CAUSE NO. 199-02538-04		
DUANE DRAPER, ET AL.,	)	IN THE DISTRICT COURT OF
	)	
PLAINTIFFS,	)	
	)	
VS.	)	COLLIN COUNTY, TEXAS
	)	
PPG INDUSTRIES, INC., ET AL,	)	
	)	
DEFENDANTS.	)	199TH JUDICIAL DISTRICT

ORAL DEPOSITION OF

ETHAN A. NATELSON, M.D.

DECEMBER 10, 2007

VOLUME 1

ORAL DEPOSITION OF ETHAN A. NATELSON, M.D., produced as a witness at the instance of the Plaintiff(s), and duly sworn, was taken in the above-styled and numbered cause on the 10th day of December, 2007, from 1:37 p.m. to 4:35 p.m., via telephone, before Kimberly J. Carter, CSR in and for the State of Texas, reported by machine shorthand method at the offices of Schirrmeister Diaz-Arrastia Brem, LLP, 700 Milam, 10th Floor, Houston, Texas 77002, pursuant to the Texas Rules of Civil Procedure and the provisions stated on the record or attached hereto.

A P P E A R A N C E S

FOR THE PLAINTIFF:
Mr. Andrew J. DuPont (via telephone)
LOCKS LAW FIRM
The Curtis Center
601 Walnut Street, Suite 720 East
Philadelphia, Pennsylvania 19106
(215) 893-0100
adupont@lockslawpa.com

FOR THE DEFENDANT E.I. DUPONT DE NEMOURS AND COMPANY:
Mr. Andrew C. Schirrmeister, III
SCHIRRMEISTER DIAZ-ARRASTIA BREM, LLP
700 Milam, 10th Floor
Houston, Texas 77002
(713) 221-2500
acs@sdablaw.com

FOR THE DEFENDANT PPG INDUSTRIES, INC. AND THE SHERWIN WILLIAMS COMPANY:
Mr. Christopher Stofko (via telephone)
DICKIE, MCCAMEY & CHILCOTE, P.C.
Two PPG Place, Suite 400
Pittsburgh, Pennsylvania 15222-5404
(412) 281-7272
CStofko@dmclaw.com

FOR THE DEFENDANT ILLINOIS TOOL WORKS, INC.:
Mr. Bryan D. Pollard
WILSON, ELSER, MOSKOWITZ, EDELMAN & DICKER, LLP
5000 Renaissance Tower
1201 Elm Street
Dallas, Texas 75270-2161
(214) 698-8000
bryan.pollard@wilsonelser.com

FOR THE DEFENDANT BASF:
Mr. Joseph C. Orlet
HUSCH & EPPENBERGER, LLC
190 Carndelet Plaza, Suite 600
St. Louis, Missouri 63105-3441
(314) 480-1500
joseph.orlet@husch.com

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ETHAN A. NATELSON, M.D.,

having been first duly sworn, testified as follows:

EXAMINATION

BY MR. DUPONT:

Q. Good afternoon, Dr. Natelson.

A. Good afternoon.

Q. My name is Andrew DuPont. As you know, I'm an attorney for the plaintiffs in this case. And we're doing your deposition here today by telephone, which can sometimes be a little awkward, but if at any point you're not hearing me clearly or I'm not hearing you clearly, we'll kind of both have to speak up. Is that okay?

A. That's fine. I can hear you very clearly, so it's a good connection.

Q. Excellent. I'm hearing you well as well.

So you've given your deposition before, so I assume you are familiar with the kind of ground rules that we like to lay?

A. Yes.

Q. I'll go over them anyhow just to make sure we are on the same page.

If at any point I ask you a question that you do not understand, please feel free to let me know, and I will repeat or rephrase the question to the point

that you do understand it. Is that okay?

A. Yes.

Q. Excellent. Particularly, when we're doing this deposition by telephone, if you have a response to give, it needs to be verbal. A nonverbal response, such as a nod or a shake of the head, can't be taken down by the court reporter and certainly can't be seen by me. Similarly, an "uh-huh" or a "huh-uh" is difficult for the court reporter to take down in a manner that we can easily go back and see on the record, so if you can use a "yes" or a "no," if that would be appropriate, I would appreciate that as well.

If you do respond to a question, I will assume that you responded based on the way the question was phrased and that you understood the question, and the record will reflect that. Do we have an understanding on that?

A. Yes.

Q. Great. Thank you.

Doctor, could you give us your full name, please?

A. Ethan A. Natelson.

Q. Dr. Natelson, you are a hematologist oncologist?

A. A hematologist. I have done some oncology,

but my certification is in hematology.

Q. And where are you currently practicing?

A. At the Methodist Hospital in Houston, Texas.

Q. What positions do you hold at that hospital?

A. Well, I'm the Director of the Transitional Internship Program or now the Transitional Residency Program at Methodist Hospital, and I'm a member of the Directors of the Internal Medicine Residency at the Methodist Hospital. And I have an appointment through Cornell Medical School, which has an arrangement with Methodist Hospital, as an associate professor of medicine.

Q. You're currently involved in your practice in treating individuals with AML?

A. Yes.

Q. And in the context of your practice when you diagnose a patient, you use something called a differential diagnosis?

A. Yes.

Q. What factors do you consider in your differential diagnosis when determining the etiology of an acute myelogenous leukemia?

A. Well, I look at the history of the patient: what the family history might be, what the occupation of the person might be, whether there's been any exposure

to radiation or other chemicals, whether there's underlying genetic defects or associated disorders, the type of acute leukemia that it is, whether there's been a preexisting myelodysplastic phase to the leukemia or whether it has suddenly occurred, what types of chromosome abnormalities are involved in the leukemia or if there are any abnormalities in the chromosomes.

Those would be the predominant things I look at. Also history of smoking. I think I mentioned the family history. If there is an allegation of chemical exposure, the question is when was that exposure relative to the onset of the leukemia and what type of exposure it was and what levels of exposure.

Q. When you're looking at the family history, you're looking for a family history of leukemia?

A. Well, in most instances there is no family history of leukemia in a person with acute myeloid leukemia. There might be with chronic lymphocytic leukemia, which frequently has a familial basis. In AML, it only occasionally does.

There are other associations. There are certain congenital chromosome abnormalities that favor the diagnosis of AML, such as Down syndrome. There are certain acquired abnormalities, such as pernicious anemia, which is a type of anemia that can cause

chromosome abnormalities. Those are some of the things we might look for.

Q. With respect to occupations, which occupations are you looking for when you want to know if a person's occupational history could be causative or a causative factor in their development of AML?

A. Well, we know that certain forms of radiation can cause acute myeloid leukemia, so we're interested to know if this person was associated in any occupation where he might have had radiation exposure. If we're looking in terms of other occupations, and with respect to the allegations in this case, whether a person might have had exposure to some type of industrial chemical, such as benzene, that might be a factor in the leukemia.

Q. So benzene exposure is one occupational history factor you're looking at in your differential diagnosis, correct?

A. Yes.

Q. And would you agree with me that mechanics are one profession or job type that is known to have a history or an expectancy of benzene exposure?

A. Well, to a certain degree because certain solvents may have trace amounts of benzene in them.

Q. And you would agree with me that painters are a job title that are expected or known to have an

exposure to benzene?

MR. SCHIRRMEISTER: Object to the form.

A. I suppose it's the type of paints they're using, but in solvents, oil-based paints, there would be the potential for trace amounts of benzene in certain of the solvents.

Q. (By Mr. DuPont) Similarly, auto body repairmen working with solvents would be another job title or category which you would want to consider in the occupational history of someone that's presented to you with AML?

A. Yes.

Q. And that is because that is another occupation that is known to have benzene exposures?

MR. STOFKO: Objection to form. This is Chris Stofko.

MR. SCHIRRMEISTER: Object to the form.

MR. STOFKO: Andrew, can we have an agreement, any objection by one defense counsel is good for everyone?

MR. DUPONT: That's fine, Chris.

MR. STOFKO: Thanks. I appreciate it. That way, I won't interrupt as much.

Q. (By Mr. DuPont) Doctor, do you recall the question?

A. Perhaps you could repeat it.
Q. Sure.
And automobile body repairmen are similarly a job title or category which are known to have exposures to benzene?
A. Well, they might.
MR. SCHIRRMEISTER: Object to the form.
A. I mean, a body repairman might be anything from someone who takes a bumper off and puts a new one on or pounds out a dent. But if they're working with solvents, they might have a potential for benzene exposure.
Q. (By Mr. DuPont) When a patient presents to you with a newly diagnosed AML and you're conducting your differential diagnosis, do you ask that patient for specific job types, or do you ask them for just a broad characterization of the work they do and what they work with?
A. I typically ask them what types of occupations they've been involved in, do they have any unusual hobbies or work with any particular chemicals, what sort of drug exposures they might have had in the past, things of that nature.
Q. And what hobbies are you looking at?
A. Again, hobbies that someone might have

exposure to heavy metals, such as arsenic, or benzene, for example, in the form of solvents or glues, things of that nature.

Q. Would you agree with me that family history can be ruled out as a causative factor in a differential diagnosis for Mr. Duane Draper?

A. We have no family history of acute leukemia in his particular instance.

Q. Okay. So would the answer to that be yes, you'd agree with me that family history can be ruled out as a causative --

A. Well, we have no evidence for that. We have no evidence for that.

Q. I'm sorry, Doctor. One instruction I didn't give you is that it's difficult for the court reporter to do her job if we are speaking over each other. And it's also more difficult when we're on the phone and you can't see me kind of physically stopping my question, and I can't see you stopping your response. So if we can try not to talk over each other, I would appreciate that.

But my question to you is: There is nothing with respect to Mr. Draper's family history which would indicate that that is a causative factor in your differential diagnosis; is that correct?

A. That is correct.
Q. And there is nothing in Mr. Draper's medical or social history that would indicate to you that radiation is a causal factor in the differential diagnosis for his AML?
A. Yes. That is correct.
Q. Similarly, there's nothing in his occupational history that would indicate to you that radiation is a causal factor that must be considered in his particular differential diagnosis for AML?
A. No. We have no evidence of radiation exposure.
Q. You mentioned genetics as being another factor that you would look at?
A. Yes.
Q. What, specifically, are you looking at with respect to genetics?
A. Well, there are some articles that suggest that children of women with breast cancer, for example, have a higher risk of acute myeloid leukemia. We know that the genetics that have to do with Down syndrome, for example, predispose to acute leukemia. Certainly, we have no evidence he had Down syndrome. Those would be the things I would be looking at.
Q. And we also have no evidence that Mr. Draper's

mother had breast cancer?

A. No.

Q. So we can, similarly, rule out genetics as a causative factor in his AML?

A. Well, I don't know that one can ever rule that out. We don't know what causes most patients with AML, and so all we can say is he has no family history of it.

Q. There's no evidence in this case to suggest that Mr. Draper's genetics or genetic history is a causative factor in his development of AML?

A. That is correct.

Q. You mentioned that the type of AML would be a factor to consider in your differential diagnosis?

A. Yes.

Q. What do you mean by that? What types are you considering?

A. Well, predominantly the chromosome array that we see in the acute leukemia.

Q. Are there any chromosome arrays which you can definitively say are not associated with exposure to benzene?

A. Well, we don't have many patients with benzene-induced leukemia that have been carefully studied by chromosome analysis. The literature suggests that there is a frequency of abnormalities in the 5th

and 7th chromosome similar to what we see at chemotherapy. There, presumably, are other abnormalities that are possible from benzene exposure. But I cannot say with certainty what all of the abnormalities that might occur from benzene are.

Q. Likewise, you can't say what chromosome abnormalities are not related to benzene exposure?

A. Not with certainty. We, of course, can look at the medical literature to see if they're present or have been described. If they haven't been described, it means that's unusual, but it might not mean they couldn't occur.

Q. Is there any medical literature that you have seen that you can cite for me today that would indicate that the type of chromosome damage Mr. Draper has is not related to benzene exposure?

MR. SCHIRRMEISTER: Object to form.

A. Well, Martyn Smith and Zang published an article in which they reviewed the world's literature on chromosome abnormalities and benzene or possibly induced by benzene, and the only mention of inversion 16 in the entire article is a sentence that says it occurs in de novo AML.

MR. DUPONT: Respectfully, I move to strike because I don't think that answers my question.

Q. (By Mr. DuPont) My question is: Can you cite for me a specific study that affirmatively says the type of chromosome damage that Mr. Draper suffers from is not caused by benzene exposure?

MR. SCHIRRMEISTER: Object to the form.

A. No.

Q. (By Mr. DuPont) You mentioned AML arising and then myelodysplastic phase or preceding MDS. What types of MDS would you consider arising prior to diagnosis of AML as being a causative factor in development of AML being related to benzene exposure?

A. Well, the classical type of MDS that we see preceding benzene exposure would be what's referred to as a hypoplastic type of MDS where the bone marrow has undergone some suppression and has abnormalities in cell development. That would be the type that's been seen after benzene poisoning and would be the most -- the most suggestive of a benzene effect. Certain myelodysplastic syndromes, such as sideroblastic anemias, for example, have never been described from benzene exposure.

There are other syndromes. The classification of myelodysplasia is very cumbersome, complex, and has undergone many revisions since it was proposed around 1982. And an illness called chronic

myelomonocytic leukemia was originally grouped as a myelodysplastic syndrome. It now is not. It also has not been related to benzene exposure.

Q. Smoking was another factor you mentioned. What is the shortest smoking history in terms of pack per year or pack year history which you would relate to being a causative factor in the development of AML?

A. Well, I don't know the answer to that in terms of what would be the shortest. The attorney general's report tells us that a 20-pack-year history doubles the risk, so I would accept a 20-pack-year history as potentially conducive to AML. It could be less, but I don't know that.

Q. In terms of what the literature establishes, though, it's your position that you have to have at least a 20-pack-year history for a doubling of the risk of AML associated with cigarette smoking?

A. I think that would be true, yes.

Q. And anything less than 20 packs per year smoking history or a 20-pack-year smoking history, you would not consider a causative factor in development of AML?

MR. SCHIRRMEISTER: Object to the form.

A. I couldn't be certain about that. In other words, I use that reference point because that is a

reference point that has been pointed out in the literature. It could be less, but I don't have any evidence of that.

Q. (By Mr. DuPont) You cannot say affirmatively that any history of smoking less than 20 pack years is causative in a diagnosis of AML?

MR. SCHIRRMEISTER: Object to the form.

A. That is correct.

Q. (By Mr. DuPont) What level of smoking history must one have to reach a tripling of the risk for AML?

A. I don't know that, and I don't know if -- if that is known with accuracy.

Q. Doctor, you've testified before that you need to see a tripling of the risk in order to make an association between exposure to a toxin and development of AML?

A. I don't think that's correct. I'm not sure which deposition you're referring to, but what I, typically, have said is that -- in the past is that what you're looking for is consistency, and an association of less than tripling might be indicative of an association if you have consistency among the number of studies. If you only had a single study to go by, then I would say that you need, certainly, more than a doubling of the risk.

Q. What are all the studies that you consider with respect to smoking being causative of AML?

MR. SCHIRRMEISTER: Object to the form.

A. As we sit here, I can't tell you all of the studies. One of the articles I referenced was a review of that by a prominent hematologist in 2007, and he has lots of references there. I referenced a few others in my report. I'm sure there are many papers that attest to that.

Q. (By Mr. DuPont) If a -- strike that.

If a patient came to you with newly diagnosed AML and there was no chromosome abnormalities found, would you say to that patient that their AML could not have been caused by benzene exposure?

A. No.

Q. If a patient came to you with a newly diagnosed AML and their chromosome damage presented as an inversion of the 16th chromosome, would you say to that patient that their AML could not have been caused by exposure to benzene?

MR. SCHIRRMEISTER: Form.

A. I would have no basis for saying that it could be. In other words, there's no precedent for that in the literature, so I would say, as I normally say in any case of leukemia, we cannot, with certainty, say the

etiology. But it would be highly unlikely to be due to benzene.

Q. (By Mr. DuPont) I just want to get an answer to my question.

If a patient came to you with a newly diagnosed AML and an inversion 16 were found that's the chromosome damage, would you say to that patient that their AML was not caused by benzene exposure?

MR. SCHIRRMEISTER: Object to the form.

A. As I've just said, I cannot say with certainty what causes AML, so I cannot say with certainty what doesn't cause AML.

Q. (By Mr. DuPont) But nonetheless, you couldn't tell that person that the presence of an inversion 16 meant that their AML was not caused by benzene exposure, could you?

MR. SCHIRRMEISTER: Object to the form.

A. That is correct.

Q. (By Mr. DuPont) You'll agree with me that benzene is a confirmed human carcinogen?

A. Yes.

Q. And when did benzene first become a confirmed human carcinogen?

A. I don't know the date of that confirmation.

Q. You'd agree with me that the routes of

exposure to benzene include inhalation?

A. Yes.

Q. They include dermal absorption?

A. Yes.

Q. And they include ingestion?

A. Yes.

Q. If you were considering the manner in which an individual was exposed to a solvent containing benzene, would you eliminate dermal exposure from your analysis knowing that benzene can be dermally absorbed through the skin of a worker?

MR. SCHIRRMEISTER: Object to form.

A. No.

Q. (By Mr. DuPont) To do so would be eliminating one-third of the risk of the routes to exposure, correct?

MR. SCHIRRMEISTER: Object to the form.

A. Of those three that you mentioned, yes.

Q. (By Mr. DuPont) And to do so would be to underestimate the individual's exposure. Wouldn't that be correct?

MR. SCHIRRMEISTER: Object to the form.

A. It would be correct, keeping in mind that I'm not a toxicologist, so I can't comment about the various percentage of exposure that one gets from these various

routes of exposure.

Q. (By Mr. DuPont) Doctor, what is latency?

A. Latency, with respect to leukemia, is when you -- it's the period of time between when you have reached a dose of the chemical that is leukemogenic and the time that you actually develop the leukemia.

Q. You predicted my next question, which is: At what point in time for an individual who's occupationally exposed to benzene do you begin the latency clock running? In other words, at what point in time in that individual's work history do you start calculating the latency period?

A. Well, again, it would be when they've had a dose, a cumulative dose, that is considered sufficient to cause AML.

Q. Doctor, do you have Mr. Draper's medical records with you?

A. Yes.

Q. Can you tell me when is the most recent medical records that you've received for Mr. Draper? What are the dates of those records?

A. If you'll hang on a second, let me see if I can find it.

Q. Please take your time.

A. I believe they would be from this disk I have

here labeled 12/14/05.

Q. So would it be true that the most recent records you received are from 12/14/05; otherwise, December 14, 2005?

A. Let me just see what I said in my letter if I commented about that. Well, no. Somewhere in here I have a blood count from August the 7th, 2007 -- I mentioned that in my letter -- that showed his blood count to be normal. So in this pile of records somewhere are at least blood counts from 2007.

Q. Have you reviewed the medical records up through December 14, 2005?

A. Yes.

Q. Would you agree with me that the treatment provided to Mr. Draper as evidenced by those medical records is reasonable and necessary for the treatment of his AML?

A. Yes.

Q. Have you received bills relating to his medical treatment?

A. Actually, I don't see any of that in this material I have here. They may be on this disk, but I didn't print them out.

Q. Do you recall reviewing medical bills relating to Mr. Draper's medical treatment?

A. I'm sure I did not.
Q. Did you report the -- review the report of Ginny Stegent, an expert on behalf of the plaintiffs in this case?
A. If it's in my materials here, I have. I don't recall it, as we are speaking.
Q. Doctor, I have several invoices in my copy of your file, one dated October 10, 2007, and another dated May 23rd, 2007. Do you have those in front of you?
A. Let's see. Yes. I have my invoices.
Q. And how many invoices do you have in front of you?
A. Four.
Q. Four of them.
What are the dates on those invoices?
A. Let's see. December 26, '05; April 23rd, '07; October 10, '07; November 6, '07. These may be rebilling some of these, but those are the dates on the invoices. But the actual charges are four.
Q. What are the charges reflected on the December 26th, 2005, invoice?
A. $937.50.
Q. And what do those charges relate to?
A. Review of some records.
Q. Medical records?

A. Yes.
Q. Any other records?
A. No.
Q. The April 23, 2007, invoice, what are those charges for, and how much are those charges?
A. Those are $625. They're records of Mr. Draper's deposition testimony, some medical records, some non-medical records, some expert reports of Arthur Frank, Burton Davidson, Edward Agura, Martyn Smith, Mark Nicas.
Q. I'm actually looking at a May 23, 2007, invoice for the same balance, which appears to be -- involves similar materials, and it says a second invoice?
A. It could be that that's just a rebilling.
Q. Okay. I understand.
A. And, by the way, while we're talking and you had asked me about medical records, I have a letter from the treating physician on January 30th, 2007, on Mr. Draper.
Q. Okay. How does that letter describe Mr. Draper's course of treatment?
A. I'm not sure what, specifically, you're asking.
Q. Well, does he describe it as a complicated

course of treatment? Was he responding well to treatment?

A. Well, the letter isn't long. I can go through it. He indicates he had his leukemia discovered in the fall of 2002. He had initial treatment that was not complicated for acute leukemia, and he promptly went into remission. And then he remained in remission for a time, but in January of 2004, he relapsed. And he received additional treatment, which did not get him into remission but then additional treatment subsequently that did get him in remission.

And then because he was having trouble staying in remission, he had an allogeneic marrow transplant, which he indicates was well tolerated, and that he's been in complete remission since that time.

Q. And you would agree with me that "complete remission" does not mean that an individual is cured from the leukemia?

A. That is correct.

Q. There is an October 10, 2007, invoice. How much is that for?

A. $1,625.00.

Q. And then November 6, 2007, deposition transcripts?

A. Yes. That's --

Q. What's that for?
A. $750.
Q. And you're currently billing at $400 per hour for deposition and trial testimony?
A. Yes.
Q. And $250 an hour for all other testimony?
A. Yes.
Q. Excuse me. All other services?
A. Yes.
Q. You mentioned in conjunction, I believe, with your April 23rd, 2007, invoice that included review of the deposition transcript from Mr. Draper. Is that the March 21, 2006, deposition transcript?
A. Well, let me look and see what I have here.
That's March 21, 2006.
Q. Is that the only transcript of Mr. Draper's deposition that you have reviewed?
A. Yes.
Q. What services were provided by you in conjunction with the November 6, 2007, invoice? In other words, what were you billing for that?
A. That had to do with an office conference with Mr. Schirrmeister, review of the report of Mr. Russell and of Mr. Keller.
Q. Was that the first time that you had the

opportunity to review either Mr. Russell or Mr. Keller's report in this case during that conference that's reflected in your November 6, 2007, invoice?

A. I don't know. I don't remember when they came, but, obviously, they came before this bill was sent.

Q. Did you review either Mr. Russell or Mr. Keller's reports prior to preparing your own report in this case?

A. I don't know the answer to that without some detailed study of these records. The date of the report is October the 10th, and so if I had something prior to October the 10th, I would have looked at it before I wrote that report.

MR. DUPONT: Before I get too far afield, let's mark the invoices as Exhibit A.

(Exhibit A marked.)

Q. (By Mr. DuPont) Doctor, let me ask you: Did you make any notations on Mr. Draper's deposition transcript?

A. No.

Q. Okay.

MR. DUPONT: Let's mark the transcript as Exhibit B, and the -- I think it's January 30, 2006, or 2007 letter of Dr. Edward Agura as C.

(Exhibits B and C marked.)

MR. SCHIRRMEISTER: What do you want to mark as B?

MR. DUPONT: Mr. Draper's deposition transcript.

Q. (By Mr. DuPont) Doctor, have you made any notes concerning this case?

A. No.

Q. Prior to your final report which you issued, did you have any drafts of your report?

A. I probably did, but the way this operates is that I might get the medical records first, before any reports or other information, depositions and so on, so what I normally do is I type out a summary of what the case is, the medical facts of the case. And then as the -- all of the materials are assembled, then I modify that and add other features and add references and finish the report.

Q. Do you have your initial summary or any of the subsequent drafts of your summary that you made prior to executing your final report?

A. No. I normally don't keep any hard copies, and I revise the letter on my computer so all that ends up being there is the final report.

Q. Prior to executing your final report, did you

show a draft of your report to any of the attorneys for the defendants in this case?

A. No.

Q. Prior to executing your final report, did you show a draft of your report to any of the other experts that have issued reports on behalf of the defendants in this case?

A. No.

Q. Did you review the expert reports of any of the other, I'll call, either doctors or toxicologists testifying or providing reports on behalf of the defendants in this case?

A. I reviewed what reports I have here, which are mentioned in those bills. Let's see. I reviewed a report written to Mr. Schirrmeister from Brad Russell.

Q. When did you receive Mr. Russell's report?

A. The draft -- the letter is entitled "October 8, 2007," and I received it, according to this e-mail I have, 10/10/07.

Q. And who is that e-mail from?

A. Let's see. It's from Andrew Schirrmeister to me, carbon copy to Mr. Stofko.

Q. And you understand that that is a draft of Mr. Russell's report?

A. No. I don't know that. I just have the

report. Oh. It does -- excuse me. I'm sorry. It does say -- on the e-mail, it says "draft report."

MR. DUPONT: Let's go ahead and mark that report, along with the e-mail, as Exhibit D, if you don't mind.

(Exhibit D marked.)

Q. (By Mr. DuPont) Did you see the drafts of any of the other expert reports for -- that were submitted on behalf of the defendants? In other words, did you see drafts of any of those reports before the final copy was executed?

A. No.

Q. Did you speak with any of the other doctors or toxicologists that issued reports on behalf of the defendants prior to issuing your report?

A. No.

Q. Were you asked by any of the attorneys for the defendants in this case to address specific issues in your report or cite any specific literature in support of your report?

A. No.

Q. Were you provided with any literary references from any of the attorneys on behalf of the defendants in this case?

A. No.

Q. Were you provided with any notes or memorandums that provided kind of a template or an outline of what your report should look like from any of the attorneys for the defendants in this case?

A. No.

Q. Doctor, I have a document in front of me that's entitled, "Dr. Ethan Natelson's File Inventory, December 3, 2007," with the caption "Duane Draper versus PPG Industries, Inc., et al." on top. Do you have that as -- is that part of your file?

A. Not that I know of. I'm not sure what that is that you're referring to.

Q. Okay. And I'm not suggesting that it is. I'm just trying to -- I'm trying to think of an easy way to do this.

Okay. Let's do this. Do you have your December -- excuse me -- your October 10, 2007, report in front of you?

A. Yes.

Q. The back of your report, there's a number of literary references?

A. Yes.

Q. And those are the references that you relied upon in drawing the opinions and conclusions in your report?

A. Yes.

Q. Part of your opinions concern -- and I'm going to butcher this, and then we'll abbreviate it -- "Topoisomerase II Inhibitors and Their Effect on Chromosome Damage in the Lymphocytes and Myeloid Cells." Generally, that's accurate? That's a topic that you discuss?

A. Yes.

Q. And one of the conclusions you reach is that -- we'll call it topo II -- topo II inhibitors, the chromosome damage that they cause in lymphocytes is not relevant to chromosome damage in myeloid cells. Is that accurate?

A. Let me just see what I wrote there. What I said is that most chemotherapeutic agents cause chromosomal abnormalities in the lymphocytes of patients receiving these drugs whether the drugs are known leukemogens or not. Such abnormalities are not sustained and are rapidly repaired and then return to normal.

And I go on to mention that one of the drugs we used for many, many years in chemotherapy is methotrexate, which produces striking abnormalities in chromosomes and lymphocytes, both in the test tube and in people, and yet is not known as a leukemogen.

And so that and many other articles call into question the notion that simply demonstrating abnormalities in a lymphocyte has much to do with acute myeloid leukemia in an entirely different cell line. And, specifically, with reference to inversion 16, I also provided Mr. Schirrmeister with papers that show that the -- given patients who have inversion 16 leukemia, their lymphocytes don't bear that particular abnormality.

Q. Okay. Why don't we start with that last portion of what you just said. Can you point to me on your literary references at the end of your October 10 report those papers you rely upon with respect to your analysis of the lymphocytes and inversion 16?

A. Well, let's go back to that paragraph. Well, let's see. It started out with references 48, 49, 50 through 51, and then subsequent to this report I gave some additional references to Mr. Schirrmeister concerning inversion 16 and a few of the other balance translocations which demonstrate that they don't carry chromosome abnormalities in the lymphocytes. And you probably should have copies of those articles because they were to be added to my file.

MR. SCHIRRMEISTER: Those are the ones that we read into the record before the deposition

began, so I'm not sure if the court reporter took those down.

MR. DUPONT: Okay. They're attached to a note that says: "Dear Mr. Schirrmeister, These three articles are not listed in my report" --

THE WITNESS: That's correct.

MR. SCHIRRMEISTER: You want me to mark those?

MR. DUPONT: Can we take that note, along with the reports that that note applies to, and mark them as the next exhibit?

MR. SCHIRRMEISTER: I'm not sure if we have a copy of the note.

THE WITNESS: Yeah. I don't have the note.

MR. SCHIRRMEISTER: Is it the -- do you have the note in front of you, Andrew?

MR. DUPONT: I do.

MR. SCHIRRMEISTER: Does it say "Mariette, McDevitt and Seymour"?

THE WITNESS: No. The one he's talking about are these three. These are the three that I gave you. Those are ones I just brought today.

MR. SCHIRRMEISTER: Oh. Okay.

THE WITNESS: These are ones that I

previously gave you, and that's the ones he's talking about, I believe, in that e-mail.

MR. SCHIRRMEISTER: And then we have an additional three today that were brought to the deposition. So we have six in all.

MR. DUPONT: And the note that starts: "These three articles are not listed in my report, but I would add them to the references that I would cite in the Draper case," is that there with those initial three reports?

MR. SCHIRRMEISTER: I don't have the note.

MR. DUPONT: Okay.

MR. SCHIRRMEISTER: I'll just list the studies, and then there was an additional three as well.

MR. DUPONT: Okay. We'll mark those two sets of three as two separate exhibits.

(Exhibits E - J marked.)

MR. SCHIRRMEISTER: Okay. We're up to J. And then there are also four abstracts, which I'll mark as just one.

MR. DUPONT: Okay.

(Exhibit K marked.)

Q. (By Mr. DuPont) Doctor, do you consider yourself to be an expert in the mechanisms of benzene-induced leukemia?

A. No.
Q. Do you consider yourself to be an expert in the manner in which chemotherapeutic topo II inhibitors cause leukemia?
A. No.
Q. Do you actively research in the area of the manner in which chemotherapeutic topo II inhibitors cause leukemia?
A. No.
Q. Have you ever researched that in terms of active laboratory research?
A. Not topoisomerase II. I have been engaged in projects with topoisomerase I, which is a similar type enzyme.
Q. Who would you consider to be the lead researchers in the area of the manner in which topoisomerase II inhibitors cause leukemia?
A. I don't know.
Q. Who would you consider to be the lead experts in that area?
A. I don't know the answer to that. I know Richard Irons has studied that, Dr. Eastman has studied that, several have published papers in that area.
Q. Would you agree with me that Dr. Martyn Smith is an expert in the area of the manner in which

topoisomerase II inhibitors cause AML?

MR. SCHIRRMEISTER: Object to the form.

A. Well, much of Martyn Smith's work has been done in lymphocytes, and as I've said, lymphocytes are not the cell in question here. They're myeloid cells. So the relevance of much of his work in lymphocytes I have difficulty relating to acute myeloid leukemia. He has written many papers, he has various theories and hypotheses that he mentions in his papers, but much of this is based on lymphocyte work, which I think can be very deceptive.

MR. DUPONT: Move to strike because I don't think that answers my question.

Q. (By Mr. DuPont) My question, sir, is whether you consider Martyn Smith to be an expert in the manner in which topo II inhibitors cause leukemia.

MR. SCHIRRMEISTER: Object to the form.

A. I don't know.

Q. (By Mr. DuPont) Do you consider Dr. Smith to be an expert in the manner in which benzene metabolites cause leukemia?

MR. SCHIRRMEISTER: Object to the form.

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

Q. (By Mr. DuPont) Doctor, what are the different types of topo II inhibitors?

A. Well, there are many drugs that inhibit an enzyme. And enzyme inhibitors can be, for example, as the one we've studied in topoisomerase I, a drug that blocks the active site of an enzyme. Those would be drugs like camptothecin, which is the ones I'm familiar with. There are other drugs that can directly damage certain enzymes. There are drugs that can act as competitive inhibitors of enzymes. At the end of the day, the bottom line is they inhibit the natural function of topoisomerase system, which is to help in reproduction of DNA.

Q. You understand there to be a group of topo II inhibitors that are referred to as catalytic inhibitors?

A. Yes.

Q. And a group of topo II inhibitors that are referred to as topoisomerase II poisons?

A. Yes. That's the term that Dr. Martyn Smith used in his deposition.

Q. Can you tell me which of the studies that you've cited to or relied upon relate to topo II catalytic inhibitors?

A. Well, the studies that I cite have to do with the hematology of topoisomerase inhibitors; in other words, the consequences. And, for example, in clinical medicine it's very difficult to assess the effect of a

single topoisomerase inhibitor on a patient because of the fact that when they're used in chemotherapy, they're often multiple ones are used at the same time.

So that in general when you look at hematology literature on the consequences of topoisomerase II inhibitors, their results are often painted with a broad brush. For example, I have one article that I've referenced in my report that's an international --

Q. Doctor, what I'm asking is: Which reports address specifically topo II catalytic inhibitors versus the topo II poison?

MR. SCHIRRMEISTER: The witness was answering the question.

A. Well, I'm trying to answer the question. Dr. Martyn Smith identified etoposide, for example, as a topoisomerase II poison, and he alleged that that does not cause inversion 16 in his deposition.

Now, what I'm looking at here is an article that's an international workshop on inversion 16 by experts in this field. And what the experts in this field show is they look at large numbers of people who have inversion 16 and also another translocation called 15;17. And what they show in their tables is the most common drug associated with inversion 16 is etoposide.

That's the drug that Martyn Smith says doesn't cause inversion 16.

And, in fact, in the text of that article, it says: "In the inversion 16 subgroup, doxorubicin," which is adriamycin, "and etoposide were the most frequently administered topoisomerase II inhibitors." Talking in the inversion 16 group, excuse me, "Doxorubicin and etoposide were the most frequently administered topoisomerase II inhibitors, whereas doxorubicin and mitoxantrone were the most frequently administered T-II inhibitors in the translocation (15;17) group. The difference in frequency of administration of etoposide between the two subgroups approaches statistical significance."

So, in other words, what these authors are saying, in contrast to what Martyn Smith says, is that etoposide looks to be prominent in the cause of topo -- of inversion 16, and mitoxantrone, another type of inhibitor, prominent in the cause of 15;17 translocation.

MR. DUPONT: Move to strike.

Q. (By Mr. DuPont) My question is: Which references that you cited to in your report or which you've brought with you to your deposition today deals specifically with topo II catalytic inhibitors versus

topo II poisons?

MR. SCHIRRMEISTER: Object to the form.

A. None of the references do.

Q. (By Mr. DuPont) Okay. The etoposide is a topo II poison as opposed to a catalytic inhibitor?

A. That's what Dr. Martyn Smith says.

Q. Well, do you disagree that etop- -- excuse me -- that etoposide is a topo II poison?

A. It causes inhibition of the enzyme, which is the key issue. How it does that, I'm not an expert on.

Q. So your interpretation is that if topoisomerase II causes inhibition of an enzyme, it must be a catalytic inhibitor as opposed to a poison?

A. No. I'm saying that there are many mechanisms to inhibit an enzyme, and I'm not an expert on all the various mechanisms to inhibit topoisomerase II. There are several. But I am familiar with the medical literature on the consequences of inhibiting the activity of topoisomerase II, and I've just read you quotes from an article dealing in that subject.

Q. Prior to your executing the October 10, 2007, report, and prior to your deposition today, you have not been able to identify for me any literature that you've considered directly as it relates to topo II catalytic inhibitions?

A. I would have no reason to search out such literature.

MR. SCHIRRMEISTER: Object to the form.

MR. DUPONT: Counsel, why don't we take a brief break? We've been going about an hour.

MR. SCHIRRMEISTER: Okay.

(Recess from 2:47 p.m. to 3:00 p.m.)

Q. (By Mr. DuPont) Dr. Natelson, before the break, we discussed the McDevitt article to some extent?

A. We mentioned it, yes.

Q. Okay. And that's the 2007 publication?

A. Yes.

Q. The purpose of that article was to perform a pilot study to validate the use of FISH technique to examine chromosomal abnormalities in peripheral blood cells?

A. In patients with acute leukemia, yes.

Q. All right. And that study found, in fact, that it was a -- using FISH to analyze chromosomal aberrations in peripheral blood cells was, in fact, a valid method?

A. With certain chromosome abnormalities. In other words, they primarily studied people with trisomiate, which causes many things other than acute myeloid leukemia and can be found in lymphoid cells and

also chromosome 5 and 7 abnormalities. They didn't study inversion 16, for example.

Q. Okay. Well, they studied breaks in chromosomes at 11q, didn't they?

A. I think deletions at 11. I don't have it in front of me. But yes. Something that had to do with the chromosome 11.

Q. And what that study did was actually validate the use of looking at chromosome aberrations down in the peripheral cells as a method for comparing aberrations found in the myeloid cells. Would that be correct?

A. Well, it was a method for looking at these changes in people who actually have acute leukemia in place. In other words, not a method for predicting leukemia. These are people who already have leukemia.

And the real question that Martyn Smith's work deals with is, can one look at people who are exposed, find abnormalities in the lymphocytes and suggest that that might have a relationship to acute myeloid leukemia. In other words, could that be predictive of risk. And as I showed you with some of these articles, we already know that it's not because there are many drugs out there that cause these kinds of abnormalities in lymphocytes, but they don't cause acute myeloid leukemia.

MR. DUPONT: All right. Move to strike.

Q. (By Mr. DuPont) And I'd just like to get an answer to the question that I asked, and that is: This study actually demonstrated, meaning McDevitt -- actually demonstrated that the same types of chromosome aberrations that you see in myeloid cells caused by secondary sources are the same type that you see in peripheral blood cells caused by secondary leukemia -- secondary sources; is that correct?

MR. SCHIRRMEISTER: Object to form.

A. I think I said that, that in patients with acute leukemia in place, one can find commonalities of chromosomes in the lymphocytes in certain of the patients in that study with certain chromosome abnormalities.

Q. (By Mr. DuPont) And the commonality is between the lymphocytes and the myeloid cells, correct?

A. Yes.

Q. But it's your position that that commonality only exists with respect to certain types of chromosome damage?

A. Well, I don't know that, but at this point, we're discussing a case with inversion 16. And as we've shown, that study has been looked at in people with inversion 16, and those abnormalities are not seen.

Q. And, Doctor, your position, though, is that McDevitt looked at specifically inversion 16 and ruled inversion 16 out as a type of chromosome damage that's consistent in myeloid cells and lymphocytes?

A. Well, I don't know why he chose the subjects that he did, but, specifically, he used FISH looking at people who had certain particular chromosome abnormalities.

Q. But his study doesn't stand for the proposition that inversion 16 is not a chromosome damage that's seen both in the lymphocytes and the myeloid cells?

A. No. He did not mention inversion 16 in the article.

Q. Doctor, in coming to your opinions in this case, did you consider the differences in the incidences of inversion 16 caused by topo II poisons versus topo II catalytic inhibitors, or did you group them all together?

A. I grouped them all together because that's the way the hematologic literature approaches this issue.

Q. And what is the basis for your saying that? What is the basis for you saying that it's valid to group all of the topo II inhibitors together as opposed to looking at individual groups of topo II inhibitors?

A. Well, that's what these articles say. In other words, we have a situation where we very rarely use a single topoisomerase inhibitor on a single patient. They're often multiples. And so it's very, very difficult to separate in clinical medicine which particular topoisomerase inhibitor did what.

But you look at frequency, he says. This article I quoted mentioned -- from Anderson, and it looked in this particular article that etoposide had the most frequent association with inversion 16. But the article pointed out, as many other articles do, that many drugs cause inversion 16. They don't have to be topoisomerase inhibitors.

Q. Which, if we're going to look for specific references that you relied upon for the position of looking at topoisomerase inhibitors generally as opposed to individual groups of topoisomerase inhibitors, those would be citations 48 through 51 plus the six studies that you brought today?

A. Well, those would be some of them. Of course, I wasn't specifically trying to focus on that point, but these articles do address that.

Q. Can you cite for me any other articles that address that?

A. Well, let's just see. I don't see any other

articles that I brought today that specifically get into that subject, aside from the Anderson article. Actually, there are two Anderson articles. Let me see if I really cited the both of them.

For example, the article I cited in my report is entitled -- it's by Anderson, called "Balanced Chromosome Abnormalities." And this is another article by Anderson from 1998. And in this particular one, for example, they noted, as I think Martyn Smith said, "Translocations to 11;23 predominated following therapy with epipodoxophyllins, whereas patients with translocations such as inversion 16 most often had received anthracyclines." That's the adriamycin type. "In a multivaried analysis taking age into consideration, however, these drug-specific associations were no longer significant. Younger age and not a specific type of DNA topoisomerase inhibitor seems to predispose specifically to the development of TMDS and TAML with translocations."

And so that this article would say, essentially, the same thing that the other Anderson article, which is a few years later in the larger workshop, is that it's very difficult to separate clinically the actions of these topoisomerase inhibitors in humans because of the fact they're often used in

combination. They're not typically used as single drugs. And that's what these people say.

Q. And your review was limited to the clinical aspect because you're not a researcher on benzene's effects on the chromosomes, are you?

MR. SCHIRRMEISTER: Object to the form.

A. No. In other words, I consider myself a clinical hematologist. I'm quite familiar with the literature, as a hematologist uses it, on chromosomes and acute leukemia. I'm not a biochemist. But I'd leave it at that.

Q. (By Mr. DuPont) Do you have anything more recent than the two Anderson studies in support of your position that it is appropriate to look at topoisomerase II inhibitors as a group as opposed to specific types of topoisomerase II inhibitors in evaluating their ability to cause an inversion at chromosome 16?

A. No.

Q. What literature have you looked at that examines topoisomerase II that those types of inhibitors -- their effects on the myeloid cells with respect to chromosome damage?

A. You're talking about the use of those inhibitors in tissue culture, for example, or something like that?

Q. In tissue culture or any other methodology of studying the issue.

A. I haven't look at that specifically.

Q. Have you researched any articles that relate to looking at topoisomerase II inhibitors causing chromosome damage in progenitor cells?

A. Well, which progenitor cells? Are you talking about bone marrow myeloid cells?

Q. Stem cells, any other progenitor cells.

A. Well, I think one of the papers I referenced had to do with Dr. Iron's work looking at certain topoisomerase II inhibitors in bone marrow myeloid cells, which would be stem cells.

Q. And which paper was that?

A. This 47, the Stillman paper.

Q. Do you have an opinion, as we sit here today, as to whether or not topo II catalytic inhibitors cause inversion 16?

A. I would have to say that the literature suggests that it does, can.

Q. All right. And that opinion would apply to both inversion 16 found in lymphocytes as well as myeloid cells?

A. I would have no idea what would happen in lymphocytes. I don't know that -- I don't have any

articles that have to do with trying to get inversion 16 out of a lymphocyte.

Q. What do you consider the longest latency period to be for an AML arising as a result of exposure to a topo II catalytic inhibitor?

A. Well, in general, the leukemias that are caused by topoisomerase inhibitors are thought to be primarily short latency, let's say less than five years, as opposed to the broad category of therapy-induced leukemias, which, generally, is given around 2 to 10 or 2 to 12 years.

Q. And I want to be clear as to topoisomerase II catalytic inhibitors. Do you know what the latency period, the longest latency period found in the literature is for that?

A. I can't tell you that it's broken out. As we said, the literature, the hematologic literature, simply uses the term "AML from topoisomerase II inhibitors" without specifying their biochemical mechanism of action.

Q. I want to jump back a little bit. When we were talking about the latency period for occupational benzene exposures, you told me that you would start the latency period running from the time in which the cumulative dose reached an amount sufficient to cause

AML; is that correct?

A. Yes.

Q. What do you consider a cumulative dose sufficient to cause AML to be?

A. Well, if you look at the various studies that the -- that we rely on, the Pliofilm study, and according to Dr. Wong's analysis, that's about 200 part per million cumulative exposure to catch leukemia in that particular group. In another one I reference that's a somewhat similar but newer study, about the same number of people, they only found statistically significant incidence at around 200 part per million year exposure. I reference another one where they didn't find an increase at 40 part per million year exposure.

So I don't know with certainty what that total dose might be, but it's very high, and certainly more than 40 parts per million and probably more than 100 part per million cumulative dose exposure.

Q. Do you have an understanding of what OSHA's permissible exposure limit is for benzene?

A. In very general terms, you know, 1 part per million, I believe.

Q. And OSHA applies permissible exposure limit based on an eight-hour workday, correct?

A. Yes.
Q. And a 40-year work life?
A. Yes.
Q. So if you and OSHA -- strike that.
If you take the cumulative dose for a 40-year work life of a part-per-million-per-day exposure, that gives you a 40-part-per-million-year dose, does it not?
A. Yes.
Q. And OSHA, in setting the permissible exposure limits, were required to consider the effects of the economy on reducing the permissible benzene exposure levels; is that correct?
MR. SCHIRRMEISTER: Object to the form.
A. I would not be an expert on what OSHA uses as their reasoning.
Q. (By Mr. DuPont) Okay. Nonetheless, OSHA believes that you will see a doubling of the risk of AML at 40 part per million years with cumulative exposure?
MR. SCHIRRMEISTER: Object to the form.
A. I don't know that.
Q. (By Mr. DuPont) Assuming that that's what OSHA believes, your threshold would be, what, five times that level?
MR. SCHIRRMEISTER: Object to the form.

MR. STOFKO: Object to the form. Chris Stofko.

A. Well, as I've cited, the literature suggests it's considerably higher than that.

Q. (By Mr. DuPont) Okay. So the federal government has it wrong?

MR. SCHIRRMEISTER: Object to the form.

A. No. The federal government wants to be protective, and so they would want to set a limit less than what might cause large amounts of leukemia.

Q. (By Mr. DuPont) Do you know what the federal government found the risk of AML to be at 40 parts per million years of exposure?

A. No.

Q. You mentioned that there was three epidemiological studies that you rely upon. The first was authored by Otto Wong. The second one also dealt with 200 part per million years exposure. Who was that authored by?

A. Costantini. That's No. 28.

Q. And the third study that you claim does not find a doubling of the risk at 40 parts per million years of exposure, which study is that?

MR. SCHIRRMEISTER: Object to the form.

A. No. 23, the Bloeman study, B-L-O-E-M-A-N.

Q. (By Mr. DuPont) As a clinician, do you have to find a doubling of the risk association in order to accept in the treatment of a patient that there is a causal relationship between an agent and a result?

MR. SCHIRRMEISTER: Object to the form.

A. Not if there's -- well, I would like to see more than two if you had a single article, but what you're looking for, as I think I said before, is consistency. If you had a number of studies that all found a doubling of the risk, that would make it very likely to be true. If you simply found one study that found a tripling of the risk and it wasn't confirmed with other studies, that wouldn't prove much to me.

Q. (By Mr. DuPont) Doctor, you're familiar with the Chinese studies authored by Hayes, et al.?

A. Yes.

Q. And they find a cumulative dose of 40 part per million years?

A. Well, that's what they allege.

Q. I'm sorry?

A. That's what they allege, yes.

Q. And you're familiar with the Deborah Glass Health Watch study?

A. Yes.

Q. And they find a cumulative dose of 8 part per

million years with a more than doubling the risk for leukemia?

A. There is no doubling of the risk in that study because the most recent version of that shows that the incidence of leukemia in that cohort is identical with the national average for leukemia.

Q. My question is that the 2003 Deborah Glass Health Watch study shows a cumulative dose of 8 part per million years benzene causing more than a doubling of the risk for leukemia. Isn't that correct?

MR. SCHIRRMEISTER: Object to the form.

A. That's what she alleges, yes.

Q. (By Mr. DuPont) All right. You would agree with me that based on those studies, there's been a downward trend in the cumulative dose of benzene leading to a doubling of the risk of AML?

MR. SCHIRRMEISTER: Object to the form.

MR. STOFKO: Object to the form. Chris Stofko.

A. I wouldn't agree with that because, as I said, the only thing the Glass study demonstrates is that when you don't have a control group that has the same -- particularly in a small study, when you don't have a control group that has similar incidence to the general population, you can come to outlandish conclusions. And

in that study, they had about eight cases of leukemia in about 18,000 people. In the Pliofilm study, with only a tenth as many people -- about 1,800 people -- they had seven cases of leukemia.

So just looking at that study, you would guess that it wasn't a hotbed of AML, and in the later analysis by Gunn, it shows that the incidence of leukemia was identical to the -- to the population at large.

Q. (By Mr. DuPont) What do you rely upon for ascertaining the amount of benzene exposure Mr. Draper had?

A. Well, I am not a benzene modeler, and I don't know what exposure he had, so I have to rely on the various experts that model it based on what compounds he used, how frequently he used them, what level of benzene was in those compounds, whether it was ventilated, not ventilated. There's considerable guesswork there, but that's what those types of experts do. And so I certainly, on my own, couldn't come up with an accurate estimate of what exposure he had.

Q. Have you reviewed the transcript from Dr. Smith's testimony in this case from his deposition?

A. Yes.

Q. And what are your comments on that? First of

all, did you take any notes on that transcript?

A. No.

Q. What are your comments on his testimony?

A. Well, I guess the overall thing that I noticed that was of interest to me about the testimony was that he believes that cigarette smoking can cause inversion 16. Now, I'm not aware of any literature that supports that, but let's assume that's so. He says it's so.

He also indicates that most people with AML, perhaps 80 percent, don't have it from industrial benzene exposure. So, therefore, a person with no industrial benzene exposure can catch acute leukemia and inversion 16, and someone who's a smoker, according to Martyn Smith, can catch acute leukemia with inversion 16, yet when it comes to his differential diagnosis, those two areas are not considered. In other words, it's like they don't exist.

And so what he's telling us is he believes that an extraordinarily rare cause of leukemia, in his mind, is what caused this illness over what he admits are the far more common possibilities.

Q. Are you telling me that Dr. Smith hasn't considered cigarette smoking in his differential diagnosis, or are you telling me that he's considered it and found that benzene exposure is a much more

significant risk than that presented by cigarette smoking?

A. Well, he doesn't discuss in that deposition why. In other words, he says he believes -- he simply comments when asked whether smoking can cause inversion 16, he said yes. He also commented that most leukemia is de novo without any known exposure to benzene. His conclusion, though, is this man has leukemia from benzene. And he doesn't go into why he eliminates cigarette smoking or what makes Mr. Draper immune to de novo AML.

Q. What is the risk of AML from -- strike that.

You'd agree with me that there's a much higher population of individuals who smoke cigarettes in this country than there are individuals who are occupationally exposed to benzene?

A. Yes.

Q. And you would agree with me that the difference in the size of those populations is an order of magnitude, isn't it?

A. Yes.

Q. And due to the orders of magnitude difference in those exposed to cigarette smoke and those occupationally exposed to benzene, that has a substantial effect on the risk of contracting AML from

cigarette smoke versus the risk of contracting AML from benzene exposure, doesn't it?

MR. SCHIRRMEISTER: Form.

A. I think you have to specify the group. In other words, many people can be occupationally exposed to small amounts of benzene, but that's not why they catch leukemia. Many people smoke cigarettes, but that's not why they catch leukemia. They're sort of apples and oranges. But what you can say at the end of the day is that there are many more people catching leukemia from cigarettes than occupational exposure to benzene.

Q. (By Mr. DuPont) And that's because there's many, many more people smoking cigarettes than there are occupationally exposed to benzene. Would that be correct?

A. Well, that's true.

Q. Okay. Besides benzene, can you name any other leukemogens in cigarette smoke?

A. Well, there's some radioactivity alleged to be in cigarette smoke. There are many chemicals. But we don't know why cigarette smoking causes leukemia, and I can't name any other known leukemogens besides benzene.

Q. Would you agree with me that it's the benzene content of cigarette smoke that is associated with the

leukemia risk from cigarette smoking?

MR. SCHIRRMEISTER: Object to the form.

A. No. We know that it's not. There's not enough benzene in cigarette smoking to cause leukemia. And the truth of the matter is, we really don't know that it's a chemical in the cigarettes. It could be an effect that the cigarette smoke has on the lungs cause production of certain compounds in the body. I don't think we even know that it's a specific compound in the cigarettes. All we know is that cigarette smokers have a higher risk of AML.

Q. (By Mr. DuPont) Can you point to any studies that establish that radioactive content of cigarette smoke that causes leukemia?

A. No.

Q. Can you tell me what percentage of the risk of AML, of causing AML, benzene accounts for within cigarette smoke?

A. I would say very little because the part-per-million-year accumulations from cigarette smoking, depending on who writes about it, could be as low as a half-a-part-per-million-year accumulation or as high as five or six. But neither one of those numbers, in my mind, is high enough to cause AML.

Q. Did you come up with a cumulative dose of

benzene exposure consumed by Mr. Draper by virtue of his smoking cigarettes?

A. Did I? No.

Q. In looking at the report of Bradford Russell, were you aware in using that data in your evaluation of whether Mr. Draper's AML was caused by benzene exposure -- were you aware that Mr. Russell did not account for dermal absorption of benzene?

A. I don't recall. I did read what was sent to me. I don't remember all of the details. I just remember the final conclusion that he mentioned.

Q. Doctor, what is the Environmental Protection Agency's position on whether or not there's a threshold for which benzene does not cause leukemia?

MR. SCHIRRMEISTER: Object to the form.

A. I couldn't tell you accurately that.

Q. (By Mr. DuPont) Do you know what the IARC's position is with respect to a threshold below which benzene cannot cause leukemia?

MR. SCHIRRMEISTER: Object to the form.

A. No.

Q. (By Mr. DuPont) Same question for the National Institute of Occupational Safety and Health.

MR. SCHIRRMEISTER: Object to the form.

A. No.

Q. (By Mr. DuPont) Doctor, what is your opinion as to the relative risk from cigarette smoking presented to Mr. Draper's leukemia compared to benzene exposure? And let's start off with your assumption that he had .123 part per million years of benzene exposure.

MR. SCHIRRMEISTER: Object to the form.

A. Well, I would say that neither -- if that were the case, neither the occupational exposure nor the cigarettes would have enough benzene to cause AML.

Q. (By Mr. DuPont) Well, I want to know benzene exposure occupationally versus cigarette smoking.

A. Well --

MR. SCHIRRMEISTER: Object to the form.

Q. (By Mr. DuPont) What is the risk between -- for causing leukemia created by the cigarette smoking as a whole versus the benzene exposure occupationally at the assumption Mr. Russell makes of .123 part per million years?

A. Okay. I would say that there is no increased risk at the .125 part per million years. I would say that as a 20-pack-year history person, he's got a double the risk for a cigarette-induced AML, so, therefore, his risk is twice what the risk would be from his occupational exposure.

Q. And if we were to change Mr. Draper's

occupational exposure to 8 part per million years cumulatively, how would that affect your assessment of the risk of his contracting AML from occupational benzene exposure?

MR. SCHIRRMEISTER: Object to the form.

A. I'd say 8 part per million years would still be too low to be the threshold to catch AML.

Q. (By Mr. DuPont) Regardless, unless his exposure is above 200 part per million years, you would testify that his exposure was too low to cause his AML?

A. Well, I don't know exactly what it is. As I've said, it could be as low as 100, it could be slightly lower than that, but we know it's higher than 40 based on the studies and the literature. And I would say at the levels you're talking about, 8 or .125, those are just not in the ball game.

Q. And also for you, 40 part per million years would not be in the ball game as creating a doubling of the risk of causing AML?

A. I would say it would be very unlikely.

Q. And under no circumstances would you accept that someone with 40 part per million years cumulative exposure to benzene contracted AML from benzene exposure?

A. Well, as I've said, I think, repeatedly, I

cannot tell why any given person with AML has that AML. There are certain things we look at. You -- if you showed me a person, for example, who had a period of myelodysplasia, was a heavy benzene worker, had a minus 7 chromosome defect and an estimate was made because it is, in fact, just an estimate of 40, I would say, "Well, that's a consideration because maybe really that 40 was really 80 or 100 or 200." Because all of these estimates are just guesses.

But I think when you're dealing with levels as low as 8 or below that, it's just, for me, not a consideration, especially when you're talking about a type of leukemia that's never been described from benzene exposure at any level.

MR. DUPONT: Object to the nonresponsive nature to that question.

Q. (By Mr. DuPont) Doctor, is it your position that AML M4 has never been described in the literature as arising from benzene exposure?

A. Well, M4 leukemia -- I think in the Chinese studies there was M4 mentioned, and inversion 16 can be either an M2 or an M4. I would say that the descriptive nature of the leukemia is probably there from benzene, but the chromosome analysis is not.

Q. So you would agree that M4 leukemias are

related to benzene exposure, correct?

MR. SCHIRRMEISTER: Object to the form.

A. I think they could be. Yes.

Q. (By Mr. DuPont) But you would disagree to the extent that the M4 has an inversion of 16?

A. Well, the inversion of 16 needs to be demonstrated. In other words, what these -- the FAB classification that we look at by morphology is oftentimes very iffy, and pathologists don't always agree on how they should be classified.

And most people don't pay much attention to that classification anymore, and it's clear that the chromosome analysis is the key issue in determining prognosis and many other things. And, to a certain extent, the M classification is obsolete. But be that as it may, if we're talking about inversion 16 leukemia, I need to see the chromosome analysis.

Q. Doctor, when did chromosome analysis -- strike that.

You'd agree with me that Mr. Draper's polycystic kidney disease has been caused by his acute myelogenous leukemia and treatment?

A. I would say it has absolutely nothing to do with his acute myeloid leukemia and treatment.

Q. Would you agree with me that his polycystic

kidney disease has been exacerbated or worsened from his AML and treatment therefor?

A. I don't know that. It would be possible.

Q. Well, have you treated patients with polycystic kidney disease before?

A. Yes.

Q. Have you treated patients with leukemia that have also had polycystic kidney disease?

A. I can't remember a single one who's had that combination. Polycystic kidney disease is actually quite common. I've seen many patients with it over the years, but I just simply can't recall seeing one with acute leukemia.

Q. Are chemotherapeutic agents used for the treatment of AML toxic to the kidneys?

A. They certainly can be, yes.

Q. Well, in fact, they are, aren't they?

A. Depends on which agent you're talking about. In other words, the platinum type agents are highly toxic to the kidney. Agents like cyclophosphamide or vincristine are not particularly toxic if the patient is well hydrated. Some are intermediate. It depends on which agent you're talking about.

Q. Would you agree with me that if a chemotherapeutic agent used for AML was toxic to the

kidneys, then that chemotherapeutic agent would exacerbate or make worse the polycystic kidney disease condition?

A. Well, if chemotherapy damaged the kidneys -- and I would also point out that when you're giving people chemotherapy, you often need to give them antibiotics if they get infections. And so sometimes if you have a person who has a kidney problem from chemotherapy, it isn't the chemotherapy drug; it's the consequences of the chemotherapy drug in terms of other medications. But it isn't unusual to get kidney damage in people being treated for acute leukemia, but frequently it's not the drug itself.

Q. But would it also not be unusual that a chemotherapeutic agent that is toxic to the kidneys would make worse any preexisting kidney disease?

A. Well, certainly, if you have preexisting kidney disease and you give any drug, whether it's chemotherapy or antibiotic, and it further damages the kidneys, that worsens the situation with the polycystic kidney disease.

Q. Doctor, is it your opinion that methotrexate is not carcinogenic?

A. That is the position of the medical literature.

Q. So the answer is yes, it's your opinion that methotrexate is not carcinogenic?

A. Methotrexate does not cause acute leukemia or non-Hodgkin's lymphoma, to be more specific.

Q. Well, I want to know, generally: Is it your opinion that methotrexate is not carcinogenic?

A. I don't know about other tumors, skin tumors and so on. I can only comment about non-Hodgkin's lymphoma and acute myeloid leukemia.

Q. How about lymphoma? Does methotrexate cause lymphoma?

A. I noticed that was very interesting that Martyn Smith said that. He implied I wasn't familiar with the literature, epidemiologic literature on that subject, and, in fact, that's why I included one of the articles I did by this Mariette person, because that article gives the answer to what you're asking.

MR. DUPONT: Objection as nonresponsive.

Q. (By Mr. DuPont) Is it your opinion that methotrexate does not cause lymphoma?

A. The literature is very clear that methotrexate does not cause non-Hodgkin's lymphoma.

Q. You're saying "non-Hodgkin's lymphoma" and I'm saying "lymphoma." My question is: Is it your position -- is it your opinion that methotrexate does

not cause lymphoma?

A. Well, there's a study -- in fact, the one I referenced from France found an increase in Hodgkin's disease, but Hodgkin's disease is only about a tenth in frequency of non-Hodgkin's lymphoma. And we know that Hodgkin's disease can be caused by viral diseases which are caused by immunosuppression. So I would say that particular study suggested an increase in Hodgkin's lymphoma but not non-Hodgkin's lymphoma.

Q. Okay. We're kind of dancing around this and my question is very specific and I just want to get a response to my question.

Is it your opinion that methotrexate does not cause lymphoma?

A. That is my opinion. Correct.

Q. Would you agree with me that benzene is a more potent carcinogen than cigarette smoke?

A. It depends on the dose.

Q. Well, if we were to take equal doses of benzene and cigarette smoke, would you agree with me that benzene is a more potent carcinogen than cigarette smoke?

A. Absolutely not because smokers get enormous amounts of lung cancer, 2,000 times what the general population does. They don't get any -- they might get a

doubling of the risk of leukemia. And we don't know that that leukemia is due to benzene.

Q. Would you agree with me if we were to take equal doses of exposure to benzene and cigarette smoke, that benzene is a more potent leukemogen than cigarette smoke?

A. Well, I don't see how you can do that experiment. In other words, if you take someone who's a heavy smoker, let's look at the high side of what benzene they might accumulate. That might be 5 or 6 part per million years. Well, 5 or 6 part per million years of somebody who gets it solely from benzene doesn't have AML.

Q. Well, you're comparing the benzene content of cigarette smoke to benzene exposure outside the context of cigarette smoke?

A. I thought that's what you asked.

Q. Okay. Well, perhaps I wasn't clear, for which I apologize.

Is it your position that it's the benzene content of cigarette smoke that causes leukemia?

A. My position, it is not the benzene content.

Q. If we were to look at somebody occupationally exposed to benzene at an equal dose of cigarette smoke in another individual, would you agree with me that

looking at the cigarette smoke dose as a whole, not just as benzene content, versus the occupational benzene dose, that benzene is a more potent leukemogen than cigarette smoke as a whole?

MR. SCHIRRMEISTER: Object to the form.

A. I don't know how to answer the question. In other words, I don't exactly know what you're asking. If you're asking me whether somebody who has the same content of benzene, I can figure that one out because we've said that benzene and cigarettes may only be a maximum of 5 or 6 part per million years, and that doesn't cause leukemia.

But if you're asking me -- I can't figure it out exactly. I can't make the comparison that you're asking.

Q. (By Mr. DuPont) Doctor, are you familiar with any cohort studies that implicate the importance of dermal absorption of benzene in assessing the cause of leukemia amongst those exposed to benzene?

A. No.

Q. Would you agree with me, Doctor, that it's also important to analyze, in addition to cumulative doses of exposure, intermittent peak exposures that an individual sustains to benzene?

A. Well, in general, the literature suggests that

the cumulative dose is the major -- the major number we're looking for. And, of course, a very high peak dose ultimately gives you more cumulative dose.

Q. Okay. But would you agree with me that intermittent peak doses are more important in determining the cause of leukemia from benzene exposure than is just looking at cumulative doses?

MR. SCHIRRMEISTER: Object to form.

A. No. I don't think the literature would confirm that.

Q. (By Mr. DuPont) Do you know what the EPA's position is on that?

A. No.

MR. DUPONT: Why don't we take another short break? Is that okay with you, Doctor?

THE WITNESS: Yes.

(Recess from 3:51 p.m. to 4:03 p.m.)

Q. (By Mr. DuPont) Doctor, would you agree that hematotoxicity increases the risk of benzene-induced leukemia?

A. Hemato- -- I'm not certain what you're asking. You mean if I take somebody who already has an existing damaged marrow and then gave them benzene?

Q. Well, what is your understanding of what hematotoxicity is?

A. Well, hematotoxicity would be if I gave a chemical or a drug and I saw a fall in the blood counts; in other words, a fall in the white blood cell count, the red blood cell count, the platelet count, or reduced cellularity of the bone marrow. In other words, something that damages production of blood would be hematotoxicity.

Q. Okay. Would you agree with me that somebody whose blood is hematotoxic has an increased risk for developing benzene-induced leukemia when they're exposed to benzene?

A. I think what you're asking me is if I have somebody who's got abnormal blood counts and then I hit them with benzene, are they more likely to catch leukemia than someone who has normal blood counts when I hit them with benzene?

Q. Correct.

A. I think that's the question you're asking. I would have no idea.

Q. Have you reviewed any studies that address that issue? In other words, are there any studies that stand for the proposition that hematotoxicity increases the risk of benzene-induced leukemia?

A. Well, maybe in this way. The Aksoy studies, the Turkish shoe worker studies, what Dr. Aksoy noticed

is that if he had people who got marked depression of their blood counts as a consequence of the benzene exposure, even though the blood counts recovered, several years later, they might come down with leukemia.

In other words, what he was showing is that they had enough benzene to actually depress the bone marrow. And I think if you had enough benzene to actually depress the bone marrow, then that suggests there might be a risk of later development of leukemia.

Q. What if you had enough benzene to depress the white blood count?

A. I don't know -- I don't know the answer to that, and I guess it would depend on how low. You know, if I saw a person -- and I did see one such person several years ago who was a petrochemical worker who repeatedly in the arena would get his white cell count knocked down to very low levels, and then he would recover when he was taken away from the exposure. And he went through that cycle several times. Ultimately, he developed acute leukemia. Whether or not a single depression would do that, I don't know the answer to that.

Q. So what you're telling me is yes, you believe decreased white blood cell counts can lead to an increased risk of benzene-induced leukemia if someone is

exposed to benzene while his counts are decreased. You just don't know how low those counts have to be or how frequent the exposure has to be?

MR. SCHIRRMEISTER: Form.

A. Well, I can't think of a particular study that takes people with low blood counts already and then exposes them to either benzene or chemotherapy and sees if they get higher rates of leukemia than someone with normal blood counts that they expose to benzene or chemotherapy drugs, for example. I don't know the exact model for what you're describing. All that I can comment is that if someone has enough drug to depress the bone marrow, obviously, they've gotten a very substantial dose of that drug.

Q. (By Mr. DuPont) How much benzene exposure is necessary to depress the white blood cell count?

A. I don't know that with accuracy. I think -- I really can't say. I have that information, I think, in my files, but I'd probably better not comment on that. I don't recall the exact numbers.

Q. Would you agree with me that there's no threshold for the hematologic effects of benzene exposure, particularly on white blood cell counts or lymphocytes?

MR. ORLET: Object to the form.

A. No. I don't understand that question because that would suggest that eating a banana might lower your white count. There's benzene in bananas. I mean, you have to have a substantial dose of benzene to lower the blood count, and a very substantial cumulative dose to catch leukemia.

Q. (By Mr. DuPont) How substantial of a dose do you have to have to benzene to lower your white blood cell count?

A. I'd have to say -- this is an educated guess -- I think in the range of acutely 50 part per million or something of that nature. I'd have to really look it up from my files. I can't tell you with any bona fide accuracy.

Q. Are you familiar with Elizabeth Ward's study on the rubberworker pliofilm cohort that was published in 1995?

A. No. I'm generally familiar with that cohort. I don't know that particular study.

Q. Doctor, are you familiar with any studies that link topo II catalytic inhibitors to inversion 16 in AML?

A. I just commented on -- or previously, on the Anderson study that mentioned several types of topoisomerase II inhibitors that could be linked to

inversion 16, and these included the mitoxantrone type and the etoposide type and the adriamycin type.

Q. And excuse my ignorance, but are any of those, or any other types reported by Anderson, catalytic inhibitor topo II's?

A. I don't know the biochemistry of topoisomerase II well enough to tell you that. I know they inhibit the enzyme.

Q. If you could turn to the -- I think you said October 8th draft of Mr. Russell's report. Can you tell me what the cumulative dose calculation he makes is in that report?

A. Now, which report, my letter or the other report?

Q. You testified earlier, and we marked it as one of the first three exhibits, that you received the October 8th draft report from Bradford Russell.

A. Let's see if we can find that.

MR. SCHIRRMEISTER: Here it is.

A. Okay. I'm sorry. I have the report. And what is it, again, that you're asking?

Q. (By Mr. DuPont) I'd just like for you, since I'm not there with you, unfortunately, and I can't look at it -- if you can tell me what cumulative dose of benzene he calculates in that draft of his report.

A. Well, this is --

MR. SCHIRRMEISTER: Might want to just -- he can read the last paragraph into the record.

MR. DUPONT: Yeah. That would be fine.

MR. SCHIRRMEISTER: Just read that.

A. Yeah.

"If Mr. Draper's estimated daily exposure to trace benzene while working as a painter or painter's helper was .0668 part per million for eight hours per day, 5 days per week and 48-50 weeks per year, then this would be equivalent to .0668 part per million years. Mr. Draper painted with DuPont paints for approximately 3.083 years during the period from 1979 to 2003, therefore, his estimated cumulative exposure to trace benzene from painting with DuPont products would have been .0668 part per million years," star, "3.083 years or .021 part per million years."

Q. (By Mr. DuPont) From looking at that draft report, can you tell whether or not Dr. Russell is calculating exposure to benzene from solvent use as distinguished from paint exposure?

A. Well, he says: "With the summary data presented, it is possible to estimate Mr. Draper's 8-hour time-weighted average concentration to trace benzene based on potential exposures during 6 major

tasks." And -- let me see about that. Well, he lists in here aerosols, gas, diesel, thinners, wash thinner, PrepSol, putty, paints. So he's mentioned a number of items.

Q. You previously said that the products Mr. Draper worked with contained trace amounts of benzene. What is your basis -- first of all, what do you mean by "trace amounts"?

A. Not pure benzene. In other words, if one -- if he used a thinner, it might conceivably have small amounts of benzene in it, as you might say a contaminant or a trace material, but it's not pure benzene.

Q. What is your understanding of how much benzene is in petroleum-based thinners?

A. I don't know how much is, and I'm sure it varies by product.

Q. Do you have an understanding of a range of contents of benzene in petroleum-based solvents?

A. No.

Q. Can you tell me whether it's more or less than .1 percent?

A. I would think it would be less than .1 percent.

Q. And what's your basis for saying that?

A. Well, because I don't think it would be much

higher. In other words, the solvents are made usually by differential distillation, and the amount of benzene that would be in the particular cuts they use for these solvents wouldn't be very much.

Q. Would you agree with me that Mr. Draper was being exposed on a daily basis to benzene when working with the defendants' products?

MR. SCHIRRMEISTER: Object to the form.

MR. ORLET: Object to the form.

A. Probably, yes.

Q. (By Mr. DuPont) Have you spoken with anyone from DuPont concerning the benzene content of DuPont products?

A. No.

Q. Have you spoken with any of Mr. Draper's coworkers regard the manner in which DuPont or any other defendants' products were used?

A. No.

Q. And you haven't treated Mr. Draper?

A. That's correct.

Q. Doctor, have you ever testified on behalf of a plaintiff in a case where it was alleged that benzene exposure caused an injury?

A. No.

Q. Have you ever diagnosed a patient and found

that that patient's AML was caused by benzene exposure?

A. Well, as I mentioned, I had one patient who I felt confident his leukemia was caused by chemical exposures, but he had exposures to a number of things besides benzene. But benzene was one of them and certainly could have been the most important chemical.

Q. Doctor, you testified in a case concerning Charles Wilson?

A. Yes.

Q. Have you given any testimony since that deposition? And just to refresh your recollection, that was on September 17, 2007.

A. Yes. The only other one I have is on a case called Dorsey McCann. That was on September 26th, '07.

Q. Do you recall who represented the plaintiff in that case?

A. No.

Q. On behalf of which defendant or defendants did you issue an opinion in that case?

A. Well, the case was Dorsey McCann versus The Sherwin Williams Company, and there probably were a number of defendants. I can't remember which -- the attorney that originally asked me to see the case, who he was representing.

Q. Was it Mr. Stofko that asked you to review the

case?

A. I don't think so, but I don't remember. Could have been.

Q. And what was your opinion in the McCann case?

MR. SCHIRRMEISTER: I don't think it's appropriate to question the witness about opinion in another case. I'm going to instruct him not to answer. You can get the deposition from the other attorney, if you want it.

Q. (By Mr. DuPont) Well, can you tell me what disease was allegedly caused by benzene exposure in the McCann case?

A. It was AML.

Q. Can you tell me what subtype of AML it was?

A. No. I can't remember that in enough detail to comment.

Q. And did that individual, Mr. McCann, have any damage to his chromosomes?

A. I don't remember whether he did or not.

Q. And it was your testimony in that case that the gentleman's benzene exposure did not cause his AML?

MR. SCHIRRMEISTER: We're not going to go there. It's a different case. Witness is instructed not to answer.

MR. DUPONT: Counsel, I think it's more

than relevant to his opinions in this case.

MR. SCHIRRMEISTER: Well, it may be, but that case is pending, and I'm not going to have the witness testify about another case in which he's not represented by his counsel.

Q. (By Mr. DuPont) Doctor, if we were to break the amount of time that you spend professionally on testifying in litigation versus treating patients, how does that breakdown fall in terms of -- is it 50/50? 75/25?

A. Oh, I would say it's much less than 75/25. I can give you this figure because I was asked something like that a year or two ago, and so I was asked relatively how much income I made from this, which I suppose in some respects would correlate to time spent. And what we calculated is for the ten-year period from 1995 to 2005, roughly about 12 percent of my income was from testimony. And so extrapolating, conceivably 12 percent of my time was too.

Q. When did you first testify in -- first start testifying in litigation?

A. Around 1992.

Q. Since then, approximately how many cases have you given testimony in?

A. Well, keep in mind that many on this list are

not benzene cases, but the total number of cases I've testified in are 55 since 1992.

Q. And in how many of those cases did you provide an opinion on behalf of the plaintiff in the litigation?

A. I could count them up. Let's see. Looks like nine cases I testified for the plaintiff.

Q. Did any of those cases involve exposure to benzene?

A. No.

Q. Did any of those cases involve an individual who was injured by exposure to any chemical?

A. In some cases it was injured by exposure to drugs, which I guess you could classify as a chemical.

Q. All right. And would those have been cases relating -- brought against the manufacturer or maker of the drug, or cases brought against another physician that had administered the drug?

A. Usually another physician.

Q. So those would have been medical negligence cases?

A. Yes.

Q. Do you have any criticisms of the opinion issued by Dr. Agura in this case?

A. I have to look at it again.

THE WITNESS: Do we have that anywhere in

the material?

MR. SCHIRRMEISTER: Andrew, do you mean does he have any criticism of Mr. Draper's care and treatment for his condition?

MR. DUPONT: No. I want to refer to -- I believe it's a January 30 --

THE WITNESS: Oh. 2007 patient letter? Yeah. If I can find that.

Q. (By Mr. DuPont) January 30, 2007.

A. I can't find it. I don't know where it is.

MR. SCHIRRMEISTER: Here it is.

A. All right. The letter from Dr. Edward Agura.

Q. (By Mr. DuPont) Yes, sir.

A. Yes. And what is it you're asking about it?

Q. I'm asking if you have any comments on his opinions set forth in his report.

A. Well, his opinion is based on the fact that Mr. Draper told him he had exposures to solvents and Dr. Agura says that -- he opines that the occupational exposure contributed to the leukemia. I don't agree with that.

Q. Okay. So you would not agree with Dr. Agura that occupational exposure to solvents containing benzene contribute to leukemia?

A. No, they can. But in this particular case.

Q. And you understand that Dr. Agura is Mr. Draper's treating oncologist hematologist?
A. Yes.
Q. Doctor, have you brought with you all of your correspondence between yourself and any attorney for a defendant in this case?
A. Yes.
Q. Can we go ahead and mark those as the next exhibit?
A. I don't know what that would be, aside from various e-mails here, which I have. Here are three. See if I've got any --
Q. I'd like to include any e-mail, letter, fax, notes, anything of that nature.
MR. SCHIRRMEISTER: What's the next number?
THE REPORTER: L.
(Exhibit L marked.)
MR. DUPONT: Okay. Do we have that?
MR. SCHIRRMEISTER: Yep. It's marked.
MR. DUPONT: What's left? Do we have the original citations from his report? I just kind of want to sort out what I want to attach as an exhibit and what I don't want to attach.
MR. SCHIRRMEISTER: You have all of his

literature references. You got something -- what was it you asked Dr. Natelson about, index of authorities or something like that? What was the language that you used?

MR. DUPONT: You know, it might have been something that your office put together.

MR. SCHIRRMEISTER: Yeah. It was.

MR. DUPONT: "Dr. Ethan Natelson File Inventory, December 3, 2007"?

MR. SCHIRRMEISTER: Right. My office prepared that.

MR. DUPONT: Okay.

MR. SCHIRRMEISTER: That's a complete set of his file, other than what we've marked today.

MR. DUPONT: So is this -- all right.

MR. SCHIRRMEISTER: He brought some additional articles. He brought some published -- some abstracts. There's a Russell draft, I think, that we've marked, and some additional e-mail correspondence. I believe that's it.

MR. DUPONT: Let me ask you: Do you have a copy of the CD-ROM that you used to forward me his file?

MR. SCHIRRMEISTER: Do I have one?

MR. DUPONT: Yes. With you there.

MR. SCHIRRMEISTER: No, I don't.

MR. DUPONT: I guess what I'm trying to get at is -- and we can go off the record.

(Recess from 4:32 p.m. to 4:35 p.m.)

(Exhibits M, N and O marked.)

(Exhibit P to be marked.)

MR. DUPONT: Back on the record real quick.

Q. (By Mr. DuPont) Doctor, have you understood all my questions today?

A. Virtually all of them.

Q. Okay. And have I treated you fairly?

A. Absolutely.

Q. Excellent.

MR. DUPONT: Well, I appreciate your coming down, and hope we speak with each other again.

THE WITNESS: All right.

MR. SCHIRRMEISTER: Thank you.

(Deposition concluded at 4:35 p.m.)

(Signature was waived.)

CAUSE NO. 199-02538-04		
DUANE DRAPER, ET AL.,	)	IN THE DISTRICT COURT OF
	)	
PLAINTIFFS,	)	
	)	
VS.	)	COLLIN COUNTY, TEXAS
	)	
PPG INDUSTRIES, INC., ET AL,	)	
	)	
DEFENDANTS.	)	199TH JUDICIAL DISTRICT

REPORTER'S CERTIFICATION
DEPOSITION OF ETHAN A. NATELSON, M.D.
DECEMBER 10, 2007

I, Kimberly J. Carter, Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, ETHAN A. NATELSON, M.D., was duly sworn by the officer and that the transcript of the oral deposition is a true record of the testimony given by the witness;

That examination and signature of the witness to the deposition transcript was waived by the witness and agreement of the parties at the time of the deposition;

That the original deposition was delivered to Mr. Andrew J. DuPont;

That the amount of time used by each party at the deposition is as follows:

Mr. Andrew J. DuPont 2:30

Mr. Christopher Stofko 0:00
Mr. Joseph C. Orlet 0:00
Mr. Bryan D. Pollard 0:00

That $_____ is the deposition officer's charges to the Plaintiff for preparing the original deposition transcript and any copies of exhibits;

That pursuant to information given to the deposition officer at the time said testimony was taken, the following includes all parties of record:

Mr. Andrew J. DuPont, Attorney for the Plaintiff
Mr. Andrew C. Schirrmeister, III, Attorney for the Defendant E.I. DuPont De Nemours and Company
Mr. Christopher Stofko, Attorney for the Defendant PPG Industries, Inc., and The Sherwin Williams Company
Mr. Bryan D. Pollard, Attorney for the Defendant Illinois Tool Works, Inc.
Mr. Jospeh C. Orlet, Attorney for the Defendant BASF

That a copy of this certificate was served on all parties shown herein on _____ and filed with the Clerk pursuant to Rule 203.3.

I further certify that I am neither counsel for, related to, nor employed by any of the parties or attorneys in the action in which this proceeding was taken, and further that I am not financially or otherwise interested in the outcome of the action.

Certified to by me this _____ day of _____, 2007.

_____

KIMBERLY J. CARTER, Texas CSR No. 7721
Expiration Date: 12/31/08
FAST PACE REPORTING, INC.
Firm Registration No. 389
2425 West Loop South, Suite 200
Houston, Texas 77027
(713) 650-3500