 01:36:02

Was he exposed to sufficient quantities of 01:36:04

benzene to increase his risk of developing AML at all? 01:36:06

A Depends on who you ask. 01:36:13

Q I am asking you. 01:36:15

A Well, I don't think he was because I don't 01:36:15

believe that the linear model is appropriate. 01:36:18

Q And I am gathering from your response that 01:36:22

there is a -- there are different views on that topic 01:36:24

held within the scientific community; is that correct? 01:36:28

A On the linearized multi-staged model, it is a 01:36:32

policy decision that EPA uses to model most carcinogens. 01:36:34

Q So you are telling me that the EPA has adopted 01:36:47

that, that model? 01:36:54

A The linearized multi-stage, yes. They invented 01:36:56

it. 01:37:00

Q I see. 01:37:01

Do other governmental agencies use that 01:37:06

approach? 01:37:10

A I know of some that do and some that don't. It 01:37:12

depends on the chemical, depends on the disease, depends 01:37:15

on the agency. You know, even when you use a linearized model, there are increased risks associated with that modeling effort below which you don't have to consider them. 01:37:18 01:37:23 01:37:30 01:37:34

Congress ruled on that in 1983 that anything below 10 to the minus 6 was considered de minimus. So even with a linearized model, there are increased risks; one times 10 to the minus 7, for example, that the EPA would consider insignificant. 01:37:35 01:37:38 01:37:43 01:37:47 01:37:49

Q What is the risk of developing AML from benzene exposure at a cumulative lifetime dose of 40 part per million years? 01:37:55 01:38:02 01:38:11

A I can't remember. I don't know. 01:38:23

Q Is it -- is the risk more than one times 10 to the minus 6? 01:38:25 01:38:29

A Yes. 01:38:31

Q More than one times -- more than one times 10 to the minus 5? 01:38:31 01:38:35

A I don't think so, but I am not sure. Now that is using this linearized model, which is purely a theoretical exercise. 01:38:37 01:38:38 01:38:43

A more appropriate comparison of that 40 part per million years is going to be what does the quantitative epidemiology tell us about people who had those exposures. 01:38:46 01:38:50 01:38:53 01:38:56

And there you will see that there is not a statistically significant increase in AML associated with those cumulative levels. 01:38:58 01:39:00 01:39:03

Q You are familiar with the OSHA benzene standard? 01:39:19 01:39:21

A Yes. 01:39:22

Q When that was adopted, it's true, is it not, that the standard was set, even though at the level of the standard, there would be an increased risk of leukemia from benzene using that model greater than one times 10 to the minus 5? 01:39:25 01:39:30 01:39:37 01:39:41 01:39:50

A I would have to review that document. 01:39:53

Q In fact, it was greater than one times 10 to the minus 4, wasn't it? 01:39:55 01:39:58

A I don't recall. I would have to review it. 01:40:01

Q Okay. Regarding your opinion No. 6, you write, "It is not possible to rule out the role that cigarette smoking played in the etiology of his disease." 01:40:10 01:40:32 01:40:36

You are referring to Mr. Hazlehurst; right? 01:40:42

A Yes. 01:40:43

Q Are you -- I understand that. 01:40:43

But are you going to tell the jury that it is scientifically probable that cigarette smoking did contribute to his AML? 01:40:44 01:40:49 01:40:54

A I don't know how to answer that. I wouldn't 01:41:01

say that because I don't know how anyone could make that determination. 01:41:02 01:41:05

What I will tell them is that the literature is pretty clear that a cigarette smoking history, as large as his or larger, does increase one's risk of developing AML. That is as far as I could take it. 01:41:05 01:41:08 01:41:12 01:41:17

Q Okay. Your last point here, No. 7, "The scientific literature on the mechanic profession does not indicate that this profession puts one at an increased risk of developing AML"; correct? 01:41:20 01:41:28 01:41:31 01:41:35

A Yes. 01:41:38

Q You have reviewed the studies -- certain studies on that topic; correct? 01:41:39 01:41:41

A Yes. 01:41:43

Q Did any of those studies report a statistically significant increased risk of AML in mechanics? 01:41:44 01:41:48

A I think there is probably one. 01:41:52

Q But you discount that study? 01:41:55

A That is not true at all. Just because there is one finding that finds an excess but then there is four or five findings that do not find an excess, you have to look at it collectively. 01:41:58 01:42:00 01:42:03 01:42:06

You have to weigh the entire literature collectively. You can't pick out one study and say, "This is it. This is the one." 01:42:07 01:42:09 01:42:12

Q Well, let's take a look at those studies. We will get to them in just a second. 01:42:14 01:42:23

Exhibit 8 is a list of literature that you have prepared regarding indirect benzene exposures and hematologic malignancy; is that correct? 01:42:26 01:42:30 01:42:36

A Well, yeah, a few studies that I have seen. Most of these are associating or talking about childhood leukemias -- 01:42:40 01:42:44 01:42:47

Q Okay. 01:42:47

A -- which has relatively little relevance here. 01:42:48

Q And Exhibit 9 is your literature list regarding gasoline in AML? 01:42:51 01:42:54

A Yes. 01:42:55

Q And now Exhibit 10 is your literature list regarding mechanics and AML; correct? 01:42:56 01:42:58

A Yes. 01:43:01

Q And these are the studies that you considered on that topic; correct? 01:43:04 01:43:07

A Yes. 01:43:09

Q And how many of the studies on this list showed a statistically significant increase of AML in mechanics? 01:43:13 01:43:18 01:43:24

A None. 01:43:39

Q How many of them showed a statistically significant increase in leukemia? 01:43:57 01:44:00

A One. 01:44:19

Q Which one? 01:44:20

A Hunting, but there was only one AML case. 01:44:20

Q That is the Hunting study? 01:44:26

A 1995, so that is pretty small. There were two leukemia cases, only one AML. It would be hard to put too much emphasis on that study. 01:44:28 01:44:32 01:44:36

Q What about the Pasqualetti study? 01:44:38

A In the Pasqualetti study, the mechanics exposed to gasoline did not have an elevated risk. And the mechanics or groups of workers -- I don't recall which ones they were -- exposed to aromatic hydrocarbons did have an increased risk. 01:44:43 01:44:47 01:44:51 01:44:53 01:44:57

Q How many of these studies have sufficient statistical power to detect a significant increase in the risk of AML? 01:45:01 01:45:03 01:45:07

A I don't -- I don't know the answer to that. You would have to go through and do a power calculation on every one of them. 01:45:17 01:45:18 01:45:22

Q You haven't done that; correct? 01:45:23

A No. 01:45:25

Q So without knowing that, the fact that some studies don't report a significant increase cannot be interpreted to mean that there isn't one; correct? 01:45:25 01:45:27 01:45:31

A No, I disagree with that. 01:45:34

Q Take a look at Exhibit 12, please. 01:45:53

A Okay. 01:45:58

Q What are these documents? 01:46:04

A I actually thought that it was something that -- that Jones Carr sent me from Dr. Gale, but it may have been from Dr. Brautbar, so I am not exactly sure what it is. 01:46:08 01:46:10 01:46:18 01:46:21

Q On the first page, have you made some handwritten notations? 01:46:23 01:46:27

A Uh-huh, yes. 01:46:28

Q Would you tell me what they are. 01:46:29

A Well, I originally thought that the values written down for the Rushton '97 paper were incorrect and I am still not sure they are not, but they are close. 01:46:31 01:46:33 01:46:37 01:46:39

And there is some -- it's written in there several different ways, but the problem with that is a little misleading just picking that one data point out. 01:46:40 01:46:43 01:46:47

Because if you look at the next exposure bin, which is written over on the left-hand column, greater than 0.4 part per million, then the AML risk is almost halved, 0.43. 01:46:50 01:46:53 01:46:57 01:47:01

So you can find these little quirky increases, but when you look collectively at the overall dose response, it just doesn't hold up. 01:47:03 01:47:07 01:47:11

And that's the same for the last two points down at the bottom where the 01 to -- sorry, 0.1 to 0.2 is listed, but the 0.2 to 0.4 is not listed. And then the 4.4 to .8 is, but the .8 to 1.6 is not. That's just inappropriate. 01:47:13 01:47:15 01:47:21 01:47:27 01:47:37

Q Okay. Exhibit 13 is an invoice that you submitted to the Jones Carr firm? 01:47:38 01:47:44

A Yes. 01:47:47

Q That was for -- 01:47:48

A Oh, this is how you knew. I am sorry, keep going. Keep going, I am sorry. 01:47:50 01:47:52

MS. KIMBALL: Sorry to interrupt, but, Raphael, I am having trouble hearing you. If you could just speak up a bit. Thank you. 01:47:58 01:47:59 01:48:03

MR. METZGER: Sure. I guarantee you I am not saying anything of great interest, but I will speak up a little bit. 01:48:04 01:48:08 01:48:10

MS. KIMBALL: Thank you. 01:48:11

Q BY MR. METZGER: This was prepared September; correct? 01:48:13 01:48:14

A That seems right, yes. 01:48:16

Q Have you done work on this case since that date? 01:48:18 01:48:20

A There was another invoice that I have submitted for $1,500 -- oh, this was 1,500. So there is another 01:48:20 01:48:23

one around for 1,300. 01:48:29

Q Okay. 01:48:31

A And then I have done work in preparation for 01:48:31

today that I haven't totaled up. 01:48:33

Q And what work did you do in preparation for 01:48:35

today? 01:48:37

A Look at all the literature, review all of the 01:48:37

medical records, get ready for coming to this 01:48:40

deposition, prepare the studies that I sent. 01:48:43

Q Did you meet with the lawyers? 01:48:45

A We met last night for dinner. 01:48:48

Q Okay. 01:48:50

A And yesterday afternoon I met with Chris Carr. 01:48:51

Q And you told us that you had a conversation 01:48:56

about an hour with Dr. Sheehan? 01:48:57

A That's what I was laughing about. That is how 01:48:59

you knew that, because I had written it on this piece of 01:49:01

paper. Yes, I talked to Dr. Sheehan about a week ago. 01:49:04

Q Have you spoken with any other experts about 01:49:07

this case? 01:49:09

A No. 01:49:09

Q So tell me now, Exhibit 14, regarding your 01:49:11

conversation with Dr. Sheehan, what did he tell you -- 01:49:15

what did you ask him, what did he tell you during that 01:49:17

conversation? 01:49:21

A The only -- the only thing that I really wanted to know was why he didn't think it was appropriate to take total -- an internal dose from milligrams from dermal and any other source and back-calculate part per million years from it, because I always thought that you could do that. 01:49:22 01:49:25 01:49:30 01:49:36 01:49:40 01:49:43

And so we chatted about that. He explained his methodology, what he did, and how he divided up, parsed out the individual exposures, which made sense to me and I could understand what he was doing. 01:49:45 01:49:47 01:49:54 01:49:58

But the only real question I had was this back-calculation, which is why he left it in milligrams instead of converting it into a part per million year basis. And you can't do that. 01:50:02 01:50:05 01:50:08 01:50:12

You can convert it in into a part per million year basis, which is what I thought, but you have to make assumptions about how much the breathing is going to be and all these sort of exposure assessments to get back to what it would equate to. And he didn't think that was appropriate because you don't have that information. 01:50:14 01:50:17 01:50:21 01:50:24 01:50:27 01:50:29 01:50:31

So the milligram -- expressing exposure to benzene on a total milligram basis just allowed for an easy comparison between the different sources of benzene for these individual plaintiffs. 01:50:32 01:50:35 01:50:40 01:50:44

Q Okay. 01:50:47

A That is pretty much what I got out of that 01:50:48

conversation other than a few pleasantries since I 01:50:51

haven't talked to him in a while. 01:50:54

Q Do you know him? 01:50:56

A Uh-huh, yes. 01:50:57

Q How do you know him? 01:50:58

A Dr. Sheehan worked for Exponent when I was at 01:50:59

Exponent in 2004. He was out here in Oakland and I was 01:51:02

in Boulder and we didn't work on any projects, but there 01:51:07

was a company retreat that I met him at and we were on 01:51:12

several conference calls and that kind of thing. 01:51:15

Q Okay. While we are talking about Dr. Sheehan's 01:51:18

assessment, was there anything in his assessment that 01:51:27

you disagreed with? 01:51:31

A No. 01:51:32

Q You thought his methodology was fine? 01:51:34

A Well, to the extent that I understand all that, 01:51:36

I thought it was okay. Same with Dr. Nicas. I 01:51:39

didn't -- you know, I can't really say that I understood 01:51:42

his nearly as well. It wasn't -- to me it wasn't 01:51:44

written as clearly as Pat Sheehan's was. 01:51:51

Q Do you have any disagreements with Dr. Nicas' 01:51:53

methodology or his calculations or his exposure 01:51:57

assessments of the individuals? 01:52:01

A Well, I think there is some discussion about the appropriateness of using the Fedoruk study and whether there is better data out there. 01:52:02 01:52:04 01:52:08

But, you know, to my way thinking, in my point of view was that even if you accept Dr. Nicas as being absolutely correct, that is still insufficient benzene exposure to relate -- to cause these people's disease. 01:52:10 01:52:14 01:52:19 01:52:23

Q I understand. Please answer my precise questions though. 01:52:26 01:52:30

Do you criticize in any way Dr. Nicas' methodology? 01:52:31 01:52:35

A That is outside of my area, so no. 01:52:36

Q Do you criticize in any way Dr. Nicas' exposure assessment? 01:52:38 01:52:41

A Not at this point. It is outside of my area, no. 01:52:43 01:52:45

Q Is the evaluation of Dr. Sheehan's methodology also outside of your area? 01:52:46 01:52:49

A Yes. 01:52:51

Q Is the evaluation of Dr. Sheehan's dose assessment also outside of your area of expertise? 01:52:52 01:52:59

A I would say in this format, yes. 01:53:00

Q In this format? 01:53:03

A Well, I am a toxicologist. I can do dose calculations myself. But in a formal setting like this, 01:53:04 01:53:07

he is the expert. That is his area. He would be the one to defend it. 01:53:09 01:53:13

Q Okay. Exhibit 15 is something that counsel provided you? 01:53:14 01:53:20

A Sorry, I have -- oh, here it is. 01:53:22

Yes, yes. 01:53:26

Q Are there any other notes or writings that you prepared regarding this case? 01:53:37 01:53:40

A No, except for the possibility of Mr. Machado, which I will -- 01:53:43 01:53:46

Q Right. 01:53:49

A -- because I did the two, so I am pretty sure -- well, in fact I copied one opinion and then I would have changed it, so clearly I had something written for Machado so I will find that and make sure you get it. 01:53:49 01:53:51 01:53:54 01:53:56 01:54:02

Q Okay. And have you told me all of your opinions in this case -- in these cases? 01:54:03 01:54:12

A Yes, particularly since we went over those little summary sheets. 01:54:15 01:54:19

Q There is one thing I want to ask you. Yeah -- well, we haven't done that for Mr. Machado. 01:54:27 01:54:31

Are there any other opinions that you recall that you wrote regarding Mr. Machado that you have not yet told me today? 01:54:34 01:54:37 01:54:41

A No, no. 01:54:42

Q Okay. Okay. Subject to the receiving the 01:54:43

Machado summary that you prepared and any questions I 01:56:05

might have regarding that, I have concluded my 01:56:08

examination. 01:56:14

Any questions by other counsel? 01:56:14

MR. FOLEY: Let's go off the record for one 01:56:16

minute. 01:56:18

MR. METZGER: Sure. 01:56:18

THE VIDEOGRAPHER: Now going off the record. 01:56:19

Time is 1:56 p.m. 01:56:19

(A discussion was held off the record.) 01:56:21

THE VIDEOGRAPHER: We are now back on the 01:58:03

record. Time is 1:57 p.m. 01:58:04

(Deposition Exhibit 16 was marked for 01:58:06

identification.) 01:58:06

MR. METZGER: All right. Dr. Pyatt has found 01:58:07

another invoice, which has been marked as Exhibit 16. 01:58:09

Q It is dated 7-28-08; correct, doctor? 01:58:12

A Yes. That was the 1,300 one. 01:58:16

Q Was that the first invoice that you prepared 01:58:18

for this case? 01:58:19

A Yes. 01:58:20

MR. METZGER: Okay. So I think we are done for 01:58:21

the day. 01:58:32

And I propose that the court reporter may forward the original transcript to the Jones Carr firm, who will then forward it on to Dr. Pyatt; Dr. Pyatt, you can have 30 days from the Jones Carr's firm receipt of it to sign the transcript; you may sign it under penalty of perjury so you don't have to round up a notary; you can make any changes or corrections that you wish to the testimony; if you do that, I would ask that you make those on the pages where the testimony actually occurs and also to list them on a correction sheet which the court reporter will provide you at the end of the transcript so that we may know where those changes are. 01:58:34 01:58:36 01:58:40 01:58:44 01:58:49 01:58:53 01:58:56 01:58:59 01:59:03 01:59:06 01:59:09 01:59:11

THE WITNESS: That is fine. 01:59:15

MR. METZGER: If you would then forward that on to the Jones Carr firm, they will notify all counsel of the changes forthwith, and then forward the original on to me; and I will preserve it, lodge it with the court in advance of trial or hearing on reasonable request; if for any reason the original is lost or not signed, a certified copy may be used with full force and effect. 01:59:16 01:59:18 01:59:21 01:59:24 01:59:29 01:59:32 01:59:35

So stipulated? 01:59:39

MR. FOLEY: Yes. 01:59:41

MR. METZGER: Okay. 01:59:42

THE VIDEOGRAPHER: Now going off the record. Time is 1:59 p.m. This is the end of Volume 1, tape No. 01:59:42 01:59:44

4 and concludes today's deposition. 01:59:48

01:59:51

(ENDING TIME: 2:00 P.M.) 01:59:54

STATE OF CALIFORNIA)
) ss
COUNTY OF LOS ANGELES)

I, the undersigned, declare under penalty of perjury that I have read the foregoing transcript, and I have made any corrections, additions or deletions that was desirous of making; that the foregoing is a true and correct transcript of my testimony contained therein.

EXECUTED this _____ day of _____,

20___, at _____, _____
(City) (State)

_____
DAVID W. PYATT, Ph.D.

REPORTER'S CERTIFICATION

AUTHENTIC COPY
The original certified E-Transcript
filewas electronically signed
using Reallegaltechnology.

I, SHERI A. PLY, CSR No. 6507, a Certified Shorthand Reporter in and for the State of California do hereby certify:

That the foregoing proceedings were taken before me at the time and place therein set forth, at which time the witness was placed under oath by me;

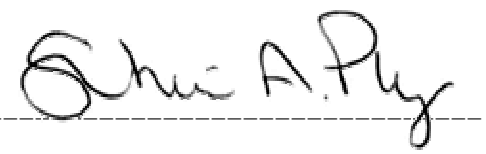
That the testimony of the witness and all objections made at the time of the examination were recorded stenographically by me and were thereafter transcribed;

That the foregoing transcript is a true and correct record of the testimony so taken.

I further certify that I am not a relative or employee of any attorney or of any of the parties, nor financially interested in the action.

I declare under the penalty of perjury under the laws of the State of California that the foregoing is true and correct.

Dated this 19th day of December, 2008.



SHERI A. PLY, CSR No. 6507