SUPERIOR COURT FOR THE STATE OF CALIFORNIA
FOR THE COUNTY OF LOS ANGELES
SOUTHEAST DISTRICT

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DAVID GERCHMAN and THERESA GERCHMAN, Plaintiffs,	:	CASE NUMBER VC 060 106
vs.	:	Assigned to: Hon. Raul A. Sahagun
BERRYMAN PRODUCTS, INC.; CRC INDUSTRIES, INC., which will do business in California as Pennsylvania CRC INDUSTRIES, INC.; (Continued)	:	Dept. SE "F" Complaint Filed: December 12, 2012

- - -

Thursday, July 26, 2012

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Oral deposition of GREGORY SARNA, M.D., taken pursuant to Notice at the offices of Hogan Lovells, 1999 Avenue of the Stars, Suite 1400, Los Angeles, California beginning at ^10:00 a.m., Pacific Time, before Brigitte A. Strain, a Federally Certified Registered Professional Reporter and Notary Public.

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Continued:

FORD MOTOR COMPANY; :
LOCTITE CORPORATION :
n/k/a HENKEL CORPORATION :
ILLINOIS TOOL WORKS, INC. :
Solely as successor-in-interest :
to Permatex, Inc. :
RADIATOR SPECIALTY COMPANY :
UNITED STATES STEEL CORPORATION :
SAFETY-KLEEN CORP. :
SAFETY-KLEEN SYSTEMS, INC. :
3M COMPANY :
E.I. DUPONT DE NEMOURS & COMPANY :
SEA FOAM SALES COMPANY :
JUSTICE BROTHERS, INC. :
HANSON MERRILL CORPORATION :
a/k/a ERNIE'S AUTO PARTS :
HENKEL CORPORATION :
Individually and as :
Successor-in-Interest to :
LOCTITE CORPORATION and :
HENKEL LOCTITE CORPORATION :
and DOES I THROUGH 100 :
Inclusive :
Defendants :

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I N D E X

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E X H I B I T S

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EXHIBIT NUMBER	DESCRIPTION	PAGE MARKED
Sarna-1	Invoices of Dr. Sarna	
Sarna-2	E-mail from Mr. Skaar To Dr. Sarna	
Sarna-3		
Sarna-4	Notes and Comments of Dr. Sarna	
Sarna-5	Scientific Articles	
Sarna-6	Article - Chelghoum, Kane	
Sarna-7	Article - Scarselli	
Sarna-8	Articles - Pukkala, Ji, Guo, Costantini, Hotz, McLean	
Sarna-9	Articles - Pedersen-Bjergaard, Mauritzson, Sonali Smith, Martyn Smith, Mitelman, Brownson, Doll, Adami, Thomas, Chelghoum, Kane, Sandler	

Exhibits (continued):

EXHIBIT NUMBER	DESCRIPTION	PAGE MARKED
Sarna-10	Article - Triebig, McLean	
Sarna-11	Article - Wester and Abstract	
Sarna-12	Article - Miller	
Sarna-13	Research Report, Miller Article - Williams	
Sarna-14	Article - Glass - Health Watch Exposure Estimates	
Sarna-15	Article - Glass - Leukemia Risk	
Sarna-16	Article - Hayes	
Sarna-17	Article - Ward	
Sarna-18	Article - Qing Lan, Polymorphisms	
Sarna-19	Article - Qing Lan, Hematotoxicity In Workers Exposed to Low Levels Of Benzene	
Sarna-20	Chemico-Biological Interactions	
Sarna-21	Article - Collins	
Sarna-22	Article, Martyn Smith	
Sarna-23	Legal pad of handwritten notes	

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GREGORY PAUL SARNA, M.D., after having been first duly sworn, was examined and testified as follows:

- - -

BY MR. DuPONT:

Q. Good morning, Doctor. How are you?

A. I'm doing well, thank you.

Q. My name is Andrew DuPont. You understand I'm an attorney for David Gerchman and Theresa Gerchman. Is that correct?

A. I understand that, yes.

Q. All right. And I know you have given depositions before, so I'll briefly review the format that we use here. I don't know if you have done one by phone and a little mobile hook up in the past, but for that reason in particular, if I ask you a question, sir, that you do not understand, will you please let me know that?

A. Yes, I will.

Q. Can we have an agreement that if you do answer a question that I ask you

it's because you have understood the question and you have answered the question that I asked?

A. Well, certainly I'll -- it will be because I believe I understood the question. If I have not understand the question -- understood it, I expect that will become evident subsequently.

Q. All right. If you -- well, I need to understand, as we're going forward today, that if you answer one of my questions it's because you understood it. I won't have the opportunity to re-ask my question based on your claim that you didn't understand it unless you tell me that. Do you understand that?

A. Sure. I will answer your questions as I understand them.

Q. All right. Will you agree to answer my questions here today, as opposed to giving information that I'm not asking for?

A. Oftentimes there are gray zones in terms of answering questions, I try to be complete and educate. I'll ask you to stop

me if you think I'm going off track. But it's usually not black and white when an answer stops and when explaining an answer goes on.

Q. Okay. Have you reviewed your Notice of Deposition?

A. I never got a Notice of Deposition personally.

Q. Okay. Have you brought documents with you here today?

A. I have six boxes of materials and records that I reviewed behind me. I've got a copy of my notes in front of me. I have some copies of the billing e-mails in front of me. And I have a pile of references that are pertinent, although I don't have every reference in my report, nor every reference from your experts' depositions or reports.

Q. Okay. Do you have with you the references that you're relying upon for your opinions in this case?

A. I have many to most of them. There may be other ones that I don't have. I

would not limit myself to these. I generally would say that these reinforce the body of knowledge and will be able to give me precise data in terms of that body of knowledge. But I will be answering based upon my general knowledge, not limited to these reports.

Q. Are you going to be able to identify for me today, whether you have the documents here or not, each study that you relied upon for your opinion?

A. Probably, yes. I'll do my best.

Q. So I understood you to say that you have six boxes of materials that you reviewed. Are those materials that you received from the lawyers for the defendants in this case?

A. Yes. That's what I'm talking about, six boxes of material from the lawyers, which include some disks as well as paper.

Q. Okay. And you have, did I hear you say, e-mails with billing information?

A. I have copies of that, yes.

Q. Did you bring your references?

A. I have my report and most of the references referred to in my report in front of me. And some that may not be in the report that are relevant as well.

Q. Does that cover all the documents you brought here with you today?

A. Pretty much.

Q. Do you have a copy of your CV with you?

A. ^I did not bring a copy of my CV. Certainly that can be e-mailed to you by counsel or by myself.

Q. Okay. I have a copy that was provided to me earlier in the case. The most recent publication I see on it appears to be an abstract, and it looks like it is dated 2011. The primary author was Jahanzeb, ^J-A-H-A-N-Z-E-B?

A. That would be a current CV.

Q. So this CV that was provided to me contains all of the publications that you have authored or co-authored?

A. To my knowledge, yes.

Q. I've seen where you testified before that you have never published a peer-reviewed article or a textbook chapter concerning the causes of leukemia. Is that still correct?

A. I have had peer-reviewed articles on treatment of leukemia from my early days from UCLA, but not on causes of leukemia.

Q. And is it also correct that you have never published on the subject of benzene?

A. It depends, I suppose, upon your definition of published. I presented lectures at the benzene legal conferences and there have been handouts that have material from me. If that's a publication, then that would count. I would not consider that a publication, but I am bringing that up to be complete.

Q. Okay. These were lectures at benzene legal conferences, is that what you said?

A. Yes.

Q. Which conferences were they?

A. They were a series of conferences. One in -- first one maybe seven years ago in, I think, Marina Del Mar. A couple -- one was in Las Vegas. I'm blocking the name of the law firm -- the firm that put on the conferences, but I gave lectures on lymphoma twice, on RARS^ once, and I think MDS another time in general, although I'm not positive of that.

Q. Have you spoken at Defense Research Institute conferences?

A. Defense Research Institute? No.

Q. I would like you to grab your billing e-mails, if you could, and tell me what period of time do they pertain to and what is the total billed amount?

A. Not a billing e-mail, but you probably will ask regardless, so I'll bring it up proactively. The first communication I have regarding this case was on April 26th, in an e-mail from Mr. Skaar, Hogan Lovells, saying that they are including medical

records for my review. And that was based upon a phone call that day, and the medical records were by e-mail.

Then there -- in terms of billing, on May 4th there was a bill for $7,375. On June 9th there was a bill for $3,250. On July 4th, there was a bill for $3,625.

Q. What is your billing rate for the work that's reflected in those invoices?

A. $500 an hour.

Q. What is your rate for deposition testimony?

A. Deposition testimony, at my office in Cedars, is $600 an hour. Away from my office is $1,000 an hour.

Q. And which rate are you charging today?

A. I'm away from my office.

Q. All right. Is that something new?

A. No. That was as of 2009, I believe.

Q. So since 2009, you have charged

$1,000 an hour for a deposition away from your office?

A. It's a hardship for me to leave my office. I can't handle patient materials. I have to cancel patients. I have travel time. So all that is incorporated in the increased rate. Generally people are informed ahead of time of the difference in rate, and if they wish to have it in my office we accommodate.

Q. Okay. But that wasn't my question. My question was, is it your testimony that since 2009 you have charged $1,000 per hour for depositions outside of your office?

A. It's my testimony that I recollect that, but that recollection may be wrong. I don't have the date of the billing sheet in front of me, so that may be an error. But it's certainly been almost for two years.

Q. All right. Let's go to your references. I'm sorry, let's take your three billing e-mails and mark them as Exhibit 1,

please.

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(Whereupon the documents were marked, for identification purposes, as Sarna Exhibit Number 1.)

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BY MR. DuPONT:

Q. And then I would like to take your April 26, 2012, e-mail and mark that as Exhibit 2, please.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 2.)

- - -

BY MR. DuPONT:

Q. Do you call that your engagement e-mail?

MR. THOMPSON: Is that what you would call it, Doctor?

THE WITNESS: Probably not engagement, but good enough.

BY MR. DuPONT:

Q. Do you have a separate

correspondence that's your engagement in this case?

A. No. I just -- the term engagement has many meanings, so I wouldn't have chosen that term, but that's fine.

Q. Do you have any separate correspondence that deals with your being hired to work on this case?

A. No.

Q. Is April 26, 2012 the earliest date of your work on this case?

A. Yes.

Q. Have you ever worked with Mr. Barry Thompson's office before?

A. When he was at Reed Smith, yes.

Q. On how many occasions?

A. Frankly, I don't know. Part of the issue is, often there are multiple defendants who as a group have gone together. And one of the lawyers may be a lead lawyer, but there are many other firms involved and lawyers involved. So I really don't have the answer to that question.

Q. Your prior work with Barry

Thompson and his office, has that been in benzene litigation?

A. Yes.

Q. Am I correct that you never testified on behalf of a plaintiff in benzene litigation?

A. Not in a benzene litigation.

Q. Have you ever testified on behalf of a plaintiff in any toxic tort exposure case?

A. Yes.

Q. Which type of case was that?

A. Richard Burton versus one of the tobacco companies. And I have been retained and not testified in a case regarding contaminated heparin.

Q. I'm sorry, contaminated?

A. Heparin.

Q. Did you provide a causation opinion in the tobacco case?

A. I was a treating physician, but I was also used for expert, and did provide a causation opinion.

Q. Was it a specific causation

opinion? In other words, that your patient's injury was caused by tobacco, or was it a general causation opinion?

A. Both.

Q. Tobacco and how it causes certain conditions?

A. Both specifically and general.

Q. How long have you been testifying as an expert on behalf of defendants in benzene litigation?

A. After the first benzene conference, probably six or seven years ago, I had my first benzene case, I think. So the answer would be six or seven years. That may be wrong, but that would be my best answer at this time.

Q. Do you have a list of your cases that you have testified in with you?

A. No.

Q. Do you have an up-to-date list that you keep at your office or otherwise?

A. No.

Q. I'm sorry, I didn't hear your response.

A. I have a list of depositions and testimony and my cases for that. I don't have a list of cases that I have been retained in or consulted on.

Q. Can you provide that list of depositions and testimony to your counsel, so that we can mark that as Exhibit 3 please?

A. I will provide it at a later date, yes.

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^^(whereupon the document was identified to be marked as Sarna Exhibit Number 3.)

- - -

BY MR. DuPONT:

Q. In how many cases per year have you been retained by defendants in benzene litigation in those six or seven years?

A. I haven't counted. Probably, if I had to make a guesstimate, which is better than a guess, but it's not necessarily correct, it would be somewhere between four and seven a year.

Q. And how much have you earned

per year in your work in benzene exposure cases?

A. I haven't kept track of that.

Q. How much time have you spent on this case from July 4th to the present?

A. May I see my exhibit -- first exhibit of billing records, please?

Q. Yes.

MR. THOMPSON: Andrew, you're talking about since that invoice was issued.

MR. DuPONT: Yes, from July 4th, 2012 to the present.

THE WITNESS: I don't have that figure in front of me. I expect it's at least 25 to 30 hours, and maybe more.

BY MR. DuPONT:

Q. So altogether you're looking at about $30,000 billed from the time you were engaged in this case to the start of your deposition here today?

A. That probably is a fair rough figure. It could be more or less.

Q. Is that an average amount that you bill for your testimony on behalf of defendants in benzene cases?

A. No.

Q. Is it more or less?

A. More.

Q. More?

A. More.

Q. How much have you typically billed per case?

A. There isn't a typical, but some cases are initial consult only, which is $1,500. Some cases are quickly settled and don't require reviewing six boxes of records. Some cases require an extensive detailed report. So it will vary tremendously.

A case that goes to court where I have to testify in trial, and has a 30 or 40 or 50 page report, is going to be a lot more expensive and a lot more work than a case where I give an initial consultation and nothing more. It all ranges between those.

Q. How many toxic tort exposure cases do you handle on behalf of defendants

outside of the benzene litigation realm?

A. I haven't counted them. There have been others. There have been three or four ^hexavalent chromium cases. I can recall a ^creosote case. There was an asbestos case. That's probably five or six. There may be some more, but that's five or six over seven to 10 years.

Q. When did you begin to practice medicine?

A. When did I begin to practice medicine, was that the question?

Q. Yes, sir.

A. 1970 was my internship. So if by practicing medicine you mean internship, the answer is 1970.

Q. So in your 42 years of practicing medicine, have you ever had a patient which you have concluded contracted leukemia as a result of exposure to benzene?

A. I don't recall such a patient.

Q. Have you ever had a patient where you determined that their illness, no matter what the type of illness was, was

caused by exposure to any chemical product?

A. Certainly I have seen patients with mesothelioma and patients with lung cancer, both of whose illnesses were related to chemical products.

Furthermore, leukemics who smoke may have been related to tobacco. Tobacco is, behind chemotherapy and radiation, the number one attributable cause of acute myelogenous leukemia with a relative risk of 1.3 or so. So I have certainly seen heavy smokers who have had AML. And there would have been a statistical possibility that they were caused by chemicals.

Q. When you determined that these individuals with leukemia who had been smokers, that the cause of leukemia was cigarette smoking, did you have any type of quantification of how much of a ^leukemogen they were exposed to?

A. I didn't determine whether it was a cause or not in the individual cases. The amount of exposure would only have been a question of a history of how many pack years

of smoking.

Q. Have you ever had a patient that you concluded they had a disease that was caused by exposure to solvents?

A. I can't recall such.

Q. Have you ever conducted any studies concerning the ability of benzene to cause leukemia or any other hematopoietic disease?

A. No.

Q. Have you ever been involved in any epidemiology study?

A. I can't recall publishing one as a first author. And if you mean -- well, the answer to your question is no. Certainly I have written chapters in books on a variety of diseases where epidemiology is discussed. So I have written on epidemiology, but I have not been involved in a study.

Q. Have you written on the epidemiology of benzene?

A. Generally one would talk about the epidemiology of a disease rather than the epidemiology of a chemical compound, but

taking the gist of your question and answering that, the answer is, I have not published on benzene's role as a leukemogen or carcinogen or inducer of other malignancies.

Q. Are there oncology and hematology textbooks that you believe are authoritative in your field?

A. Authoritative is a broad word. Certainly the DaVita^ textbook is the oncology textbook I would tend to use. And there are a variety of hematologic textbooks that are used.

The issue of authoritative is a relative issue. In most chapters there are things that may be clearly true and there may be things that may be authors' opinions or nebulous types of statements, particularly dealing with epidemiology. So I do not in general go to the textbooks of hematology and oncology to answer epidemiology questions. They're often useful to answer therapeutic -- give information on therapeutic issues, but even then the time --

Q. Doctor, my question is, are there any hematology textbooks that you consider authoritative?

A. For what purpose?

Q. For any purpose.

A. Yes. ^daVita's Oncology textbook is one that's authoritative for guiding clinical practice. In hematology, probably Williams and ^Winthrop are useful textbooks.

Q. I have been provided with a 25 page report. Would you call this your -- a 25 page document?

A. It's a document. I wouldn't call it a report, although it has aspects of a report. If it were a report, it would have been polished. It's not polished. It's a stream of --

Q. What can we refer to this as?

A. Notes with comments.

Q. Okay. Let's mark your notes with comments as Exhibit 4, please.

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(Whereupon the document was

marked, for identification purposes, as Sarna Exhibit Number 4.)

- - -

BY MR. DuPONT:

Q. Have you ever been hired to consult on issues concerning benzene causing disease outside of the context of defending defendants in litigation?

MR. THOMPSON: Do you understand the question?

THE WITNESS: To my knowledge -- may I have the question again, please?

BY MR. DuPONT:

Q. Have you ever been hired to consult on the issue of benzene causing disease outside the context of defending corporations in benzene litigation?

MR. THOMPSON: If you agree with that characterization.

THE WITNESS: Yes. I have been hired for -- on Workmen's Comp cases regarding benzene causing disease, which is not the same as the -- defending a corporation. But in

principle it's the issue of whether benzene caused the disease. So the strict answer to your question is, yes. And the explanation is in Workers' Compensation cases.

- - -

(Discussion held off the record.)

- - -

BY MR. DuPONT:

Q. Doctor, you were discussing Workers' Compensation being your total Workers' Compensation matters. Was that always on behalf of the defendants in the Workers' Compensation matters?

A. Yes.

Q. Have you ever been retained by a government agency or a non-government agency, such as the World Health Organization or IARC, to consult on matters of a chemical causing a disease?

A. Yes.

Q. Yes?

A. Yes.

Q. In what respect?

A. The United States government asked me to consult on an issue of chemical exposure at Camp ^LeJeune and I did so.

Q. And was the United States government a defendant in that litigation concerning chemical exposures at Camp LeJeune?

A. I don't recall whether they were a defendant or a potential defendant. I consulted, I did not testify or provide further information after consulting.

Q. When did that occur?

MR. THOMPSON: Did you say when?

MR. DuPONT: Yes.

THE WITNESS: Probably four years ago, but I may be off.

BY MR. DuPONT:

Q. Is that the only work you have done on behalf of a government agency or a non-government agency that relates to the issue of chemical exposure causing a disease?

A. To my knowledge, that's the only government agency that I have worked

with in such a matter.

Q. Was it the Department of Defense that hired you?

A. No.

Q. Which agency?

A. I'm not sure, but it wasn't the Department of Defense.

Q. Do you have a file for your correspondence in this case?

A. The billing information I have given you. There have been a bunch of e-mails saying, here are records, which I have not given you.

Q. Do you have a list of all documents you have received and reviewed in this case?

A. No.

Q. How many articles have you brought with you today?

A. Would you like me to count them?

MR. THOMPSON: Maybe you can estimate.

THE WITNESS: I'll count. A

quick estimate is 44, plus there are some other things that are not articles that are relevant. One is a little thing on benzene toxicity standards and regulations from ATSDR. One is the letter that came out of Dr. Shadduck's deposition from Shell. Another is Dr. Harrison's addendum to his deposition or report, whatever it was, review of records and opinions.

BY MR. DuPONT:

Q. Okay. Are you qualified to provide an opinion regarding industrial hygiene?

A. Can you define industrial hygiene?

Q. Do you consider yourself an industrial hygienist?

A. No.

Q. Did you hear my question?

A. Yes. My answer was no.

Q. I'm sorry, I didn't hear your response.

A. I'm sorry, I mumble at times.

Q. Are you qualified to provide an opinion as an industrial hygienist?

A. Again, I need a definition as to what sort of opinion. There are overlaps in areas between a variety of disciplines. And to the degree that they overlap in my area of expertise the answer is, yes. But I'm not making estimates of exposure, if that's what you mean. Quantitative experts -- I'm not making quantitative estimates of exposure. Qualitative, yes.

Q. Okay. So to make sure I understand, you're not qualified to provide a quantitative exposure assessment?

A. Correct.

Q. Correct. Do you feel that you could provide a qualitative exposure assessment?

A. Yes.

Q. Do you feel that an oncologist/hematologist can make an opinion on benzene causing a disease, including AML, make qualitative exposure assessment?

A. I think that's one portion --

piece of information that goes into a conclusion or decision. I don't think it's the sum of information or the totality. I think that it's part of the equation.

Q. If all you had was a qualitative exposure assessment, could you provide an opinion regarding benzene causing a particular disease, including AML?

A. If that qualitative exposure is minuscule, then the answer is yes.

Q. What do you consider minuscule?

A. That's a complex answer. I'm not going to give you just a simple answer, it will require discussion. Do you want that?

Q. How do you define it? Do you define it by years, do you define it -- how do you qualitatively define minuscule exposure?

A. Both in terms of the cumulative exposure and in terms of the exposure on a daily basis. Particularly relevant to me is the exposure over the preceding ten years.

Q. How do you used the term

cumulative exposure on a qualitative basis?

A. You say there was a lot, there was a little, there was minuscule, there was something intermediate. You say that it is contaminant level only, or you say that it is more than that. You say that it is something that according to the literature had very low exposure, or something that according to the literature has had high exposure. So those are the types of things that one could do qualitatively.

Q. When you say according to the literature, do you feel that you're qualified as a hematologist to look at the literature on benzene exposed workers and compare that to a worker and determine whether or not their exposure was low or high?

A. The literature I'm familiar with is both from the point of view of measuring exposure, and I quote for you the Scarselli and Williams articles as to moderate exposure. And the literature as to reverse engineering, the issue of exposure by whether or not there is an increased risk of

leukemia in that profession. And I would believe that I am qualified as a hematologist/oncologist to look at that literature and come to a conclusion.

Q. What's your opinion as to the specific form of leukemia David Gerchman has been diagnosed with?

A. Let me find my notes. The broad diagnosis would be AML, or acute myelogenous leukemia. The more specific by FAB^ classification would M5B monocytic leukemia. By World Health criteria it would be AML not otherwise specified, subgroup acute monoblastic and monocytic leukemia.

Q. Have you derived any opinions on David Gerchman's life expectancy considering his diagnosis with AML and his medical course of treatment and progression of his AML?

A. Yes.

Q. I'm sorry?

A. Yes.

Q. What is your opinion?

A. If one puts aside his current

condition, which I do not have up-to-date information on, but if one puts that issue aside and just deals with the issue of prognosis for someone who has had a second stem cell transplant from an unrelated donor for acute myelogenous leukemia that has relapsed after a first transplant, the figures that his physicians support him have been a 20 to 30 percent success rate and a comparable risk of dying due to the procedure, and I would agree with those figures.

The information I most recently had was that he was critically ill with complications of severe graft versus host disease, and that there was some concern about whether he would survive the hospitalization. If he has rallied and is much better, that is one thing. If he has continued critically ill, that is another. Based upon the information I would have, my expectation is that he would not survive and not be cured. But certainly there would be the possibility that he's improved, and both

the reality and my expectation would change if I had such data.

Q. Do you have any opinions concerning the appropriateness of the treatment that was provided to David Gerchman for his AML?

A. I'm sorry, was the question opinion or concern?

Q. Do you have any opinion?

A. I think he was well treated.

Q. Do you have any concern?

A. No.

Q. I'm sorry, I did not hear your response.

A. No, I have no concern. I believe his treatment has been excellent.

Q. Okay. Do you have any opinion as to what David Gerchman's life expectancy was prior to the time he was diagnosed with AML?

A. Not a formal opinion.

Q. Do you have an opinion?

A. He had obesity, which had improved somewhat with treatment for obesity.

He had psoriasis. He was otherwise reasonably healthy. He was at the time of the diagnosis, I think, 51, if I recall correctly. And the average life expectancy for someone in good health at 51 is probably to about 85. Morbid obesity would probably lower that to about 75 to 80. He had a history of smoking, but it was distant. So probably if I had to opine at this point, off the top of my head, it would have been a 25 to 30 year estimate of survival.

Q. Okay. Have you reviewed any medical bills for David Gerchman's treatment?

A. I believe that I was sent in the six boxes medical bills, but I frankly did not review them.

Q. So you have no opinion as to the reasonable and appropriate nature of those charges?

A. I have an opinion, even though I have not reviewed them.

MR. THOMPSON: Let's not put your opinions out there on things you have noT reviewed, Greg.

BY MR. DuPONT:

Q. What's your opinion?

A. That medical care in the United States is too expensive.

Q. Do you have an opinion as to the reasonable and appropriate nature of the charges for David Gerchman's medical care?

A. I have the opinion that his care was reasonable and appropriate. The charges I have not looked at, other than giving you my general statement about the cost of healthcare in the United States.

Q. All right. So let's turn to what your opinions are in this case.

Are all of your opinions contained within your notes and observations that were marked as Exhibit 4?

MR. THOMPSON: Notes and comments.

MR. DuPONT: Notes and comments.

THE WITNESS: I expect that they are. I will give you the caveat that there's material that I have not seen yet in terms of other people's reports

or depositions. And they may have opinions that come as a result of doing -- reviewing that material. So if there is given further material, either in terms of Mr. Gerchman's clinical course or other people's opinions or other people's reports or other people's depositions, that may require new work and may or may not evoke new opinions.

BY MR. DuPONT:

Q. Where in your notes and comments are your opinions set forth?

A. They're sort of scattered, but let me say on page two there are opinions on risk factors for AML. That's page two to three. On page seven there's some comments on exposure. And on seven, eight there's comments on the risk of AML and leukemia in general in mechanics. And on page nine there's some information or discussion in terms of chromosomal abnormalities. And there's a summary of opinions on page 10. Then there are other opinions buried in

review of Dr. Shadduck's deposition and Dr. Harrison's handout. And also there's some comments on Dr. Petty's and Dr. Williams' assessments of exposure.

Q. What is your experience in industrial hygiene?

A. I am not an industrial hygienist.

Q. So do you have no experience in industrial hygiene?

A. Hands-on experience, no.

Q. Any experience whatsoever, not limited to hands-on experience.

A. No.

Q. All right. Do you have a pen there?

A. Hold on. Yes, I do.

Q. I would like to number your own opinions so that I'm not guessing on what is and is not an opinion. So let's turn to page two of the first page you indicated where you have an opinion.

MR. THOMPSON: Andrew, Andrew, I don't think that's going to cut it.

Because as he's said, he's got opinions scattered throughout the thing. So I'm certainly not going to want to limit him to only the ones he just identified in a cursory manner by leafing through the thing. I mean, we can use that as a starting point. But I'm just telling you now, this is the -- as with the other experts, those who have provided some kind of report or notes or whatever, like Harrison did, you know we had to go through and kind of suss^ it out, and the same is probably going to be the case here.

BY MR. DuPONT:

Q. All right. Well, let's start on the first page. Do you have any opinions on page one?

A. Page one is limited to the clinical summary of what is going on. So there are no opinions as to causation on page one.

There is a statement relevant to causation that his BMI when he weighed

290 pounds was 41.6, which would be morbidly obese. And that on March 7th, which was -- March 7, 2011, he weighed 220 pounds and was still obese. Although with his bariatric surgery he had lost 70 pounds over two years. But he still was obese.

Q. Let me stop you there. Why don't you point out for me in your report the opinions that you hold that are relative to causation?

MR. THOMPSON: Take your time, Greg.

THE WITNESS: I believe that obesity was a risk factor for him. I do not believe that it was a direct cause of his leukemia. But I quoted data, two ^META analyses, one by Larson and one by ^Castillo, both of which show a risk of about 1.5 for AML with obesity. So I believe that that belongs in the discussion of causation as a risk factor.

I also discussed the smoking issue. I discussed that smoking, as I

mentioned before in my deposition, is a risk factor for leukemia. But I also said that given the lag time from when he stopped smoking, I did not believe it was a cause of Mr. Gerchman's leukemia.

BY MR. DuPONT:

Q. Okay. Is your obesity opinion set for on a particular page in your report?

A. Well, part of it is on page two and there are parts elsewhere. Again, it's not a report.

Q. All right.

A. In terms of the issues of psoriasis, I have quoted data saying that it is a risk factor. I do not consider it a cause. Whether it is a risk factor, here it is based on much less robust data than obesity or tobacco. Here I just had two case control studies with non-significant increase. So I would not find that compelling, but as part of the discussion.

I also discussed his age. His age is not the average age of someone with

AML. But a third of the patients are less than 54 years of age at diagnosis by ^SEIR data, so it is certainly quite consistent with de novo AML. De novo meaning without an identifiable cause.

Then relevant to my opinion is, I discussed on page seven that published data on auto mechanics do not show high benzene exposure. Putting aside the experts' opinions in this case, talking about auto mechanics in general, Scarselli from Italy published a geometric mean exposure of 0.02 ppm benzene with an arithmatic mean of 0.05 ppm, based upon 234 measurements of time weighted average exposure.

And Williams, which I didn't quote in my report -- which I did quote in my report, found TWAs, time weighted averages in the breathing zone ranging from 0.01 to 0.3 with a variety of products that are present since the seventies, late seventies; paint solvents, printing solvents, inks, cutting and coating oils, adhesives, mineral spirits, degreasers and jet fuel. So that to me is a

relevant issue in terms of the discussion of causation.

Q. What is your next opinion?

A. The next opinion is that literature on mechanics show they do not have an increased risk of leukemia and AML in specifically. And I start with quoting the review from 1997 by ^Houtz and Lowries. They found 17 studies of AML at that time with data from 1955 to 1991. The overall pattern was negative. There were occasional studies which suggest --

Q. Doctor, let me cut you off. I would like to review this in a little different way by getting your opinions first and then we'll go through the basis for them. Okay?

A. Okay. Then do you want me to tell you the other articles that support the premise that mechanics in the reasonable modern day are not at increased risk of leukemia, or do you want to delay that till later.

Q. We will get to that, but the

way I'm trying to organize this, I would like to first ask what your opinions are.

MR. THOMPSON: So he wants to round up all your opinions and then he'll go back.

Andrew, is that what you have in mind?

MR. DuPONT: I want the list of opinions and then we'll go through the basis for each of them.

THE WITNESS: Yes. So that broad opinion is, mechanics in the modern era are not at increased risk for AML.

The next would be the presence of the normal karyotype and the absence of a karyotype which is typical of benzene argues against AML being caused by benzene.

Somewhere in here, and I'm not sure where, would be the issue of what I consider latency, but it -- the word latency is used in different ways in the literature, and I'm not defining

latency from the time of first exposure to the time of development of leukemia. In the first place, that presupposes that exposure was the cause. And then it presupposes that if exposure were a cause, that it was caused by the first drop of exposure on the first day, both of which are premises that I do not accept.

I would define latency, or one may use the term lag time, as the time from the causative occurrence of leukemia till it is diagnosed. And typically --

BY MR. DuPONT:

Q. Doctor --

A. I'm sorry.

Q. What is that time period, in your opinion?

A. Based -- well, I won't give you what it's based upon, I'll just give you the opinion. The average would be four to five years, and generally it would be 10 years or less.

So the opinion would be, in terms of Mr. Gerchman, that his leukemia arose within the ten years -- likely within the ten year frame prior to his diagnosis.

Now, I say arose rather than use the word caused because it is not necessary for there to be an external cause of malignancy. We have spontaneous mutations all the time, which certainly can cause malignancy. And so I would not say -- use the term when the leukemia was caused. I would use the term when the leukemia arose genetically.

Q. All right. What is your next opinion?

MR. THOMPSON: Keep looking.

THE WITNESS: In terms of broad opinions on causation that is all that comes to mind. However, I do not want to be limited to this. My report should stand by itself, or my notes should stand by themselves. And if I -- there's a lot of information in here. And some information that

doesn't fit under the umbrella as I see it may still be relevant. So I do not want to be restricted to only what I just listed as opposed to what's in my notes.

BY MR. DuPONT:

Q. Well, Doctor, I'm not going to guess as to what your opinions are going to be at the time of trial, and this is my opportunity to ask you what your opinions are. So if you have any opinions beyond those, I think you've listed eight opinions, could you tell me what they are?

MR. THOMPSON: Greg, I think the only safe way to do this is for you to literally go through and see where you have an opinion beyond -- described beyond what we have already talked about. And if it takes a while, it's okay. Go through each page and see if there's anything you can add to these eight -- I actually have nine, but it kind of depends on how you write them down.

So why don't we -- if you don't mind, Andrew, why don't we go off the record, and he can look at his report and see if there's anything else. And then he can update that list and let you know if there's anything more. Is that okay with you?

MR. DuPONT: Well, I would prefer to stay on the record.

THE WITNESS: Well, could you read back for me, or can the reporter back to me what the list you have of my opinions is?

BY MR. DuPONT:

Q. Well, I'll read what the list is and then you can agree with me or not.

Number one, obesity, a risk factor, but not a cause.

A. Go ahead.

Q. Agreed?

A. Yes.

Q. Do you agree with that?

A. Yes.

Q. Number two, smoking, a risk

factor, but not a cause of David Gerchman's AML. Do you agree with that?

A. Yes. It's a cause of leukemia in some cases, but not in this case.

Q. Okay. That's your second opinion. Third opinion, psoriasis is a risk factor for leukemia, but not a cause of David Gerchman's AML?

A. Yes.

Q. Number four, David Gerchman's age was not average. And by that I assume you mean he was below average for the age of being diagnosed with AML. And one-third of the AMLs reported in the SEIR data are below the age of 54?

A. Yes. So it's not average, but it's well consistent with de novo AML. That should be clear. But my point is that there's nothing unusual about this age, even though it's not average.

Q. Okay. And you agree that it's below average, David Gerchman's age of diagnosis with AML?

A. Very few people are going to

have the average. Half will be above and half will be below. It's a median rather than an average. And use the average --

Q. Doctor, I asked you, was David Gerchman's age at diagnosis below average?

MR. THOMPSON: Andrew, just let him finish his answer, okay? And, Greg, insist that you finish your answer.

MR. DuPONT: Well, the Doctor told me to cut him off if he was rambling.

MR. THOMPSON: Well, I think he had a different view of that than you did.

BY MR. DuPONT:

Q. Okay. Fifth opinion, published data on automotive mechanics in the modern era do not show high benzene exposures. Agreed?

A. Yes.

Q. That's your fifth opinion?

A. Yes.

Q. Sixth opinion, the literature

on mechanics do not show in the -- in the modern era do not show an increased risk of leukemia or AML?

A. I'm going to clarify that. Not only do they not show an increased risk, they show there is no increased risk.

Q. Okay. Your seventh opinion is that the normal karyotype and absence of a typical karyotype found in David Gerchman argues against benzene as a cause of his AML?

A. Yes.

Q. Your eighth opinion is that the -- on the subject of what some folks refer to as latency, but you used a different term. You would expect that the diagnosis of leukemia would come within on average four to five years and generally less than 10 years of the date that the leukemia -- the date of the causative event?

A. The date that the leukemia arose with or without an extrinsic cause.

Q. Okay. So from point A, leukemia arose, to point B, AML diagnosis occurs within 10 years, average four to

five years?

A. Yes.

Q. Opinion number nine -- well, that was -- okay. So that was your eighth opinion. Okay?

A. Yes.

Q. And those were the opinions that you have given me so far; correct?

A. Yes. Now if I may take some time to look through this to see if there's anything else that should be added.

MR. THOMPSON: You left out his reference to spontaneous mutations. He's going to be offering an opinion on that too.

MR. DuPONT: Well, okay, if that's your opinion, or the opinion you want him to add, okay, but he didn't say that.

MR. THOMPSON: Yeah, he said that. If you want to look back on the transcript we can do that. But he is going to comment on whether spontaneous mutation --

MR. DuPONT: I would like to have the doctor provide his opinions.

MR. THOMPSON: He already did provide that opinion. You may disagree or maybe you didn't write it down, but he did provide that opinion.

THE WITNESS: You may not have caught it, but when I talked about the lag time I said -- took care to say whether the leukemia arose rather than was caused. And I talked about spontaneous mutations happening all the time and being a cause of malignancy without an external source.

BY MR. DuPONT:

Q. Okay. So do you have a ninth opinion that AML can be caused by spontaneously, or mutations, AML mutations?

A. Spontaneous mutation, yes.

Q. Now, you're going to look through your report and tell me what your 10th opinion is.

A. Will do.

One of the issues that may not

have been implicit in the previous relative to Mr. Gerchman was that his exposure was qualitatively small over the ten years preceding his diagnosis.

Another opinion that may not have been clear is the issue of looking at part per million year to try to determine which group of benzene exposed people are at increased risk of leukemia. And one uses that metric to determine risk. I would say 40 to 60 part per million year would be the low range for an increased risk.

The corollary to that statement would be that low dose exposure in the two part per million year range is not an increased -- does not confer an increased risk for leukemia de novo.

And then a minor point, but one that would refer to Dr. Shadduck's opinions, I would say that the fact that Mr. Gerchman's sister did not have leukemia is irrelevant.

I'm hoping I'm not missing anything. Again, my notes should stand for themselves, but that's what I find in terms

of opinions.

MR. THOMPSON: Well, I'm going to add a couple of topic areas that we're going to ask you about because this is kind of a weird process.

You're going to be asked whether you agree or disagree with the general and specific causation opinions of Dr. Shadduck and Harrison. So now is the time that you -- if Andrew wants to ask you about that, he should ask you about that.

We're also going to ask if you have an opinion on whether the dose that Mr. Gerchman had was sufficient to induce AML and whether it did in him. And whether you believe this was a primary or secondary de novo or secondary AML in Mr. Gerchman and whether you have an opinion concerning --

MR. DuPONT: Well --

MR. THOMPSON: Hey, I'm just telling you, these are topic areas

he's going to be testifying about. You asked what he's going to testify about and I'm telling you.

MR. DuPONT: No. I asked what his opinions are.

MR. THOMPSON: Excuse me?

MR. DuPONT: I asked what his opinions are.

MR. THOMPSON: Right.

MR. DuPONT: And if he's told me what his opinions are, then I need to know that that's what his opinions are.

MR. THOMPSON: Then ask him if he has opinions on those topics because he's going to be asked those questions. I'm telling you, at the time of trial he's going to be asked those questions and he's prepared to offer opinions on those things, all of which are touched on in his report, albeit he didn't spot them in the 10 minutes he had to review his 26 page report just now. All of those things

are in there. So we told you at the start that his report contains all the information and opinions, either direct or indirect, and they're all in there. And I'm telling you now, putting you on notice, those are going to be topic areas he's going to cover.

So feel free to ask about them or not, this is your shot.

BY MR. DuPONT:

Q. Doctor, do you have an opinion after what you said regarding David Gerchman's sister not having leukemia being irrelevant?

A. I'm sorry, you want me to elaborate on that statement?

Q. No. Do you have an opinion, in addition to the opinions that you listed for me?

A. I have opinions on all of the issues that Mr. Thompson raised.

Q. Okay. What is your next opinion then after David Gerchman's sister not having leukemia is irrelevant?

MR. THOMPSON: Let's go ahead and have my response read back so that Greg can tick those off and address each one of those.

THE WITNESS: One by one, please.

MR. DuPONT: Okay. So we'll start with the lawyer opinions that you're going to address?

MR. THOMPSON: Excuse me? Don't answer that question, Greg. Greg, don't answer that question. You can re-ask your question if you want and he'll be happy to answer a proper question, but don't answer that, Greg.

BY MR. DuPONT:

Q. Okay.

MR. THOMPSON: Andrew, I know you're not a California lawyer, and you're getting to know the system and you're a smart guy, you'll get it, but the purpose of the deposition in California is for him to disclose the topic areas and his opinions. We

don't have reports out here, he told you his notes --

MR. DuPONT: I understand that and I thank you for your discussion. However --

MR. THOMPSON: Don't suggest that those are lawyer opinions. Those are topic areas that he's going to talk about.

BY MR. DuPONT:

Q. ^okay. What are the topic areas that your counsel has indicated he wants you to discuss that are not your opinions here that you have listed so far?

MR. THOMPSON: Don't answer that, Greg. Don't answer that question. Go ahead ask a question. And he'll -- if you give him a proper question, he'll give you an answer.

BY MR. DuPONT:

Q. Have you listed all the opinions you have?

A. I have opinions about the issues that Mr. Thompson has raised. My

expectation is, should this come to trial, I will be asked a variety of questions by both yourself and Mr. Thompson, and unless there is an objection to my answering the question which is sustained, I will answer those questions. The questions which he's raised are relevant and I have opinions. Those opinions are intrinsic to some of the opinions and overlapping the opinions I've --

Q. What are those opinions?

A. I would like a list one by one of Mr. Thompson's comments, and I will give you the opinions on those issues.

Q. Okay. What is the list of Mr. Thompson's comments?

A. Can the court reporter give us back?

- - -

(Whereupon the court reporter read back the pertinent testimony.)

- - -

THE WITNESS: The first one was whether I agree with the general and specific causation of Dr. Shadduck and

Dr. Harrison. And my answer is that I do not believe in the general causation belief that low level exposures of benzene is the cause of leukemia. And I do not believe in the specific causation that Mr. Gerchman's cause -- leukemia was caused by benzene exposure.

BY MR. DuPONT:

Q. Okay. What is the next topic that your attorney wants you to address?

MR. THOMPSON: I can just tell you if you want me to. He's going to be asked to address whether or not he thinks the dose was sufficient in this case to induce AML, and whether it did in this person, Mr. Gerchman.

THE WITNESS: My belief is the dose was not sufficient. As a matter of conclusion based upon other factors, the dose did not induce AML in Mr. Gerchman.

BY MR. DuPONT:

Q. Okay. Are we done with your

opinions?

MR. THOMPSON: No, we're not.

THE WITNESS: Third issue?

MR. THOMPSON: You'll be asked to describe and comment on de novo AML generally and specifically.

THE WITNESS: De novo AML makes up 80 percent of AML. It is usually a normal ^karyotype, but not always. It can arise as a spontaneous event. It has been postulated by one author that there are 10 to the 27th, which is a huge number, spontaneous mutations that occur in the lifetime. And that spontaneous mutations cause far more cancers than external events causing mutations. And it should be no surprise that we have spontaneous mutations as they are a normal part of evolution. They're the reason we are not amoebae or chimaera^, because of evolution. So these things occur and are a common cause of malignancy.

MR. THOMPSON: And you will also

be asked whether that can be ruled in or ruled out in Mr. Gerchman's case.

THE WITNESS: You can't rule it in or rule it out specifically.

MR. THOMPSON: Okay.

MR. DuPONT: Now are we done?

MR. THOMPSON: That's all I can think of right now. If I come up with any other issues, I'll let you know though before the deposition is done.

MR. DuPONT: Gee, thanks.

MR. CARR: And, for the record, if it's in his report it's fair game in our views.

MR. THOMPSON: Right.

MR. DuPONT: I would like to know what his opinions are because something can be in the report and not be an opinion.

MR. CARR: Let me restate my comment. If it's in his report and admissible, we're reserving the right to have him talk about it at trial.

MR. THOMPSON: Correct.

MR. DuPONT: Okay.

BY MR. DuPONT:

Q. In terms of your opinions, have we exhausted the list of your opinions in this case?

A. To my knowledge, which is sometimes imperfect, yes.

Q. All right. Let's start from the top. Opinion number one, obesity is a risk factor, but did not cause David Gerchman's AML. You gave me two articles, Larson and Castillo, that you cited as supporting your opinion. And you said that they indicate that there is a 1.5 risk of leukemia from obesity. Is that correct?

A. Yes. Those are both two different META analyses; one published 2008, one published electronically this year.

Q. Which was 2008 and which was this year?

A. ^Larson was in the International Journal of Cancer, volume 122 pages 1418 through 1421, 2008. Castillo --

Q. So Larson was 2008. And

Castillo was 2011.

A. 2012.

Q. 2012. Is the cite for Castillo contained in your report, or your notes and comments?

A. The DOI is listed, and it's available on the Internet. And it may or may not be in the pile I have here.

Q. Okay. Are there any other pieces of literature, publications, that you rely upon for opinion one, obesity is a risk factor, but not the cause of David Gerchman's AML?

A. Those are the two that I pulled because they're META analyses, they have more weight than individual studies. There certainly are other studies that are listed in those META analyses, so this is not the extent of the world's literature on that issue. But I believe that one can get a reasonable estimate of what the literature says by looking at META analyses. And when two of them give the same result, I expect that that tells you something.

MR. THOMPSON: Andrew, just to clarify, Dr. Sarna will be asked whether he can rule in or out obesity as a contributing factor relative to Mr. Gerchman.

MR. DuPONT: You know, I have a concern that the doctor has stated his opinion, and now there are comments being made by counsel that are intending to change that opinion. And I don't think that's proper.

BY MR. DuPONT:

Q. So, Doctor, do you have the Larson and Castillo articles that are your literature references for your opinions that obesity is a risk factor, but was not a cause of David Gerchman's AML?

A. I'm sorry, what is the question?

Q. Do you have with you your articles that you rely upon as your literature references for your opinion that obesity is a risk factor, but not the cause of David Gerchman's AML?

A. I'll need to look through the pile to answer that question.

Yes.

Q. Let's mark those as the next exhibit, which I believe to be Exhibit 5.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 5.)

- - -

BY MR. DuPONT:

Q. Okay. Two, smoking is a risk factor, but did not cause David Gerchman's AML. What literature references did you rely upon for that?

A. I'll need my report back, hold on. There's one listed in my report and there are three others that I brought with me.

The one listed in my report is by Doll, D-O-L-L, 2005. I have also brought a report by ^Brownson. And a report by ^Adame. And a report by Thomas. These are all pertinent. The Adame one is not --

Q. The Adame one is not?

A. Just a second. I have to look at that.

The Adame one is not a causative study. The others I believe are.

Q. Okay. Are those all of the literature references that you rely upon for your opinion that smoking is a risk factor, but did not cause David Gerchman's AML?

A. There are other articles that talked about the risk disappearing later on. And that's why I say that it was not a cause, in addition to my opinion on lag time. I'm not sure what those other articles are, but I've seen them.

Q. Okay. Can you cite for me any articles, other than the four that you listed?

A. Let me see whether I have it with me or not, or whether it's cited in this. Hold on.

MR. THOMPSON: While he's looking at that, Andrew, have you paid any our experts yet?

MR. DuPONT: We are getting those checks ready and we're sending them out.

MR. THOMPSON: When are they going to be here? Because these guys are supposed to be paid on the spot, frankly. Like I did with your expert, Dr. Shadduck and like we did with Dr. Harrison.

MR. DuPONT: I understand that, I apologize.

THE WITNESS: Okay. I've got three other articles, although one of them might be a duplicate. Let me see whether it's a duplicate or not. No. An article by ^King. An article by Sandler. An article that's in the pile, but not relevant because it really talks about course of AML, not the incidence. But it's in the pile, by ^Chelghoum.

So do you want all of these wrapped into an exhibit, or no?

BY MR. DuPONT:

Q. Yes, I would like to make that our next exhibit. Thank you.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 6.)

- - -

BY MR. DuPONT:

Q. Do you have an opinion on what the relevant risk of AML specifically from smoking is?

A. Yes. Approximately 1.3.

Q. Three, psoriasis is a risk factor, but was not a cause of David Gerchman's AML. What is your basis for that opinion?

A. Well, I quoted in my notes the article by ^Cooper and Soderburg, and there was also an article I think by Zheng, if I'm remembering correctly, that either Dr. Shadduck or Dr. Harrison provided. Probably Dr. Shadduck, but let me see what I have here.

MR. THOMPSON: Andrew, this is

the last time I'll bring it up, but when can we expect those checks for the three experts?

MR. DuPONT: I don't have them in my hand right now, but I will confer with our accounting department.

MR. THOMPSON: Because I don't think we're going to be able to produce any more experts until we get these three paid, because they're supposed to be paid.

THE WITNESS: I don't have those articles with me. Certainly the references to the two that I noted from Cooper and Soderburg are in my notes. And I'll try to find you in a moment which was the article quoted by Dr. Shadduck that Dr. Harrison -- Dr. Shadduck quoted an article by Zheng, Z-H-E-N-G, which I believe is in Cancer Causes Control, Volume Four, page 361, 1993, which showed an odds ratio for leukemia in Chinese people with psoriasis of 5.5, based upon 10

cases. That's your expert's article. I expect he could provide it for you, but that's the reference.

BY MR. DuPONT:

Q. All right. Are those the two references that you have for the issue of psoriasis being a risk factor, but did not cause David Gerchman's AML?

A. In the primary portion of my report, meaning page two, I quote Cooper and Soderburg. On page 19 I refer to Zheng.

Q. So those are the two articles that you have cited for me --

A. There are three articles that I just mentioned.

Q. Okay. So Cooper and Soderburg are two separate articles?

A. Correct.

Q. Do you have Cooper and Soderburg with you here today?

A. No.

Q. Do you have any of the psoriasis articles with you today?

A. None of those three. I have

other psoriasis articles which deal with the issue of barrier and absorption. Which adds another opinion. That is, the presence of psoriasis did not increase dermal absorption of benzene. And that -- that will be enough for that.

Q. All right. Is it your opinion that psoriasis is a risk factor for AML specifically, or the general category of leukemia?

A. It's my opinion that it may be a risk factor for AML. I would not go so strongly as to say it is a risk factor for AML.

Q. Is it your opinion that psoriasis is a risk factor of leukemia or it may be a risk factor for leukemia?

A. May be.

Q. And when you say it may be a risk factor for leukemia, AML, are you indicating that you do not have any degree of confidence that, in fact, it is a risk factor?

A. There are two issues. One is

do people with psoriasis have an increased risk of AML? And as for that, my answer is there are the three studies that are quoted, but they're not sufficiently robust to make me conclude that it's real. It could be real.

The second is, assuming that it is real, which it may or may not be, does that mean that the psoriasis is the cause, because these people may have been treated with Imuran^ or ^methotrexate, which could have been the cause. Or in modern day, with TNF inhibitors which could have been a cause rather than the psoriasis. In which case psoriasis is a risk factor, but the cause was the therapy that was given.

Q. At this point you don't know whether it's the medication taken by the subject in those three studies or the psoriasis in and of itself that resulted in the increased risk of psoriasis that was found -- I'm sorry, the increased risk of AML that was found?

A. The data are too scanty for me

to reach a firm conclusion about whether people with psoriasis are at risk for AML. They may be, based upon the articles that have been quoted. If they are, which may or may not be the case, that may be related to methotrexate, Imuran or other types of issues, and that's not analyzed in these studies.

Q. But as we sit here today, you're not able to say whether the psoriasis in and of itself or the medication that resulted in increased risk in those three studies?

A. In those three studies, I'm not able to tell you that.

Q. Four, David Gerchman's age at diagnosis with AML was below average, but in your opinion was consistent with de novo AML?

MR. THOMPSON: Just make sure, Greg, that these are the opinions that you have.

THE WITNESS: Yes. Do you want me to expand on that?

BY MR. DuPONT:

Q. I'm going to ask you some questions about that. First of all, what do define de novo AML to be?

A. De novo is AML is AML without an appreciable cause. 80 percent of AML in the United States or so is de novo. The next major cause would be chemotherapy and radiation.

MR. THOMPSON: Just answer his question as to de novo AML, Greg.

THE WITNESS: Okay.

BY MR. DuPONT:

Q. Your definition of de novo AML is AML without an appreciable cause?

A. Correct.

Q. What do you mean by an appreciable cause?

A. Without a cause that can be identified as a cause. Let me try to clarify that. There are people who are exposed to benzene. Everyone in this room is exposed to benzene. If you pump gas you have had higher exposure than just by sitting in this room. If there's not adequate exposure and not a

pattern of chromosomes that suggest benzene, no reason to think that benzene is in the discussion of such a situation for sitting in this room or pumping gasoline for your car. So there's no appreciable cause for leukemia in that setting and that would be considered de novo.

I think Martyn Smith, who is often a plaintiff expert and has written a variety of things, blaming benzene for a variety of things, has in one of his articles said that AML is 80 percent de novo. He says one percent is due to benzene. I think that figure is high because I think he's not making the distinction between people who said that they've never been exposed to benzene and people who actually have a bona fide benzene caused leukemia. This was not always the case. Obviously in the time of Vigliani and Aksoy^ and the CAPM study and Rinsky's pliofilm study, there is much higher exposure and benzene caused leukemia was more frequent. And data from the sixties would argue that perhaps at that time 15 percent of

studies could have been related to benzene. But that was the data from the sixties, that's not current situation.

Q. What are the studies in the sixties, 15 percent of the AMLs were caused by benzene exposure?

A. It's hard to determine that because someone who's exposed may not have been caused. But when I look at Mitelman's chromosomal data, he had something like 20 percent of people had a history of solvents. And some of those had normal karyotypes and certainly not all of them were caused. So that would be about 15 percent from that figure.

MR. THOMPSON: Whenever you get a chance, Andrew, I want to take a break because we have been going for about an hour and a half or more.

MR. DuPONT: Okay. Let me ask one question, if that's all right with you, Doctor. Or you tell me if you prefer to take a break right away.

THE WITNESS: I'm okay, but

other people may need a break in the room.

MR. THOMPSON: Go ahead, Andrew.

MR. DuPONT: Sorry, why don't we go ahead and take a break.

- - -

(Whereupon there was a recess in the proceeding from 12:34 p.m. to 1:12 p.m.)

- - -

MR. THOMPSON: Okay.

BY MR. DuPONT:

Q. All right, Doctor, did you have your lunch?

A. Yes, I did, thank you.

Q. Good. All right. When we were talking before the break, we were on the issue of age of diagnosis being below average for David Gerchman, but consistent with de novo AML. That was your fourth opinion.

A. Yes.

Q. All right. Do you have any literature references for the comment you gave that less than one percent of AMLs are

caused by benzene exposure?

A. There's an article by Martyn Smith, let me see if I can find the reference to that, which says one percent. Where he gets that figure is unclear. My expectation is that's his estimate for benzene, people who have had a history of some benzene exposure. I don't believe that everybody who claims they have pumped gasoline or have some evidence of benzene exposure necessarily has a benzene caused leukemia, so I think that one percent figure is inflated. But that's the figure I can find from the literature, and what it's based upon is totally unclear.

Let me see if I can find you the article.

I do not have a printout of the article, but the reference is, "^Benzene, The Exosome and Future Investigations of Leukemia Etiology", published in ^Chembile Interactions, Volume 192, pages 155 through 159, 2011. And that information is in Table 1. And where he gets that figure is totally unclear to me.

Q. Is Martyn Smith the lead author of that 2011 article?

A. Yes.

Q. And that's your only citation for your comment that less than one percent of AMLs are benzene induced?

A. My expectation in modern day is that it's fewer than one percent. And while I have given you Smith as something that says one percent, and given you the caveat that I think that's benzene associated, not benzene caused. The other piece of information is the lack of increased leukemia in benzene exposed professions these days where they're exposed to contaminant level of benzene. And based upon that lack, I think that benzene leukemia is quite rare these days, and would estimate it to be less than one percent.

Q. And do those articles concerning benzene exposed professions these days tell you that less than one percent of the AMLs that are diagnosed are associated with benzene exposure?

A. They tell us that --

Q. Do they give you that figure?

A. They don't tell you that directly. They tell you that in these people who worked with contaminant level benzene, there is no increase in AML.

Q. And it's your interpretation of those studies, therefore, that less than one percent of AMLs diagnosed are associated with benzene exposure?

A. If you assume that the people who work with contaminant level benzene do not get increased leukemia, then I would say that they're not getting leukemia that's caused by the contaminant level of benzene. Whether there are pockets in the world that still use benzene, high concentration, not contaminant level, but pure benzene, 70 percent benzene, something like that, in those areas there might be leukemia. I can't speak to what's happening in parts of China, but in the United States I don't think it's an issue.

Q. Okay. So it's your opinion that you can -- or your comment that you can

conclude that less than one percent of AMLs are caused by, or associated with benzene exposure these days is your interpretation of the low dose benzene exposure studies. Those studies don't specifically say that less than one percent of AMLs are associated with benzene exposure?

A. I think that's an inference you can draw from that data.

Q. Okay. And it's your interpretation, it's nothing that's set forth or specifically stated in any of those articles?

A. Correct.

Q. What is the average age of diagnosis of AML in the United States?

A. I don't have the SEIR data in front of me, but roughly 65 years of age.

Q. What percentage of AMLs are diagnosed at age 51 or older?

A. Well, at age 54 or younger it's about 30 percent. So 55 or older, which is not what you asked, but what the SEIR data suggests it would be, about 70 percent.

Q. Do you know the percentage for 51 and older?

A. No.

Q. Does every oncologist/hematologist conduct exposure assessment when a person with leukemia is seen by them?

A. I don't think everybody does anything, but I can't speak for other oncologists.

Q. You don't know what percentage of other oncologists conduct exposure assessments when they diagnose or treat somebody with leukemia?

A. I can't answer that question. I don't know that.

Q. Does every oncologist and hematologist know that solvents, such as carburetor cleaners, brake cleaners, contain benzene?

A. I don't know what other -- every oncologist and hematologist knows.

Q. Is it generally known in your field that brake cleaners and carburetor

cleaners and parts washing solvents contain benzene?

A. I don't know whether that's generally known in my field or not.

Q. Is it generally known in your field that paints contain benzene?

A. I don't know whether it's generally known. And I would wonder whether you're talking about water based paints.

Q. Is it generally known that oil based paints contain benzene?

A. I don't know what's generally known in the oncologic community.

Q. Would you agree that amongst the 80 percent of individuals that you say are de novo leukemia, there's people whose leukemia could have been caused by exposure to benzene, but nobody investigated that?

A. I think the answer to that is unknowable. If they're not investigated, I have no data to answer that question.

Q. What are your references for the statement that 80 percent of -- is it 80 percent of AML specifically, or leukemia

generally that you're saying are de novo?

A. AML.

Q. What are your literature references for the position that 80 percent of AMLs are de novo? And is that figure limited to the United States or is this a worldwide figure?

A. It's the Smith article that I quoted. And I don't recall any restriction on that table telling me whether it was United States or world.

Q. Okay. So that's the 2011 Martyn Smith article, that's your basis for the statement?

A. Yes. And I have told you that I have no idea where he got that information. That's not described.

Q. Okay. Let's go to your fifth opinion. You say that published data on automotive mechanics do not show high benzene exposures. And you cited two articles for me. The first was Scarselli. The second was the other defense witness, Pamela Williams?

A. Yes.

Q. Is that correct?

A. Yes.

Q. Are those the only two references that you rely upon for your opinion that published data on automotive mechanics do not show high benzene exposures?

A. Yes.

Q. And are those the only two articles that you considered on that subject?

A. Yes.

Q. How did you come across those articles?

A. PubMed, ^E-Med, electronic record search.

Q. Do you have those two articles with you?

A. I believe I do. Let me check.

Yes, I have them.

Q. Okay. Let's go ahead and mark them as the next exhibit, please.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 7.)

- - -

BY MR. DuPONT:

Q. Do those articles report the amount of benzene exposure caused through dermal contact with materials containing benzene?

A. I'll have to look at the articles.

(Reviewing articles.)

I don't see that dermal is included in this.

Q. Okay. Have you ever studied a group of mechanics specifically to determine their risk of leukemia, or any other benzene induced disease?

A. No.

Q. Can an oncologist/hematologist derive an opinion on the ability of benzene to cause cancer based upon their observations of their patients over the course of treating patients in their career?

A. People can derive opinions based upon anything. The question is, what's the validity of the opinion.

Q. Would you think it valid for an oncologist/hematologist to say, I have seen patients over my career and I have been able to conclude that benzene is not a cause of AML, or is not a prevalent cause of AML because of what I have seen in my patients?

MR. THOMPSON: I'm sorry, could we have that question back? I couldn't hear the first part of it.

- - -

(Whereupon the court reporter read back the pertinent testimony.)

- - -

THE WITNESS: I think someone who has conducted a study of their patients and recorded whether or not -- made a list of the patients and recorded whether or not there was any history of benzene exposure and finds no history of exposure can say in the patient population that he or she serves that that's not prevalent. The ability to generalize from one patient population to another may vary,

depending upon the details.

BY MR. DuPONT:

Q. Okay. If the doctor hasn't recorded their findings in writing and compared them to another group of non-exposed or exposed individuals, would you think that that is a valid thing to do?

MR. THOMPSON: Objection, incomplete hypothetical.

THE WITNESS: Valid for what purpose?

BY MR. DuPONT:

Q. Valid scientific basis for drawing a conclusion as to whether or not benzene causes AML in any one individual.

MR. THOMPSON: Same objection.

THE WITNESS: It would not be valid to the point of publishable paper.

^MR. CARR: Andrew, this is Chris. May we have an agreement that an objection for one will be good for all.

MR. DuPONT: Yes.

MR. CARR: Will you agree that that applies to the last two objections made by Barry?

MR. DuPONT: Yes.

MR. CARR: Thank you.

BY MR. DuPONT:

Q. All right. If it's not the type of opinion that you think is worthy to be published, would you rely on that type of opinion in providing an expert opinion in a case?

MR. THOMPSON: Objection, vague and ambiguous. Incomplete hypothetical.

THE WITNESS: I have not cited such data in my comments. So I have not relied on such data.

BY MR. DuPONT:

Q. If you found that that type of opinion would not be sufficiently valid to publish in the literature, would you rely upon it in reaching your opinions as to the cause of an individual's AML as a defense expert in benzene litigation?

MR. THOMPSON: Objection. Same objections.

Do you understand the question, Dr. Sarna.

THE WITNESS: The cause of an individual patient's AML would be best judged by the details of the case rather than on the basis of anecdotal information about other patients.

BY MR. DuPONT:

Q. So you wouldn't rely on that type of anecdotal information, as you characterized it?

A. That would not be what I would reach a decision based upon.

Q. Would you consider that at all in your decision making process?

MR. THOMPSON: What do you mean by "that"?

MR. DuPONT: The anecdotal information that he characterized.

MR. THOMPSON: The anecdotal information that an oncologist gathers over the course of their practice?

MR. DuPONT: Correct.

THE WITNESS: If leukemia doctor A had said that when I practiced in the fifties I used to see people who not I attributed leukemia to benzene frequently and I don't see it anymore. That would be useful information to me. It would not rise to the level of published information which is statistically analyzed and peer-reviewed. But it would be useful information, saying that there's been a change in the doctor's practice. That doctor had very large numbers that he is seeing and that suggests a change in the pattern of leukemia etiology. That would be suggestive. I wouldn't say it rises to the level of proof, but it would be suggestive.

BY MR. DuPONT:

Q. Would you consider the bias of that doctor in determining whether or not you would rely upon the information?

MR. THOMPSON: Do you know what

he means by bias of the doctor?

THE WITNESS: I'm not sure why I would ascribe bias to a physician making such a conclusion. If I had reason to ascribe bias, I would consider it, but it's not clear to me that a person -- a hypothetical person making such a hypothetical claim would have a bias.

BY MR. DuPONT:

Q. All right. Let's go to your opinion number six. Literature on modern mechanics do not show an increased risk of leukemia or AML, they show no increased risk.

A. Yes.

Q. And you cited an article Hotz and Lauwerys, H-O-T-Z and L-A-U-W-E-R-Y-S?

A. Yes.

Q. And that's a 1994 article?

A. 1997.

Q. '97, I'm sorry. I misread that.

I'm looking at your opinion, your page five -- excuse me, page eight

through nine of your notes and comments. And this is a section where you address this opinion. And there are -- the articles your cite are Hotz and Lauwerys, McLean, it's dated 2009. ^Costantini, dated 2001, Guo, G-U-O, dated 2004, Ji, J-I, dated 2005, and Pukkala, P-U-K-K-A-L-A, dated 2009. Are those all of the references that you rely upon for the opinions that the literature on modern mechanics do not show an increased risk of leukemia or AML, they show no increased risk?

A. Yes.

Q. Let's gather those references, all of your bases for that opinion, and mark that as Exhibit 8.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 8.)

- - -

THE WITNESS: I'll take a moment to gather what I have. Hopefully I have them all. I will tell you that I

did not printout the entire Pukkala article because it's roughly a 150 pages. And I also printed out material which was relevant that was from the material on the Internet part of the article, but an appendix or an addendum to the article which was relevant to AML. So of the pages that I will provide are the pertinent pages, but not all 150 pages, plus everything else in the addendum. So hold on, let me see what I can get.

Let me take another look I'm not seeing McLean and Costantini. Maybe I don't have them, but I'll take another look. I have McLean. Okay. I have them.

BY MR. DuPONT:

Q. All right. We'll mark those as Exhibit 8.

Are those the articles that we listed, and that are cited at pages eight and nine of your notes and comments, all of the articles you considered in forming this

opinion?

A. I have first cited Hotz, which I have. I have next cited McLean, which I have. I next cited Costantini, which I have. I next cited Guo, which I have. I next cited Ji, which I have. And then I cited Pukkala, which I have the pertinent pages of.

Q. Okay. My question was, those are all the articles that you considered in forming this opinion that literature on mechanics in the modern day do not show an increased risk of leukemia or AML?

A. Correct.

Q. Do any of those articles set forth the dose of exposure that mechanics sustained, either on a cumulative basis or a time weighted average?

A. To my recollection, no.

Q. Do those articles provide details as to the type of personal protective equipment that the mechanics that were the subjects of these studies used?

A. To my knowledge, no.

Q. Do those articles provide

information as to the types of engineering controls that were available and used by the mechanics in the course of their work that were the subjects of these studies?

A. May I hear that question again, please?

Q. Sure. Do these articles that you have cited and relied upon provide information on the type of local exhaust or other mechanical controls used by and around the mechanics that were the subjects of these studies?

A. No.

Q. Do these articles that you have cited provide information on the brand names of the products that were used by the mechanics?

A. No.

Q. Do they provide information on the categories of products used by the mechanics?

A. To my knowledge, no.

Q. Opinion number seven, which was, as you said, David Gerchman had a normal

karyotype and the absence of what you called typical benzene induced karyotype, which argues against benzene being the cause of his AML.

MR. THOMPSON: If you said that, Greg. Just listen carefully to what he said.

THE WITNESS: Run that by me again, please.

BY MR. DuPONT:

Q. Okay. David Gerchman had a normal karyotype. And the absence of a karyotype typical for someone exposed to benzene argues against benzene being the cause of his AML.

A. Both of those factors, which are obviously interrelated, argue against it, yes.

MR. THOMPSON: Just to make clear, he read it differently the second time, Greg, so you should pay close attention.

THE WITNESS: Okay.

BY MR. DuPONT:

Q. Okay. What are the references and bases for that opinion?

A. The reference that I think most useful is an article by Mitelman. Let me pull it. There are a variety of articles that I haven't pulled based upon my general knowledge. These include a whole series of chromosomal abnormalities in normal people exposed to benzene by Zheng's group up at UCSF, as well in vitro studies on lymphocytes and other studies with benzene metabolites by that same group.

There are also a variety of others, which I'll discuss, but let me give you Mitelman first.

Mitelman is, "Chromosome Pattern, Occupation, and Clinical Features in Patients With Acute Nonlymphocytic Leukemia" published in 1981 in "Cancer Genetics and Cytogenetics." Do you want me to discuss this, or did you just want that information?

Q. What I would like to do is first get the list of the articles that you're relying up, and then we'll have some

discussion about that.

A. I also found useful Dr. Shadduck's literature by Dr. Smith, "Advances in Understanding Benzene Health Effects and Susceptibility", in reviews in advance of "The Annual Review of Public Health". That was useful as well.

But here I think there's a broad area of literature that is generally accepted that chromosomal abnormalities that are benzene associated are benzene associated to make a ^totalogy. And I have not quoted every -- this is not a formal report, so I have not gone to quote every article. The Mitelman article, the Smith article was supported in the broad sense, as well as the body of work by Zheng and her group.

Q. That would be part of it.

So if you were to testify at trial and you were asked what literature do you rely upon for this opinion, your response would be the Mitelman 1981 article, the articles from Zheng and her group out of University of California, Berkeley, and the

Smith article cited by Dr. Shadduck?

A. And the variety of other articles which don't come to mind specifically at this time.

Q. All right. Can you cite me any other articles that I can have an opportunity to add --

A. Off the top of my head, no.

MR. THOMPSON: Do those articles you just cited have references in them?

THE WITNESS: I expect they do. Let me go on further --

BY MR. DuPONT:

Q. Do you know --

A. May I correct my answer to your question?

Relating to the article by Smith, there are articles that are pertinent by Mauritzon, as one, and by a different Smith, S. Smith, as opposed to Martyn Smith, as another. And those I can provide you.

Q. Do you have those with you here today?

A. I believe so. Let me get those.

I can also give a reference by Pedersen-Bjergaard as well. So, yes, I pulled those as well.

Q. So then let's mark your reliance materials, your articles for your opinion on the normal karyotype in absence of a karyotype typical for benzene exposure as Exhibit 9.

- - -

(Whereupon the articles were marked, for identification purposes, as Sarna Exhibit Number 9.)

- - -

THE WITNESS: Let me also clarify that I'm using those articles to draw data. I'm not using articles to quote statements.

BY MR. DuPONT:

Q. Okay. Well, do any of those articles make the conclusion that the absence of an abnormal karyotype excludes benzene as a cause of AML?

A. Not specifically, no.

Q. Is there any author you can cite for the proposition and the conclusion that the absence of an abnormal karyotype excludes benzene as the cause of AML?

A. Absolutely excludes it, no. Argues against it, there's an article, and I'm sorry I'm blocking the author, something, a physician's view of AML, something like, a fellow who published on ^RARS as well, that argued about the chromosomal abnormalities, as I recall. And I'm sorry, I'm blocking his name.

Q. Would that be ^Ethan Natelson, one of the other defense experts in this case?

A. This would be Dr. Natelson, yes.

Q. Are you able to cite me any other articles, besides the one from Dr. Natelson, who has also been hired by defendants in this case?

MR. THOMPSON: Do you know if Dr. Natelson has been hired by the

defense, as he says? Do you know anything about that, Dr. Sarna?

THE WITNESS: This is the first I have ever heard of it.

MR. THOMPSON: So just ask him a question that he knows all the information for and he'll be happy to answer it.

MR. DuPONT: I'll ask him a question as I see fit.

MR. THOMPSON: Okay. Hold on a sec. Greg, if there is any part of what he puts into his question that you don't have information for, stop him and either ask for it or ask for clarification. Because he just told you he's going to ask you misleading questions, basically. So I'm just telling you now, make sure that if he puts something in the question you either know it or don't know it or stop him.

BY MR. DuPONT:

Q. Dr. Sarna, following your

attorney's instructions on how you can answer questions, can you tell me --

MR. THOMPSON: Don't answer that question, Greg. With that preference, don't answer that question. Wait until he asks a standalone question.

BY MR. DuPONT:

Q. Can you cite for me any article, beyond the one you cited by Ethan Natelson, who has been hired by the defendants in this case, which you claim argues against benzene exposure being the cause of AML if there is an absence of an abnormal karyotype?

A. The data from the --

MR. THOMPSON: Objection, assumes facts not in evidence. Calls for speculation by this witness. And please listen to my instruction, Dr. Sarna. If he puts stuff in there that you don't have information about, you have to tell him or he's going to assume you agree with him.

THE WITNESS: May I have the

question again, please?

- - -

(Whereupon the court reporter read back the pertinent testimony.)

- - -

THE WITNESS: The data from the articles that I have previously discussed, as well as data from articles which I don't have with me, argue against it.

BY MR. DuPONT:

Q. My question though is about the conclusion actually drawn by the author of the study. And can you cite for me an article where the author has drawn a conclusion that the absence of an abnormal karyotype argues against benzene being the cause of the AML, other than the one by Ethan Natelson, who has been hired as a defendant in this case?

MR. THOMPSON: Same objections.

THE WITNESS: There may or may not be such an article, but I can't cite one at this time.

BY MR. DuPONT:

Q. All right. Let's talk about the Mitelman 1991 article.

A. May I have that back, please?

MR. THOMPSON: '81.

BY MR. DuPONT:

Q. Excuse me, '81.

A. Mitelman is 1981. Go ahead.

Q. Did that article -- first of all, does that relate to the individuals that have been diagnosed with AML?

A. This results with individuals who have been diagnosed with what is termed acute nonlymphocytic leukemia, which would generally be AML.

Q. Okay. What are the findings of that study?

A. They looked at the chromosome genetics, meaning karyotype, on 110 subjects with no history of exposure to solvents or pesticides or petrol products. Of these 110, 75 had normal karyotypes. They also looked at, it looks like 28 -- 27 subjects who had a history of solvent exposure.

Now, these people were from the seventies, '72 through '70 -- from the early seventies, it appears. And likely their exposure was in the fifties, sixties and seventies, but it's not detailed. But it's reasonable to assume that the exposure was in that period of time.

Of the 27 subjects who were solvent exposed, three had normal karyotypes. So there is quite a difference there. 24 of 27 had abnormal karyotypes, as opposed to I think it was like 30 percent or so of the people who were not exposed, which is a rather dramatic difference. When you look at the karyotype abnormalities, they tend to have repetitive themes; minus five, 5q minus, minus seven, 7q minus. To some degree trisomy 8, and complex karyotypes meaning three or more different abnormalities.

Those types of abnormalities have also been seen in the in vitro studies and the normal exposed without leukemia studies by Zheng, and have also been seen in the alkylating agent associated leukemias

that are a subset of the therapy related leukemias.

One of your experts, I think Shadduck --

MR. THOMPSON: Greg, he just asked what the findings were.

THE WITNESS: Fine.

BY MR. DuPONT:

Q. Is there anything that you wanted to add?

A. That will do it.

Q. All right. So it's your opinion that the subjects in Mitelman were benzene exposed workers?

A. They were described as solvent exposed workers, and their professions were listed and they varied. Metal industry worker, chemical industry worker, machine cleaner, bookbinder, printer, painter, firemen, et cetera. There were a variety of different ones. And, again, these were people who were living in a different era when there was more benzene exposure than there is in the modern era.

Q. Well, the articles state that they were benzene exposed workers.

A. The article states they were solvent exposed workers.

Q. All right. And does the article provide data to establish that the workers had benzene exposure?

A. It lists their professions, it does not go beyond that.

Q. Have you seen any data outside of this article that establishes whether these subjects had exposure to benzene? In other words, data for these subjects in this study?

A. No.

Q. And it's your assumption that they did have benzene exposure?

A. Given the chromosomal pattern being so different in this group than the chromosomal pattern in the nonexposed group, I am assuming that a substantial number of them had benzene exposure and benzene associated leukemia. Not necessarily all of them. In fact, there were I think three

normal karyotypes in this group. And perhaps those three did not have benzene associated leukemia -- benzene caused leukemia. But I think there is a dramatic difference in karyotype, which tells me that something is different between these two. And what I'm told is different is solvents.

Q. Do you know, does that article or any other article on these workers tell us the time period during which -- in other words, the latency period or the period prior to diagnosis with AML that these individuals had exposure?

A. The timeframe of exposure is not defined.

Q. All right. Let's go to the next article that you have with you, what is that? The next article in Exhibit 9 would be normal karyotype, absence of the karyotype typical for benzene exposure.

A. May I have Exhibit 9 back.

The article by Mauritzson, M-A-U-R-I-T-Z-S-O-N, published in Leukemia, 2002.

Q. All right. Does that article address individuals who have been diagnosed with AML?

A. Yes. And myelodysplasia as well.

Q. What are the findings of that article?

A. They looked at the chromosomal abnormalities in people with de novo AML and de novo MDS, and compared it with those who had treatment related AML and treatment related MDS. And they found that chromosomal abnormalities in treatment AML and in treatment MDS was much more frequent than in de novo.

Q. And these were not benzene exposed individuals?

A. No, these were treatment related.

Q. Do we know exactly how benzene causes AML?

A. No.

Q. Do we know exactly how chemotherapeutic agents cause AML?

A. For both of these questions the answer is no, but we do know that the process commonly with both of these, if you're talking about alkylating agents as chemotherapy, involves abnormalities of 5 and 7, with partial deletions or full deletions as a critical event.

Q. Do we know exactly how alkylating agents cause partial or full deletions of the fifth and seventh chromosome?

A. Well, they alkylate DNA, but beyond that I don't have a specific answer.

Q. All right. What's the next article you have in your pile that's marked as Exhibit Number 9?

A. Sonali Smith, "Clinical-cytogenetic associations in 306 patients with therapy-related myelodysplasia and myeloid leukemia: The University of Chicago series."

Q. What's the date of that article?

A. 2003.

Q. This is another study of therapy related, in other words, chemotherapy caused AML and MDS?

A. Yes.

Q. It's not a study of benzene exposed workers?

A. Correct.

Q. What do you take from this article?

A. Because of the same chromosomal pattern, because both benzene leukemia and alkylating leukemia commonly involves pre-existing MDS, I take it that alkylating agent induced chemotherapy and treatment-related chemotherapy in general, although I would separate out the Topo II inhibitors because that doesn't fit, but putting that issue aside, it is a reasonable model for benzene. They have the same chromosomal abnormalities and they seem to have the same frequency of myelodysplasia beforehand, both caused myelodysplasia and AML, in contradistinction to the Topo inhibitors. So I think this is a valid

model. And as a valid model I think that it is appropriate to look at it and understand the difference in chromosomal patterns.

Q. Has there been any epidemiology study that has used chromosome damage as a basis for determining whether benzene exposure was or was not the cause of AML?

MR. THOMPSON: Objection, form, foundation. Do you understand the question?

THE WITNESS: There are the types of studies by Mitelman which point to it, but that is looking at solvent exposed first, rather than looking backwards and then seeing if there's a history of solvents. So the answer to the question I have is no.

BY MR. DuPONT:

Q. Has there been any government or non-government agencies that have concluded that the presence of chromosome damage for an abnormal karyotype is necessary to draw a causal association between benzene exposure and AML in an individual?

A. I'm unaware of any such occurrence.

Q. What's the next article that you have in Exhibit 9?

A. Dr. Shadduck's Exhibit 12 I think it was, by Martyn Smith, "Advances in Understanding Benzene Health Effects and Susceptibility."

Q. What do you take from that article?

A. Dr. Shadduck took from that article that --

Q. Sir, I didn't ask you about Dr. Shadduck took, I asked what you took from that article.

MR. THOMPSON: Greg, go ahead and answer it however you want.

THE WITNESS: Dr. Shadduck took from that article that you could see the same type of chromosomal abnormalities in benzene related and unrelated cases based upon Smith's use of treatment related therapy. I take from it that treatment related therapy

is a valid model, just as Dr. Shadduck apparently accepted from Dr. Smith. But my take on it is that you look at the frequency, not whether it shows up ever. And there is a marked difference in frequency from the data that I have shown you.

BY MR. DuPONT:

Q. Okay. So I'm going to ask the question again because my question is what do you take from it? What do you take from this article?

A. My take is that there are differences in frequency between the 5, 7 and 8 abnormalities. I also take from it that Dr. Smith believes, based upon the models that he uses, which are labeled in Figure 1 through 8, that benzene causes leukemia through chromosomal abnormalities of 5 and 7 particularly. And that de novo cases typically have a normal karyotype.

If you look at his Figure 1, he has alkylating agents and he says benzene works along with fast life, which is loss of

7, deletion 7q, loss of 5, deletion 5q, and then at the bottom he has de novo cases which are listed as de novo, meaning no cause. So they start with the normal karyotype.

Q. Do you consider Dr. Smith's opinions authoritative?

A. These particular opinions, yes. There are other opinions he's had that I have disagreement with.

Q. Because they don't favor your side of the case?

MR. THOMPSON: Objection.

THE WITNESS: Because I have looked at his opinions and the data on which they're based and I find they're fallacious.

BY MR. DuPONT:

Q. What opinions would those be?

MR. THOMPSON: Are we talking about an AML case or are we talking about an NHL case, or something else?

MR. DuPONT: I don't know, the doctor can answer my question.

THE WITNESS: He's published a

Meta-analysis on lymphoma, or what he's called a Meta-analysis. The first part of that is a listing of anecdotal positive odds ratios out of studies. These include studies that may have 10 different odds ratios, some of which are positive, some of which are negative.

When you look case by case at the studies that he calls positive and look at the breadth of what the conclusion is, which I have done for either the first five or 10 cases that he cites, they don't, in my judgment, support the concept.

I think you can cherry pick positive results or negative results and cherry picking is not the way to reach a conclusion.

The second part of that article deals with cohort studies. He agrees that the cohort studies don't show an increase in lymphoma, but attributes this to a healthy worker effect. In

my judgment, healthy worker effect does not decrease the risk of lymphoma. A healthy worker effect is largely due to people who have smoking and other types of problems being excluded and I don't --

BY MR. DuPONT:

Q. All right. Let's move beyond the Smith article and now let's talk about --

MR. THOMPSON: Hold on. You're the one who --

BY MR. DuPONT:

Q. I want other opinions that Smith has that you disagree with.

MR. THOMPSON: Say that again? He's not done with his answer.

BY MR. DuPONT:

Q. I said, let's move beyond the Smith article and the Meta-analysis on non-Hodgkin's lymphoma and ask, are there other opinions that Smith holds that you disagree with?

MR. CARR: No, I'm going to ask that you withdraw the question, if

that's what you want to do. Because we're not on the video record, the record would reflect that he's been interrupted and cut off before his answer is complete. So the record is going to look like that's all he had to say, and it's not. So I think you either need to withdraw the question or do something else other than cut him off and tell him to do something else.

MR. DuPONT: Well, I would like to move beyond the Smith article and the Meta-analysis of NHL. Because when I asked him what opinions he had, I didn't ask him for a dissertation on this one article.

MR. CARR: You asked him --

BY MR. DuPONT:

Q. Doctor, what opinions did Dr. Smith hold that you disagree with beyond his conclusions and his analysis in the Meta-analysis --

MR. CARR: Andrew, I'm okay with

that, so long as you make the record clear that you stopped him in his last answer.

THE WITNESS: I did not expect to be asked about Dr. Smith's opinions in other areas.

MR. THOMPSON: Hang on a second, Dr. Sarna. Andrew, can you make clear on the record that you stopped his last answer and you're now moving on?

BY MR. DuPONT:

Q. I want like to ask Dr. Sarna about opinions that Dr. Smith holds beyond the subject of the NHL Meta-analysis article.

MR. CARR: And would you agree that he was answering that question and you stopped him?

MR. DuPONT: As he advised that I should do.

MR. CARR: That's a yes, right? You stopped him before he was finished.

MR. DuPONT: I stopped him before he was finished because he was

going off on a tangent that had nothing to do with the question.

MR. THOMPSON: Okay. So, no, we don't have an agreement now. Andrew, remember, I warned you at the start of your question if you really wanted to answer this. So now we've gone on for five minutes on something that I prepared you for.

MR. DuPONT: Barry, would you please stop it.

MR. THOMPSON: Stop what?

BY MR. DuPONT:

Q. What opinions, beyond Dr. Smith's non-Hodgkin's lymphoma Meta-analysis that he holds, do you disagree with?

MR. THOMPSON: If you formed those opinions at that present time. Because we haven't asked you to look at those.

THE WITNESS: I disagree with his participation in the ^Stimus article. But I'm not prepared to discuss those areas of disagreement

today as it was not part of my charge.

In terms of other areas, there may or may not other areas of disagreement, I have not looked into that. It was not expected to be part of my charge, I'm not prepared to give you an answer to that today.

BY MR. DuPONT:

Q. All right. What's the next article in your pile of Exhibit 9?

A. Pedersen-Bjergaard, "Genetics of therapy-related myelodysplasia and acute myeloid leukemia" talks again about 5q minus and 5 minus, 7q minus and 7 minus in treatment related AML and MDS.

Q. Again, these are not benzene exposed workers?

A. Correct.

Q. Does that exhaust the list of articles that you have with you here today on the subject -- that we have attached as Exhibit 9, the subject of the normal karyotype and absence of an abnormal karyotype typical for benzene exposure?

A. I believe so, but it's conceivable that I missed an article in my pile.

Q. Well, I would like to make sure we have everything. So why don't you take a look and see if you missed anything.

A. Okay. That appears to be all.

Q. Doctor, your next opinion is number eight on the latency issue, which I understand you used a different word than latency. Your opinion that AML arises generally within ten years, on average four to five years prior to diagnosis of AML. Correct?

MR. THOMPSON: Do you agree with that restatement of your opinions?

THE WITNESS: Say it again, please?

BY MR. DuPONT:

Q. The time that AML arises from a causative exposure is typically less than ten years, and on average four to five years prior to diagnosis of AML?

A. Yes.

Q. Okay. And you disagree with the use of the first date of exposure as a basis for determining latency?

A. I don't think the first date of exposure is when a disease was caused, if one even assumes that it was caused. And, therefore, if latency is defined as time from cause to presentation, that doesn't work for me.

Q. What if latency is not defined as time from cause to presentation?

A. If you define latency as time from first exposure to occurrence in the situation where there may have not been any causation, then I'll accept that. But that definition to me doesn't mean much.

Q. What articles do you rely upon and what is the basis for your opinion number eight regarding the lag time from causative exposure being generally less than ten years, on average four to five years from diagnosis?

A. Those figures are well known for alkylating agents. And I did not cite specific references, but there are multiple

references that come to that conclusion. Series of alkylating agent related chemotherapy -- alkylating agent related leukemias.

Topoisomerase inhibitor related leukemias come a bit earlier than four to five years. They come about three years and they don't have the MDS beforehand. So that's different. That would be by analogy.

In terms of specifics, there's an article by Triebig, which I'll pull.

Implications of Latency Period Between Benzene Exposure and Development of Leukemia, a Synopsis of Literature," by Gerhardt Triebig, published in 2010, Chemico-Biological Interactions, Volume 184, pages 26 through 29.

Q. And is that the only article for this opinion that you cite?

A. That is the only article that talks directly about benzene exposure and benzene associated leukemias. There is also the data which I talked about generically in terms of alkylating agent leukemia. And

there is one of the articles that I gave you on smoking that talked about the risk of smoking related leukemia disappeared after about five years, which implies that the latency period for smoking related leukemia is five years or less. So you would still have a continued increased risk.

Q. What articles do you cite for the alkylating agent inducing AML as it relates to your latency opinion?

A. That is so well established and generally known that I did not cite articles.

Q. Okay. What articles do you cite for the smoking risk that hereinafter five years as being relevant to the question of latency?

A. I need to have the exhibit dealing with smoking and leukemia handed back to me to give you the precise reference. If I may have that exhibit.

Q. I believe it's Number 6.

A. (Reviewing document.)

The article by Kane in that exhibit talks about the relative risk for

acute leukemia in smokers. Current smokers have a relevant risk of 1.4. Ex-smokers, who have stopped smoking within one to ten years, have a relative risk of 1.1. So that would be the article.

Q. All right. Is the benzene the culprit in cigarette smoke that causes AML?

A. There are thousands of other contenders for that. It is not known, or at least I don't know that.

Q. All right. Let's talk about the one study you're able to cite that relates to benzene exposure, and that's Triebig. What type of study was Triebig?

A. They were -- they looked at cases in Germany and looked at people who had a history of benzene exposure. Such cases, and what their relative risk of leukemia was after they stopped being exposed to benzene. And found that -- and I'll pull it from the abstract, "It is concluded that the epidemiologic findings are consistent and demonstrate a smaller or even absent risk of leukemia 10 to 15 years after exposure to

benzene has been stopped." So basically they looked at people who were benzene exposed and looked at what their risk was after they stopped the exposure. And by 10 to 15 years it was probably gone.

Q. Sorry, you were cutting out there. Is this an actual epidemiology study or is this a review of other literature?

- - -

(Whereupon the court reporter read back the pertinent testimony.)

- - -

THE WITNESS: It's a review of other epidemiologic studies.

BY MR. DuPONT:

Q. Do you know anything about Dr. Triebig's background?

A. I know that he is at the Institute and Outpatient Clinic for Occupational and Social Medicine at the University of Heidelberg. Beyond that I do not know.

Q. Do you consider yourself to be an epidemiologist?

A. By trade, no.

Q. I believe we have already covered, you have never conducted any epidemiology study, have you?

A. That is correct.

Q. You have never peer-reviewed any epidemiological papers?

A. I don't recall doing so.

Q. You have certainly never published any epidemiological papers; correct?

A. I have published book chapters on malignancies which cover the epidemiology. They are not restricted to epidemiology. And they are not original work. But I have published with epidemiology as part of those publications.

Q. And that was not benzene epidemiology; correct?

A. It was disease --

Q. Epidemiology of benzene induced malignancies --

A. It was disease focused etiology. And my expectation is that I did

not conclude that benzene was a cause of lymphoma when I discussed the epidemiology of lymphoma in chapters.

Q. Do you generally defer to an epidemiologist when it comes to opinions on epidemiology?

A. No.

Q. No, you do not?

A. No, I do not.

Q. You think you're more qualified than an epidemiologist in order to interpret epidemiology studies?

A. I think in every trial I have been involved in, there has been an epidemiologist on both sides who have diverging opinions. And it would be naive to consider that one -- if the opinions are different, that they're both right.

Q. That wasn't my question.

My question is, do you consider yourself to be more qualified than an epidemiologist to interpret epidemiological studies?

A. Under some circumstances, yes.

Certainly I have seen problems with epidemiologic research that other people have not, or that were not being aware of at the time of publication.

Q. How do we know leukemia actually arises in a person? Is there any article that has said, well, I've scientifically proven the precise time that leukemia has arisen in a person prior to their diagnosis of leukemia?

MR. THOMPSON: Andrew, can you just re-ask that?

BY MR. DuPONT:

Q. Okay.

Has any scientist been able to define the precise moment in an individual when that individual's leukemia arose prior to diagnosis of leukemia?

A. Are you talking about to the second?

Q. Any metric. Year?

MR. THOMPSON: With reasonable precision.

THE WITNESS: Yes. The answer

is, yes.

BY MR. DuPONT:

Q. Okay. And who has done that?

A. I don't know the author. But the studies are the studies of chemotherapy and radiation induced leukemia.

Q. Okay.

A. When someone --

Q. Has anyone done that --

A. When someone was exposed at Hiroshima and Nagasaki, you have a period of time of perhaps an hour when their leukemia was caused. When someone has had three cycles of chemotherapy over a nine week period, you have a nine week window when that was caused.

Q. So is it your opinion that somebody receiving chemotherapy is caused to contract leukemia when they're actually receiving the chemotherapy?

A. That's when the genetic insults occurred. Chemotherapy is a excreted from the body rather rapidly and it may not have been the moment it went into the IV line, but

it was still in the body for two or three days and that's when the genetic insult occurred, or else you would not have had leukemia.

Q. Does every genetic insult lead to leukemia?

A. As I mentioned before, we have perhaps 10 to the 27th mutations in our lifetime. So the answer is, no.

Q. Does every genetic insult by a chemotherapeutic agent lead to leukemia?

A. The risk of leukemia in alkylating agent therapy is probably one percent or less. So the answer is no.

Q. Does every person respond the same way to chemotherapy in terms of how their genes are affected?

A. If only one percent of people who get alkylating agents contract leukemia, then obviously 99 percent didn't.

Q. Before we take a break, do you have the Hiroshima studies as part of your basis for saying -- as part of your basis for the latency opinion?

A. I don't have as clear data on that in terms of time. So, no, I'm basing it on chemotherapy. But as a model, most of the radiation induced ones are within ten years, but I have not pulled literature on that.

Q. But the Hiroshima studies informed your analysis on the issue of latency?

A. Not so much on 5 minus, 7 minus and the fact of chromosomal abnormalities that are shared by alkylating agents, that's a better marker because the chromosomal abnormalities with radiation may be different.

Q. But generally with respect to a mutagen causing leukemia, the Hiroshima studies form your analysis?

A. It's part of the body of evidence, just like topoisomerase inhibitors which causes much -- in a shorter period of time, that I have quoted and are part of the body of evidence. But I am not hanging my hat on radiation because that does not have the same chromosomal pattern that benzene

has.

Q. All right. Let's take the break.

MR. THOMPSON: Okay.

- - -

(Whereupon there was a recess in the proceeding from 2:36 p.m. to 2:46 p.m.)

- - -

BY MR. DuPONT:

Q. Doctor, would you agree with me that there are literally hundreds of epidemiological studies that use the first period of exposure as the basis for defining latency?

A. For using the word, that definition? I don't know if there are hundreds, there are certain many.

Q. Would you agree that the vast majority of epidemiology studies use the first day of exposure as the metric for the time of latency?

MR. THOMPSON: If you know.

THE WITNESS: My belief is that

most of them who use the word latency use that, but a rose is a rose is a rose.

BY MR. DuPONT:

Q. Is it your assumption that the leukemias caused by chemotherapy arose during the time period of exposure to chemotherapeutic agents, as opposed to some time after the chemotherapy was stopped and the chemotherapeutic agents left the body?

A. The answer to the question is yes. But if I accept that it was later, that would even shorten the latency.

Q. Well, I guess what I'm getting though is, do we know that it is the mutation that takes place after the exposure to chemotherapy, as opposed to during the chemotherapy exposure that caused the leukemia?

A. I cannot distinguish whether it is during the actual infusion or afterwards, but the chemotherapy stays in the body generally for a couple of days afterwards. So statistically it is much more likely to be

after the five minute infusion rather than during the five minute infusion.

Q. Can you rule out exposures to patients that occur after the chemotherapy has left the body as the cause of the leukemia?

MR. THOMPSON: Mutations from the chemotherapy or just mutations?

MR. DuPONT: Mutations that were put into place by the chemotherapy.

THE WITNESS: Again, it would shorten the latency, not lengthen it. But the answer to the question is, there could be a -- no. Causes five, causes seven, those are direct effects of some agents and the agents is given. So, no, I don't think it would be a delayed effect.

BY MR. DuPONT:

Q. Okay. The studies on alkylating agents that you rely upon for your latency/arose opinion, did they study whether the individuals who are subjects of those studies had exposure to any mutagenic agent

prior to their chemotherapy?

A. I don't recall a study doing that, but when you have a 30 year old who gets leukemia with 5 and 7 three years after or five years after alkylating agent therapy and you see that pattern over and over again with the same timeframe in someone who is 30 or 20, one reaches then a reasonable conclusion that it's causal.

Q. But you don't know what portion of those 30 year olds worked at a gas station in high school?

A. No, I don't know that.

Q. All right. Let me change the analysis a little bit. If you had somebody who is exposed to benzene for a 40 year period of time, is it your opinion that the exposures occurring more than ten years prior to the diagnosis with AML played absolutely no role in causing the AML, even if there were exposures within ten years of diagnosis?

A. I think if there was an -- if you postulate that someone had a benzene caused AML, then it would be caused on an

average of five years prior to diagnosis, and the great majority within ten years. There might be some that are 11 years or 12 years. I don't think there would be any that would be 20 years.

Q. What I'm asking is, if you have a continuous 40 year period of exposure, that ends less than five years before the diagnosis with AML, at what point in time do you say, we can affirmatively cut off the exposures as playing any causative role in causing AML?

A. I think you look at exposures within ten years of diagnosis as being relevant.

Q. And you would say that no exposure that occurred more than ten years before diagnosis, even if there was a cumulative and ongoing exposure over 40 years, that ends within five years from diagnosis, you would say that none of the exposures more than ten years prior to diagnosis played any role in causing the AML?

A. I think cumulative exposure as

a metric can predict who's at risk for benzene related to acute leukemia. But largely that's because the risk goes up with age and because the risk normally occurs at the tail end of a high -- long career of exposure rather than someone who's only had brief exposure.

However, I think what's relevant to causation is what's in the first -- what's in ten years preceding it. Because if it were caused 15 or 20 years, it would have shown up sooner.

Q. Well, my question was different. Do you completely rule out exposure that occurred more than ten years before the date of diagnosis, even if the person had a 40 year exposure history that ended within five years of the date of diagnosis?

A. I would --

MR. THOMPSON: Do you have anything to add your answer?

THE WITNESS: I would completely rule out 20 years or more. I think it

exceedingly unlikely 15 years or more. I think it very unlikely 10 years or more.

BY MR. DuPONT:

Q. Is there any author that has ascribed to that conclusion in the peer-reviewed literature?

A. Spoken in the way that I have spoken it, I can't say that I know of someone who has done that. Again, Triebig came to his conclusion. The alkylating agent data are what they are.

Q. All right.

A. And whether Natelson dealt with this I don't remember.

Q. What role, if any, does intensity of exposure play in the latency question?

A. I think that latency is not a relevant issue if there is inadequate exposure to be causal. The latency issue assumes that there is adequate exposure to be causal.

Q. Well, if the exposures are more

intense, is the latency period likely to be shorter or longer?

A. No.

Q. So the intensity of exposure, in your opinion, has no effect on the latency period?

A. If there is a causal event, which is a function of intensity, but if you posit there is a causal event, then it doesn't matter whether there was a lot of exposure or little exposure. The causal of event occurs and it's going to take its time course as it will. That doesn't mean that low dose exposure causes leukemia. It means that if you are assuming that benzene caused leukemia, it doesn't matter what the setting of exposure was.

Q. All right. Let's move to your opinion number nine, spontaneous mutation causes AML. What is a spontaneous mutation?

A. One that occurs normally, naturally, without an existing cause. External cause.

Q. Has anybody studied the issue

and determined that there are spontaneous AMLs amongst somebody who has never been exposed to any leukemogenic agent?

A. Well, I would say de novo was spontaneous, and the literature is replete with distinction between de novo acute leukemia and treatment related acute leukemia.

Q. And it's your position that people who have de novo leukemia never had any exposure to leukemogenic, including benzene?

A. It's my opinion that people with de novo leukemia cannot have their leukemia ascribed to any external factor. The issue is one of statistics. That is, if you have a hundred million mutations that are spontaneous, and if you, because you have pumped gasoline for your car, get an additional hundred mutations, it's not very likely that a hundred mutations is relevant, given the hundred thousand spontaneous mutations. Sort of like --

Q. Doctor, my question was

different. My question was, is it your opinion that people who have de novo leukemia never had any exposure to leukemogens?

MR. THOMPSON: Any exposure to any leukemogen at any time in their life?

MR. DuPONT: Yes.

THE WITNESS: Every person in the civilized world has had exposure to benzene in the ambient air.

BY MR. DuPONT:

Q. So is there any study that has looked at individuals that have never been exposed to a leukemogen and found that they had a spontaneous mutation that caused AML?

A. Everyone in the world has been exposed to a leukemogen. So it's an impossible study to do.

Q. So what references do you rely upon for your opinions that there are spontaneous mutations that cause AML?

A. There was an article in Science, which was written the year that DNA was the molecule of the year, I believe. Or

maybe it was not DNA molecule, maybe it was DNA repair enzymes were the molecule of the year. And at that time there was a measurement of spontaneous mutations occurring within the genome. And the author concluded that there was something like ten to the 27th, which is 10 with 27 zeros after it, spontaneous mutations. And made the point that these are much more dangerous than causing cancer than any external carcinogen.

I do not off the top of my head have the specific reference. I probably can find it, although it may take some work. But the point remains, spontaneous mutations are normal and ubiquitous and multiple. And again, this is the nature of evolution. Without spontaneous mutations we would all be one cell organisms.

Q. All right. Did that article that you reference without being able to cite, conclude that spontaneous mutations cause AML specifically?

A. Cancer, yes. AML specifically, no. To my knowledge.

Q. Does that article say what percentage of the spontaneous mutations actually lead to cancer?

A. Yes. Very few. Since there are 10 with 27 zeros after it and most people get three or four cancers, you're not going to be getting the majority. The majority of them are either in areas that don't cause cancer or are repaired. It's the ones that get through that cause cancer.

Q. You said that most people get three or four cancers?

A. No, I said most people get at most three or four cancers. 40 percent of people will get cancer in their lifetime currently. If you're cured of cancer one, you're certainly at risk for getting cancer two.

Q. So do you know what percentage of these spontaneous mutations that can lead to cancer?

A. You can say that for a patient what got cancer and who lived till age 60, it was probably one over 10 to the 26th or

something like that. One over 10 to the 25th maybe.

Q. Is that something that was published in this article or something that you are concluding on your own?

A. I am concluding it based upon there being, to my recollection, 10 to the 27th spontaneous mutations in normal lifetime. I may be off on my number, but it's a big number, whatever it is.

Q. Your 10th opinion that Mr. Gerchman's exposures were qualitatively small over the 10 years preceding his diagnosis, what is your basis for that opinion?

A. Deposition said that he worked roughly half time, some said 60 percent, some said 40 percent, at GnG doing mechanic's work. And over that period of time that he only did light mechanic's work. So that would be for eight of the ten years or so. Over that period of time, he would be working with contaminant level benzene, which generally would be very low.

So based upon that, and based

upon the fact that the typical mechanic's exposure based upon Scarselli's article is small. And the typical exposure based on Williams' article in terms of modern chemicals is small. I think that it was small exposure.

Q. Okay. That's the only exposure assessment that you performed for David Gerchman for those 10 years?

MR. THOMPSON: I'm sorry, are we on the opinion where he says he thinks that the exposure was qualitatively small over ten years?

MR. DuPONT: Yes.

THE WITNESS: Just asked me about ten years.

BY MR. DuPONT:

Q. That's the only thing that you gave me.

MR. THOMPSON: Did you also look at Petty and Williams' reports in this case, Doctor?

THE WITNESS: The reports from Dr. Petty and Williams are very

different. Dr. Petty's report claims a lot of dermal exposure, which is, to my knowledge, largely unprecedented in the literature. Doctor Williams differs.

BY MR. DuPONT:

Q. Dermal exposure is unprecedented in the literature?

A. To be as large a function of the benzene load. I will let Dr. Williams and Dr. Petty deal with the details. I'm talking qualitatively, not quantitatively. But it seems to me that Dr. Williams' analysis is more in the main stream, and that that would be consistent with a low exposure. If Dr. Petty's analysis, which I could view as not in the main stream, happens to be correct, then I would say that one would have to take a look at the exposure data claimed in the Health Watch study and the pliofilm study and the other studies, and recalibrate, using Dr. Petty's method. And I expect at that point the benzene associated ppm years or other markers of concentration would have

to be in association -- those associated with leukemia would have to be revised considerably upward based upon new exposure analysis.

Q. Doctor, I would like you to answer my question, as you agreed that you would do at the beginning of the deposition.

A. What is your question?

Q. Did you qualitatively assess Mr. Gerchman's exposure more than ten years before his diagnosis, or did you just rely on the report of Pamela Williams and Stephen Petty for that information?

A. I qualitatively assessed it as more than in the last ten years, but still modest.

Q. Is it your opinion that David Gerchman's benzene exposures were consistent throughout his career as a mechanic? In other words, there was no period of time where his exposures were higher or lower?

A. I expect that his experience in the last ten years was lower than in the preceding years.

Q. All right. And what was the difference? How much greater was his exposures on an eight hour time weighted average or a peak level in the ten years before his diagnosis versus the period of time more than ten years before his diagnosis?

A. You're asking a quantitative question, and I did not make a quantitative analysis.

Q. Okay. Qualitatively, what was the difference in the exposures? How much greater was the exposures in the period more than ten years before diagnosis than the period of time roughly 10 years before diagnosis?

A. A bit better. A bit greater.

Q. A bit greater?

A. Yes.

Q. How do his exposures vary from date of 1970s to the 1980s?

MR. THOMPSON: If you formed an opinion on that.

THE WITNESS: I don't have a

quantitative analysis. I have qualitative. And I can say it's a bit greater, but that's where it ends.

BY MR. DuPONT:

Q. A bit greater in the seventies than the eighties. A bit greater in the eighties than the nineties. Is that correct?

A. I have not made a specific analysis decade by decade.

Q. Okay.

A. Other than saying that ten years prior was slight and less.

Q. All right. It's your opinion that 40 to 60 part per million years of benzene exposure will cause AML; is that correct?

A. It's my opinion that people who have had 40 to 60 years part per million exposure are at an increased risk for AML.

Q. How much of an increased risk?

A. It depends upon the exposure.

Q. 40 part per million years, how much of an increased risk?

A. As a rough estimate, 50 to 100

percent increased risk.

Q. That's 1.5 -- a relative risk of 1.5 to 2?

A. That's between 40 and 60 part per million years, yes.

Q. The question was 40 part million years.

A. Well, I'm answering between 40 to 60. I can't give you a precise answer for 40 exactly.

Q. All right. How about for 60, can you give me a precise answer for 60?

A. 40 to 60 would include that range.

Q. 61 part per million years, what's the relative risk of AML?

A. A tiny bit higher than 40 to 60.

Q. So more than two -- relative risk is more than two at 61 part per million years?

A. If it was 1.5 to 2 before, and if you ask me to pick a number, I'll pick 1.6 to 2.1.

Q. How about 80 part per million years --

A. Probably --

Q. -- what's the relative risk of AML causing -- of benzene causing AML at 80 part per million years cumulative risk?

A. Two to threefold. I'm sorry, relative risk of two to three.

Q. Does benzene increase the risk of AML at exposure levels cumulative that are less than 30 to 40 part per million years?

A. Cumulative exposure of less than 30 is not a marker, in my judgment, for increased risk of AML.

Q. Less than 40 part per million year cumulative dose of exposure, does benzene increase the risk for AML?

A. It's not a marker for increased risk at that cumulative exposure.

Q. What do you mean by a marker?

A. The disease, I believe, is not caused by exposure 20 to 30 years ago. I believe I have said it's caused by exposure in the ten years prior to diagnosis. So to

some degree the earlier exposure is a marker of risk, rather than a determinant of risk. People who have enough exposure in the ten years prior to it generally have a long history of exposure because exposure is less now than it used to be. And so that is a marker of risk.

Q. How likely is it that somebody today will get 40 part per million years cumulative dose of exposure in ten years?

A. In the United States it's unlikely.

Q. Is it possible?

A. I suppose if someone chooses to distill pure benzene and inhale it in a suicidal attempt, then it's possible. But in terms of modern work spaces, I would not expect it to occur.

Q. So, in your opinion, no one in modern day America will get benzene induced AML in, let's say, a suicidal manner intentionally inhaled benzene?

MR. THOMPSON: Put it in your own words, Dr. Sarna.

THE WITNESS: The Pukkala article, which was not yet discussed, shows that in mechanics, painters, other professions that have contaminant benzene exposure there's no increased risk of AML.

BY MR. DuPONT:

Q. Doctor, that's not my question.

A. That's my answer.

Q. My question was: In your opinion, the only way that someone in modern day America can get a benzene induced AML, if they intentionally expose themselves to pure benzene?

MR. THOMPSON: Is that the only way, that's what he's asking you.

THE WITNESS: I don't believe that with exposure under current contaminant level conditions that people are getting benzene induced leukemia, not today. Whether there's some way they can get it other than intentionally, I suppose someone could try to poison someone. I can think of

other scenarios, so I won't say that's the only way. But in the modern workplace, the evidence is, there's no increase of leukemia and my expectation is that it doesn't occur.

BY MR. DuPONT:

Q. So according to Dr. Sarna, we will not see any future cases of benzene induced AML in the American workplace without there being intentional exposures?

MR. THOMPSON: Is that what you said, Dr. Sarna?

THE WITNESS: I said that with modern exposure limits, assuming that they are followed and not egregiously done, you will not be seeing it. If there exceptions to that, for whatever reason, you may see it.

BY MR. DuPONT:

Q. Which exposure limits are you referring to, the OSHA permissible exposure limit?

A. OSHA I believe is one par part per million and Stella, five part per million

and that would be consistent, yes. I think that if I recall correctly --

Q. Are the --

MR. THOMPSON: That's okay. You have answered his question.

BY MR. DuPONT:

Q. Are the most up-to-date conclusions of the United States government on the dose of benzene exposure that is creating a risk for leukemia those which are set forth in the OSHA PELs and STELs?

MR. THOMPSON: If you know, Dr. Sarna.

THE WITNESS: I believe NIOSH may have different numbers. Let me double-check. NIOSH has lower numbers than OSHA.

BY MR. DuPONT:

Q. Okay. And NIOSH's numbers are important to consider as well; correct?

MR. THOMPSON: In what regard?

BY MR. DuPONT:

Q. NIOSH's numbers are important to consider as well when we're concerned with

whether or not benzene exposure will cause leukemia; correct?

MR. THOMPSON: If you agree with that.

THE WITNESS: What numbers are chosen to maximize safety are different from what numbers actually cause disease. When you get in an elevator, they tell you you may not have over 2000 pounds, but that doesn't mean if you have 2001 it's going to crash.

MR. DuPONT: Counsel, I think the doctor can determine whether or not he himself agrees with the statement. So if you want to make an objection, you can make an objection. But I object to your interfering with my deposition and suggesting answers to the witness.

MR. THOMPSON: I didn't suggest any answer to him. Where did I suggest an answer to him right there?

MR. DuPONT: Whether you agree

with that or not, okay.

MR. THOMPSON: What's wrong with that?

MR. DuPONT: It's not an objection. It's a speaking objection, it's improper and I would ask you to stop.

MR. THOMPSON: Just go ahead and ask your next question.

BY MR. DuPONT:

Q. What literature sources and bases do you rely upon for your opinion that low dose two part per million year exposures do not increase the risk of leukemia?

A. The literature would be based upon the absence of leukemia increase in modern day in people who have contaminant level exposure, and that's the Pukkala article. The evidence for 40, 60, whatever, would be depending upon which interpretation of the pliofilm article you choose. There are vary -- maybe five or six different interpretations of that data that I think range from 40 to 200 part per million years

as a marker or a risk factor.

Q. I was asking for the name of the article.

A. ^Rinsky is one.

Q. Give me the names of the articles that you rely upon for the position that low dose exposures at two part per million years cumulative dose do not increase the risk of leukemia.

MR. THOMPSON: Just go ahead and give him the names of the articles you recall.

THE WITNESS: The Rinsky article. The Wong analysis of the pliofilm cohort. There are several other analyses of the pliofilm cohort. The Pukkala article. The article by Glass is worth discussion because it is the only one that I know of that claims two part per million as a cause and because there are serious problems with that article that make it not credible.

BY MR. DuPONT:

Q. So Glass 2003, Pukkala, the pliofilm studies are the articles you rely upon for that opinion?

A. Yes.

Q. And those are the articles that you considered for that opinion in this case?

A. Those are the article I brought with me. On all of my opinions I'm considering the body of information I have, which may not be linked to a specific article, but linked to what I have read in general.

I would also add Wong and Rothman's Meta-analysis on petroleum industry. Brown and ^Steinman's various articles on painters. There's a Meta-analysis on diesel exposed workers. All of which show no increased risk of leukemia.

Q. Okay. What is the diesel exposed worker study you're referring to?

A. I don't have the author off the top of my head.

Q. Do you have that article with you?

A. No.

Q. What's your basis for your disagreement with Dr. Shadduck and Harrison on their opinions on general causation of AML and specific causation in David Gerchman?

A. You're going to have to tell me specifically what you want me to opine on.

Q. I don't know, your attorney says you're going to opine on that, so I want to know what you're going to say. If you don't have an opinion on that, then tell me you don't have an opinion on that.

A. In terms of general --

MR. THOMPSON: Hold on a second, Doctor. Take a look at your notes, where you made some notes about Harrison and Shadduck.

MR. DuPONT: Counsel, please do not direct the witness how to answer my question.

MR. THOMPSON: I'm just telling you, he's going to be asked questions on --

MR. DuPONT: No. No. It's not

your place to tell me anything.

MR. THOMPSON: Whatever.

MR. DuPONT: It's not your place to coach the witness how to answer questions. I'm entitled to get an answer from the witness, not an attorney for the witness.

MR. THOMPSON: Fine.

THE WITNESS: Okay. Regarding Dr. Shadduck.

MR. THOMPSON: Andrew, you're ignoring the fact that the guy made 25 pages of notes in preparation for coming here and giving his opinions. And you haven't looked through them once. All right? And so he is going to rely on those notes for his opinions in this case. The facts that he wrote down there were those that he found to be salient, as he told you earlier. So I'm telling you now, he's going to be asked questions about Shadduck and Harrison. He made notations there that were important to

him that he doesn't have committed to memory. So we're going to go through those now so you have a chance to ask the questions about the things he might comment on. So, go ahead, Doctor.

THE WITNESS: In terms of Dr. Shadduck, he cited the threshold for AML was 4.5 to 45 part per million year. That was based on the letter from Shell Oil to EPA regarding the pooled analysis of the Canadian, UK and Australian nested case control studies. And that draft report said, "Preliminary analyses indicate that the risk of MDS was statistically significant at cumulative exposure levels, i.e., 4.5 to less than 45 part per million years, below that previously reported in the literature.

Number one is the preliminary analysis. Number two is unpublished. Number three is not --

BY MR. DuPONT:

Q. Doctor, are you basically going to read your -- however this report was written, are you just going to read this in response to my question about your opinions on Dr. Shadduck and Harrison?

A. Parts of it will be taken from that, parts of it will be elaborated on and expanded.

Do you want me to continue my answer?

Q. All right.

A. It's also --

MR. THOMPSON: Do you want him to keep going?

BY MR. DuPONT:

Q. Is it your understanding that it was Dr. Shadduck's opinion that the threshold is 4.5 part per million years?

MR. CARR: Hang on a second, he's in the middle of an answer again and you've cut him off. So you either need to withdraw the question, make clear on the record that you stopped him, or let him finish his answer.

BY MR. DuPONT:

Q. Doctor, my question is: Is it your understanding that it was Dr. Shadduck's opinion that the threshold for AML being caused by benzene exposure is 4.5 part per million years?

MR. CARR: Okay. I'm going to object to you cutting off the witness again when he's in the middle of an answer. And I want the record to be clear that if you take the position at trial that his concerns with Dr. Shadduck's opinions or Dr. Harrison's opinions were not disclosed to you in this deposition, that he was in the middle of telling you what they were and you stopped him.

BY MR. DuPONT:

Q. Doctor, can you please answer my question? Is it your understanding that it's Dr. Shadduck's opinion that 4.5 part per million years is the lowest dose, or a threshold above which benzene causes AML?

A. In the course of his

deposition, one of the things he cited for exposure was the threshold for AML in 4.5 to 45 ppm per million year range based upon the letter from Shell. That's my understanding. I believe he also cited different data as well.

Q. And is your analysis concerning Dr. Shadduck's opinion based on the letter from Shell to the EPA that which are set forth in these comments and notes?

A. There may be beyond that. I have not read to see whether he has done anything else. I can certainly go over it verbally, but I expect most of it is in the notes.

Q. All right. Well, why don't you read it to yourself and then tell me if there is anything in addition.

A. Comments one and two and three and four on page 60 would encompass my comments on the Shell letter being cited by Dr. Shadduck.

Q. All right. Do you have any other criticism of Dr. Shadduck that is not

set forth in writing in your notes and comments that we have attached as Exhibit 4?

A. My notes and comments would encompass my comments on which would include at times criticisms of Dr. Shadduck's opinions.

Q. Okay. Are your comments and criticisms of Dr. Shadduck's opinions fully set forth in this writing?

A. Fully? They're not have full elaborations, no. They're set forth in principle.

Q. Okay. Is there anything that you want to add that is not written in this notes and comments regarding Dr. Shadduck?

MR. THOMPSON: Remembering that you're going to be asked about whether you agree with their opinions on general and specific causation.

MR. DuPONT: Counsel.

MR. THOMPSON: Andrew, this isn't a federal court report, man. This isn't how we do it in California. We come here and this is the time when

he discloses his opinions.

MR. DuPONT: I don't care what state it's in. I'm entitled to get an answer from the witness, not the attorney.

MR. THOMPSON: That's not an answer. I'm telling you, he's going to be asked that series of questions. I'm not giving him any answer. I'm telling him he's going to be asked to comment on that.

THE WITNESS: I disagree with Dr. Shadduck's conclusion that low dose benzene causes leukemia. And I disagree with the specific causation opinion that Mr. Gerchman's leukemia was caused by benzene exposure in the workplace.

The reasons for that are all elaborated in my notes and in my deposition. But as a blanket answer to the question, do I agree or disagree with this general or specific causation, I disagree with both.

BY MR. DuPONT:

Q. All right. And if I want to know why you disagree with it, it's set forth in this written report and this written notes and comments?

A. Certainly there are additional materials that I haven't documented that bolster the opinions, but the crux of my opinions are in this report. I'm not sure whether I have all the issues about psoriasis in here or not, but I mentioned that before as one of the opinions and I expect we'll get to that.

MR. THOMPSON: And we have been here talking for five hours.

BY MR. DuPONT:

Q. And are you able to add anything, as we sit here today, right now, that bolsters your opinions, as you said, that bolsters your comments and criticisms?

A. On Dr. Shadduck?

Q. Correct.

A. I don't have anything at this point to add.

Q. Now, moving on to Dr. Harrison. Are all of your points of disagreement and criticism with Dr. Harrison's opinions and testimony set forth in this written notes and comments?

MR. THOMPSON: Andrew, let me just clarify. He doesn't have the deposition transcript. Did you have Harrison's deposition transcript?

THE WITNESS: The only thing I had was his handout.

MR. THOMPSON: So he had only his report, so that's all he can comment on at this time.

MR. DuPONT: Okay.

BY MR. DuPONT:

Q. You have criticized and critiqued Dr. Harrison without reading his deposition testimony?

MR. THOMPSON: He hasn't gotten -- we don't have Dr. Harrison's deposition yet, buddy.

MR. DuPONT: I'm asking a question, Barry, okay.

BY MR. DuPONT:

Q. ^am I correct that you criticized and critiqued Dr. Harrison's opinions without reading the deposition testimony?

MR. THOMPSON: That is just such an unfair question, I can't believe it. We don't have his deposition yet.

MR. DuPONT: I didn't suggest you did. Okay. I'm entitled to ask what he did. If you would stop interrupting and stop interfering, I would appreciate it. Stop it.

MR. THOMPSON: Don't answer that question, Dr. Sarna. That's an unfair, ridiculous, argumentative question. He knows we don't have the transcript yet and we have told him you're going to read the transcripts of any witness testifying on causation. So don't answer that question, that's a terrible question.

BY MR. DuPONT:

Q. Doctor, are your critiques and

and criticism of Dr. Harrison and his opinions based only on his written report and not what he stated during his testimony?

A. My written comments in my notes are based upon the roughly 10 page report that I got.

Q. Okay.

MR. THOMPSON: He is going to read the doctor's testimony and comment, just so you know. Because --

BY MR. DuPONT:

Q. And are all of your opinions and criticisms in this written report, as they relate to Dr. Harrison?

A. What I did was, I commented on what was written there. That does not include all my proactive comments, but it includes all my reactive comments.

Q. Okay. Are there any comments or opinions or analyses that you need to add to your review of Dr. Harrison's report and opinions that are not set forth in here, including the basis for them?

MR. THOMPSON: I guess we're

going to go through the same exercise, in that he's going to be asked to comment on, agree with, disagree with, or expand on Dr. Harrison's opinions on general and specific causation. So you are going to be asked that, Dr. Sarna.

THE WITNESS: I expect to see his deposition. I expect I will react to that deposition. There may be things that I agree with or disagree with. There may be things to change what I have written, or there may be things to add to what I've written.

BY MR. DuPONT:

Q. Okay. Based on information you have from Dr. Harrison at this point in time, is there anything you have to add to your opinions, your analysis and your criticism of Dr. Harrison, beyond what's stated in your written notes and comments?

A. My written notes and comments reactive to the handout. It was not a criticism of Dr. Harrison, it was -- it may

be a criticism of his comments, but it was not criticism of him personally.

There may -- that was reactive comments, not proactive comments. And so it's reactively reasonably complete. Proactively there may be things lacking because this is not a formal report. And it's incomplete from the point of view of evaluating his opinions because I haven't seen his deposition.

Q. Do you have anything to add to your analysis, your criticisms or your opinions pertaining to Dr. Harrison's written report?

A. Not at this time.

Q. Or written notes?

A. Not at this time.

Q. Have you reviewed the most recent Tran group studies?

A. I think it's 2010 or 2011.

Q. How many cases of AML were found in the most recent Tran group study?

A. It was either three or four. I believe three.

Q. And have you discussed with any of the other doctors hired by defendants in this case what their views on the Tran group study are?

A. No.

Q. All right. Do you have an opinion that you cannot rule in or rule out de novo AML? First of all, is de novo a cause of AML or is it just the absence of knowledge of what caused AML?

A. Leukemia -- an AML would be considered a de novo in the absence of a known cause.

Q. Okay. So de novo in and of itself is not a cause of leukemia; correct?

A. De novo means it occurred without a known cause. It is not a cause.

Q. Okay. Without a known cause; correct?

A. Yes.

Q. There could be a cause, but we just don't know what it is; correct?

A. Everything has a cause. Most of these presumably are spontaneous

mutations.

Q. But when we talk about de novo and idiopathic we're talking about something that we just don't know what caused it?

A. Idiopathic certainly means we don't know. De novo means it occurred without a known cause. And we can surmise that most of those are spontaneous mutations, but that's a surmisal. There's no way of proving or disproving it.

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(Discussion held off the record.)

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(Whereupon there was a recess in the proceeding from 3:34 p.m. to 3:41 p.m.)

- - -

BY MR. DuPONT:

Q. Doctor, do you hold the opinion that you cannot rule in or out de novo AML?

A. There is no specific test to prove that something is from a spontaneous mutation. De novo simply is a term meaning

you don't know the cause and it appears to be without a cause. So you can rule that in or rule that out by saying there's no known cause.

Q. So your basis -- have you ruled in de novo leukemia?

A. In the absence of a known cause, I can consider this de novo.

Q. Okay. So you're going to tell the jury that it's more likely that David Gerchman's AML arose for no reason whatsoever than it is that his 40 year history of exposure to benzene caused his AML?

A. I'm going to say that David Gerchman's AML falls in the 80 percent of AMLs with no identifiable cause.

Q. And is it your opinion that that 80 percent of AMLs had a 40 year exposure history with benzene?

MR. THOMPSON: Objection, vague and ambiguous.

THE WITNESS: You're asking whether everybody in that 80 percent has had 40 years of benzene? The

answer to that is, no.

BY MR. DuPONT:

Q. Is it your opinion that anyone within those 80 percent of de novo leukemias has a 40 year occupational history of exposure to benzene?

A. There are many, many, many people who work with contaminant level benzene. These people are not immune to de novo leukemia. And I'm sure there are plenty of people who have a history of contaminant level of benzene who have developed de novo leukemia.

Q. So you would include people with a history of almost 40 years of exposure to a known leukemia causing agent --

A. The people who --

MR. THOMPSON: Let him finish his question, Doctor.

BY MR. DuPONT:

Q. Let me finish my question, Doctor.

You would consider people with a 40 year occupational exposure history of

exposure to an agent known to cause AML as being within the 80 percent of individuals who you claim develop AML as a result of no known cause?

MR. THOMPSON: Objection, incomplete hypothetical.

THE WITNESS: The people who have a history of benzene exposure who get de novo leukemia will generally get it at an average age of about 65, and will generally have started having their contaminant level exposure 40 years, 45 years earlier. And they will all have that history. They are not immune to getting de novo leukemia.

If these people all were having leukemia due to benzene, you would see a marked increase in leukemia. But you do not. The studies that I have shown to mechanics show that there is no increase in AML. Therefore, the cases that occur are spontaneous and de novo rather than benzene caused.

These people are not immune to de novo leukemias. They will get it just like anybody else.

BY MR. DuPONT:

Q. But in your opinion you can get leukemia from no known cause, even though you have exposure to a known cause of leukemia? That's your opinion?

A. Of course.

Q. Okay. And it's your opinion that you can get leukemia from no known cause even though you have an occupational exposure for 40 years to a known cause of leukemia?

A. Of course.

Q. And it's your opinion that some of those 80 percent of the individuals with AML that you attribute to no known cause leukemia had 40 year occupational exposure history to benzene?

A. Yes.

Q. Have you now described for me your opinions on de novo AML generally and specifically?

A. Yes. With the understanding

that I have already previously discussed the genetic differences between de novo AML and solvent related AML, and that plays into the -- my overall opinions.

Q. All right. Are there any articles that conclude or state or opine that you cannot rule in or out de novo leukemia as a cause of AML?

A. I know of no article that has a test for de novo leukemia. It's a diagnosis of exclusion. That rules it in or out. You rule it out if you have another cause. So someone who has chemotherapy with Cytoxan at age 18 and two years later gets a 5 minus, 7 minus leukemia, you've ruled out de novo leukemia.

Q. You rule out de novo leukemia if you have a 40 part per million cumulative dose of exposure to benzene?

A. If you had 40 part per million chemo dose exposure to benzene that ended 20 years ago and you have chromosomes that are normal now, I would rule out that benzene, yes.

Q. If you have a 40 part per million cumulative dose of exposure to benzene that ended less than 20 years ago, 20 years from diagnosis, do you rule de novo leukemia as the cause of the AML, you rule that out?

A. No, I don't rule out de novo leukemia. I look at the chromosomes, I look at the exposure in the last 10 years and I would reach a judgment.

Q. Can you opine that somebody's leukemia and AML specifically was caused by benzene exposure solely based on their chromosomes?

A. No.

Q. All right. Your last opinion is that the presence of psoriasis did not increase David Gerchman's dermal absorption of benzene. Is that correct?

A. Yes.

Q. Do you hold that opinion?

A. Yes.

Q. Let me back up. I don't think I got an answer. Are there any articles that

conclude or opine that you cannot rule in or out de novo AML as a cause of AML?

A. Every article that has an article about AML with a known cause opines inferentially that de novo leukemia was ruled out in that particular case.

Q. Well, inferentially, that's your interpretation of the article. Are there any articles where the authors have written that you can or you cannot rule in or out de novo AML?

A. I have no -- know of no article that says, in this case of leukemia which was caused by Cytoxan, we have ruled out de novo AML.

Q. Has anyone written an article that says that there is a method for ruling in or out de novo leukemia as a cause of AML?

A. I don't know whether someone has said that it's a diagnosis of exclusion, but it's relatively obvious.

Q. Is there any article that you can point me to that shares the opinion that you cannot rule in or out de novo AML?

A. I have answered the question.

Q. Can you cite for me an article that says you can or cannot rule in or out de novo AML?

A. I know of no specific article to tell you.

Q. Okay. Presence of psoriasis did not increase dermal absorption. What's your basis for that opinion?

A. I think it likely that his psoriasis was plaque psoriasis, chronic plaque. I don't have pictures. But the fact that he was self managing and presumably able to have skin exposed to solvents or chemicals argues against having ulcers or other types of psoriasis.

Assuming it is chronic plaque, that is thickened skin, which in principle should be a variable to absorption rather than. There are studies that have looked at absorption of steroids and salicylates when applied topically to psoriasis and shown no increased absorption or evidence of substantial absorption. And it is relevant

that there is a long history of treating people with psoriasis topically with coal tar, which has contaminant level benzene in it. And that coal tar is not known or felt to be a carcinogen or leukemogen and is still approved by FDA for use in psoriasis.

Q. And how long has coal tar been approved by the FDA for use in psoriasis?

A. I don't know. 30, 40 years maybe.

Q. And is it still approved by the FDA for use in psoriasis?

A. As far as I know, yes.

Q. Do doctors still prescribe coal tar for use in treating psoriasis?

A. I'm not a dermatologist, I can't answer how often it's still prescribed.

Q. I didn't ask how often, I just asked is it still prescribed?

A. To my knowledge, the answer is yes, but I'm not a dermatologist.

Q. Have you looked at any articles that addressed the rate or amount or manner of benzene exposure through the skin in

individuals with psoriasis?

A. No. I have not been able to find such articles.

Q. What articles do you rely upon for your opinions that the presence of psoriasis did not increase dermal exposure of David Gerchman to benzene?

A. I have the article by Wester, which was previously discussed, that talks about plaque having very little effect, but some, on water loss, which is not the same as absorption. In terms of absorption, there is a -- sorry, the Wester article talks about -- I misspoke. The Wester talks about absorption of hydrocortisone and there is no difference in absorption when they applied hydrocortisone to a psoriatic area versus a normal area.

And there is an article I have only in an abstract by Gip that talks about absorption of betamethasone and salicylic acid and found that there was no change in plasma cortisol levels, which argues against any absorption of betamethasone. And there

was no detectable salicylate levels in the plasma, which tells you that that was not absorbed. So those are the two articles.

Q. Is it your opinion that hydrocortisone and benzene are absorbed through the human skin the same way and at the same rate?

A. I don't know whether that's true or false. I have no data to answer that question.

Q. Is it your opinion that benzene and betamethasone are absorbed through the human skin in the same way and at the same rate?

A. I have no data to answer that question.

Q. Is it your opinion that salicylic --

A. Salicylic, like aspirin, but it's not acetylsalicylic acid, it's just salicylic acid.

Q. Okay. Salicylic acid, is it your opinion that salicylic acid and benzene are absorbed through the human skin in the

same way and at the same rate?

A. I have no data to answer that question.

Q. Have you seen any testimony that there were, in fact, open sores on David Gerchman's skin from his psoriasis --

A. No.

Q. -- or is it your assumption -- I'm sorry, you have not seen that testimony?

A. I have not seen testimony about open sores. I have seen testimony about discolored lesions which sound like plaque, but I have not seen open sores.

Q. If there were open sores on David Gerchman's skin from psoriasis, would that change your opinion that the psoriasis did not increase his dermal absorption of benzene?

A. If there were open, weeping ulcers, what I said would not apply. That would not mean that absorption was increased. It would mean that it would not apply.

Q. If there were open sores on David Gerchman's skin from psoriasis, would

his dermal absorption to benzene have been increased?

A. Unknown.

Q. Unknown?

A. Unknown to me.

Q. Okay. Do you agree that benzene has the same capability of causing AML if it is absorbed through the human skin as it does as if it's inhaled through the lungs?

MR. THOMPSON: Hold on a second. Objection, form and foundation.

THE WITNESS: I think if a benzene molecule gets to the bone marrow, it doesn't matter how it was absorbed. Whether or not there is a different handling of benzene that goes -- comes from the skin, putatively absorbed from the skin as opposed to benzene that's inhaled, I don't know.

BY MR. DuPONT:

Q. Okay. Will you take your articles on psoriasis and we will mark those

as Exhibit 11, I think. Triebig should have been 10. I want to mark your latency article, if it's not already marked, as Exhibit 10.

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(Whereupon the documents were marked, for identification purposes, as Sarna Exhibit Numbers 10 and 11.)

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BY MR. DuPONT:

Q. Exhibit Number 11 should be the Wester and Gip, which are all the articles that you relied upon and considered for your opinions that the presence of psoriasis did not increase the dermal absorption of benzene by David Gerchman. Is that fair?

A. The articles, yes, but I did comment on the coal tar issue.

Q. Have you brought any articles with you that we have not marked as exhibits?

A. Yes, but I'm not sure they're relevant. Some of them were dealing with the comments of your other doctors. Some of them were dealing with the issue of low level

exposure. There is Hayes from the CAPM study. There's the Collins article. There's some articles on autoimmune diseases, which I guess relate to psoriasis that I can pull. There's an IOM report by Miller and a letter by Miller regarding the three case controlled studies which are going to be nested on dose. There's some articles that --

Q. Let me ask you this: Have we marked as exhibits all the articles you are relying upon for your opinions?

A. Yes. But some of them are relevant to the comments on Dr. Shadduck or Dr. Harrison in terms of dose response. So some of these are issues in terms of the question of dose level. And I'm not citing them to support me, but if someone cites them to reach another conclusion I have them for discussion.

Q. Okay.

A. So I'm not citing CAPM data, but if someone wants to discuss why CAPM data is not a valid tool to say that low level benzene exposure causes leukemia, I have such

data available.

Q. All right. So what articles did you bring with you on the CAPM issue and what are your opinions on the CAPM study?

A. I brought with me Hayes, but the opinions depends upon more than Hayes. The Hayes article would claim, I think, less than 10 part per million year and it has an increased risk. But the Hayes article in the control group does not consider leukemia NOS, or not otherwise specified. And that makes the control group very uncertain. And the numbers are small enough that that could make a difference. And the Hayes group is not properly controlled for age. When you take a look at Yin's article about the breakdown of CAPM, those people who are over 30 and entry on the study make up 16 percent of the exposed group rather than 10 percent of the control group. So one has to correct for that, not just the size of the control group in general and the size of the overall group. And that means that the overall risk factors that they come up with are inflated. And how

those risk factors fit into the dose dependent risk is uncertain.

MR. CARR: Andrew, this is Carr, just for the record. Some of the studies that he brought with him have some relevance to Glass and some other issues. I do intend to ask him questions about those studies in this case. I would get copies --

BY MR. DuPONT:

Q. Have we exhausted your comments on the CAPM data? And for CAPM, we're going to abbreviate that C-A-P-M, and that stands for the Chinese Academy of Preventative Medicine.

A. Yes. It's actually CAPM NCI, but that's fine.

Q. Right. Have you given us all of your analysis on the CAPM data that you find relevant to this case?

A. Well, certainly I have not given the details, but I have given you the framework.

Q. Okay. Where do we find the

details?

A. I would have to generate them.

Q. Can you tell me what the details are now?

A. No. Because they are the specific numbers. Taking a look at the specific numbers of people in each group with specific odds ratios they came to. What the odds ratio is grossly, just taking 75,000 and 35,000, or whatever the numbers are for the two groups. How they allegedly controlled for age, but came out with a higher odds ratio rather than a lower one. Looking at the age distribution of the leukemic patients that are described by Travis in a different population, where something like 80 percent of them were in their forties or older, meaning that they were entered in the cohort in their thirties. And that's where the leukemia linked. So it would require going through those details, which I don't have in front of me. But that's the framework.

Q. Okay. So anything beyond that, you're not able to provide me at this time?

A. I have told you those -- I don't have the details to provide you at this time. I have the framework, which I have provided you.

Q. Okay. And do you have all the articles with you that you used for your analysis on the --

A. No, I didn't bring every CAPM article with me. You have to go to the Travis article, and one of the Yin articles. You have to break down by age. And the Travis article, to get the break down of the age of the leukemic patients, they weren't analyzed. It's not all in one article. You have to look widely.

Q. So I can't discuss those articles with you in detail today?

A. I did not provide a detailed part of that. Again, that's more of a rebuttal issue than a proactive issue. I don't claim to support that, it's not a proactive statement that tells me that the risk is only at high levels. It is a reactive statement that tells me if your

experts argue the CAPM data, and I did not see that argued by them in their deposition or sheets. So I did not provide that rebuttal data. But should they make that argument beyond that, I have those comments.

Q. All right. What are the comments you can give me today on the Health Watch study series?

A. Glass in 2003 and then 2005 came out with a nested case control of something like 32 or 33 leukemics. Something like 11 of which were AML. As to relative risk function of exposure, it came to the conclusion that at two parts per million exposure and higher there is increased risk.

At face value, when you look at how many people have that exposure, this would suggest that the relative risk as compared to a control group, SIR or SMR, should be roughly three. But the Gunn article shows that there's no increase in leukemia and in AML there's no increase in SMR or SIR in leukemia. So that at face value doesn't make any sense.

Then when you go on to look at how many people who got leukemia had all time high exposure with greater than 32 part per million years, an exposure of concentrated benzene, they had -- were in the highest exposure group, and the odds ratio was something like eight or nine to one or higher. And there was something like nine patients in that group where one was expected.

That means in the lower group, which is everything below the high exposure group, there was 23 patients expected. And based upon the odds ratios, that was double or triple what you expected, but that has to be less than what you expected because there was no increased risk.

So the whole thing makes no sense unless the control group, which is low exposure, had much fewer cases.

Apparently in the Millers article, IOM report of the plans for the pooled article between the Canadian, UK and Australian case control studies, the authors

of the Australian study, which I presume are Deborah Glass and others, admit there's a problem with their data and it is uncertain.

Q. Is it your understanding that the Gunn study that you referred to and the Glass 2005 were nested case control studies, the same nested case control study as the Glass 2003?

A. Gunn is a cohort study. I never said it was a nested case control. It gives you SMR and SIR. It's the same Health Watch population, but it's the cohort study.

Q. All right. And the Glass 2005, the cohort study, is a nested case control study?

A. Nested case control. The major publication is 2003, but in 2005, they go into the high exposure group.

Q. Have you seen any data or information concerning the study that combined the three petroleum distribution cohorts --

A. Only the Shell Oil letter --

Q. I haven't finished my question.

-- beyond the letter that Shell sent to the EPA reporting statistically significant increased risks of MDS at cumulative doses of 4.5 to 45 part per million years? Have you seen any data or commentary for inclusions from that study beyond the letter that was sent to the EPA?

A. No. Although I will comment that had there been a low risk of leukemia, I would have expected that they would have been duty bound to report that. And the absence of such a statement makes me wonder whether no such finding occurred.

Q. Do you know if they looked at the risk of leukemia?

A. Well, the original case controls, as far as I know, looked only at leukemia and didn't look at MDS. So I was surprised to see information come out on MDS. If they did not look at leukemia, they failed the intent of pulling the data.

Q. Okay. So my question to you was, do you know if they looked at leukemia?

A. I know they were supposed to

look at leukemia. I don't know whether they did what they were supposed to do.

Q. Okay. So you don't know if they looked at leukemia; correct?

A. As a matter of fact I do not know. As a matter of conjecture, I would be shocked if they didn't do it.

Q. Have you seen any document that sets forth the intent or the goal or the design of that study?

A. Yes.

Q. What document is that?

A. An article by Miller I believe talks about that.

Q. So you believe Miller's IOM report sets forth the objectives and the study design of that combined cohort study that Shell reported to the EPA?

A. There was an article by Miller separate from the IOM report too that was shorter.

Q. Okay. So which article do you contend provided the goal and the design for the combined petroleum distribution marker

cohort study that was reported to Shell?

A. There's more than one, but the information I have now is published, Miller, et al., International Archives, Occupational and Environmental Health, 2010, Volume 83, page 69 through 76.

Q. All right. Let's make that the next exhibit, please.

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(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 12.)

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THE WITNESS: This was talking about the exposure assessments, which were a prelude to that. There may be another article that talks about the plan that I don't have with me. But this talks about the visits to the assure exposure measurements were the same in preparation for that study.

Let me take a look at the IOM report.

(Reviewing document.)

The IOM report talks about the advantages of a pooled analysis. It talks about how it should be done and talks about pathology review. I'm not sure it exactly sets out what it is, but it's preliminary to it and fairly talks about leukemia.

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(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 13.)

- - -

BY MR. DuPONT:

Q. Is it your opinion that Exhibit 13, the IOM report, sets forth the study design and objectives for the combined case control study that Shell reported to the EPA?

A. The IOM report audited the three in preparation for the study.

Q. So it does not set forth the plan or the design of the objectives?

A. I don't see a clear distinct plan. I believe there is another article,

which I did not bring, that has that. But the IOM report and the Miller report elsewhere talk about preparation for this article rather than the specific study.

Q. Can you identify for me any other article in which you believe the plan and the objective and the goal of the combined case control study of petroleum distribution workers at Shell published to the EPA has set forth?

A. I can tell you I believe there is such an article, but I can't give you the details of it now.

Q. Are there any other articles that you brought with you that are relevant to your opinions in this case, including your analysis of Dr. Shadduck, Dr. Harrison and Mr. Petty, that we have not marked as exhibits?

A. Did you want the Glass exhibit marked?

Q. Glass 2003?

A. Yeah.

Q. Okay.

A. And do you want Glass 2005 marked?

Q. Might as well.

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(Whereupon the documents were marked, for identification purposes, as Sarna Exhibit Numbers 14 and 15.)

- - -

THE WITNESS: And did you want Hayes marked?

BY MR. DuPONT:

Q. Yes.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 16.)

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THE WITNESS: The other issue as long as I'm -- well, one of the issues that we have not discussed that you asked about my comments on your other experts would be the issue of hematoxicity at low dose benzene. These were cited as some as if they

indicated a low dose benzene caused leukemia. There were -- article by Ward that was cited I think by Dr. Harrison in this regard. Ward was the pliofilm study and did show that people with low exposure may have some changes in their blood counts. But those people did not get leukemia.

We know from the pliofilm study that however you want to use the metric, at least it was 40 part per million years, if not 200 part per million years, that in that study the threshold -- so, yes, there was hematologic toxicity, but that doesn't mean that there was leukemogenesis. That's a totally separate issue. One does not relate to the other.

And there was articles by I think Land, which were quoted as showing hematoxicity at low levels of benzene. And I would just point out that those were in the later article by Land, found to be dependent upon

specific genotypes. And this is limited to people who are Chinese. So how that applies here is unclear.

And, again, that doesn't mean that there was an increased risk of leukemia. If you choose to use abnormal blood counts as a marker of benzene exposure, it should be noted that there was no abnormal blood count in June of 2007 when Mr. Gerchman had a blood count.

Then there was something else that I wanted to discuss. Yes. The Iron study was quoted as evidence that only 24 percent of patients with benzene associated -- benzene caused leukemias have chromosomal abnormalities. This was, one, myelodysplasia, not leukemia. Two, the people with high benzene exposure had abnormal eosinophil precursors, which are not normally found. And if that is a unique syndrome to China with a specific genetic abnormality

that doesn't show up on a karyotype, that's fine. If I see someone with MDS, and abnormal eosinophils, I would not think that abnormal karyotype in that particular syndrome is necessary. But that syndrome is not relevant here.

The people with lower exposure may or may not have benzene caused MDS. We have no information on whether this population was at increased risk or not. So as we discussed before, people who have been exposed to benzene don't mean that their leukemia was caused by benzene. But let me take my --

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(Discussion held off the record.)

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BY MR. DuPONT:

Q. Are all of the studies that you rely upon for your own opinions, as opposed to your analysis of the opinions of Dr.

Shadduck and Dr. Harrison, the ones you already identified for us when I asked you about your opinions? Are the remaining articles that you have with you those which are -- those that are identified in your critique of Dr. Harrison and Dr. Shadduck?

A. There's the Collins article, which deals with peak exposure, so I don't know whether you want to consider that. I have the Gunn article which you haven't marked. I don't know whether you want to mark Gunn.

Q. Can you answer my question, please. Are the articles that you have left in your stack that we have not marked as exhibits, articles that you have brought because of your critique of Doctors Harrison and Shadduck, or articles that you are relying upon for your primary opinions in the case?

A. I would say Collins would be supportive of my primary opinions since it showed little increased risk of leukemia with cumulative dose.

MR. CARR: Dr. Sarna, and I hate to interrupt. I think he's asking you a more basic question, and that is, you have a stack left. Do you either rely on everything in the stack for the criticisms of his experts or are you relying on it in your opinions in this case? He just wants to know whether --

THE WITNESS: Criticism of your experts.

MR. CARR: Okay. For everything in the --

BY MR. DuPONT:

Q. And those criticisms and your analysis of those articles are set forth in your notes and comments that we have marked as Exhibit 4; correct?

A. If -- my expectation is that the articles that I cited and for my position are in the notes and comments. Again, I will report, this was not a report and thus it is not complete with every article that supports my opinion. There are general articles that

may support my opinion that I have not provided. But these are the ones that I felt were appropriate to provide with my notes. But it is not a report.

MR. CARR: Andrew, can I take a shot at this?

BY MR. DuPONT:

Q. All I want to know, Doctor, is: Have you told me about all the articles that you're relying upon in support of your primary opinions in this case?

A. My best answer is, yes. I'm not sure that every article --

Q. Okay. Thank you.

MR. CARR: Wait a minute, you cut him off.

MR. THOMPSON: He's got a qualifier there, sorry.

THE WITNESS: I'm not sure that every article has been provided you and there are some times that I have discussed things that I don't have the reference for, but are in my general knowledge. But I have discussed them.

BY MR. DuPONT:

Q. All right. To the extent that you have been able to identify an article that has not been provided, you have identified it for me; correct?

A. Yes.

MR. THOMPSON: Other than that he has a whole stack right here, dude.

MR. DuPONT: No, no, no, no.

MR. THOMPSON: These are articles you looked at. What do you mean, no? You know, Andrew, the problem is, like I said, you're in Philly and we are here. And he has a stack of stuff --

MR. DuPONT: The problem is I have spent five and a half -- five hours and 45 minutes going through the Doctor's opinion, having the doctor identify what he relies upon for those opinions. And now I have an attorney saying, oh, by the way, let's add these other articles which the doctor on his couldn't identify and now I as

the attorney want to identify and help him. That's the problem.

MR. THOMPSON: That's just a bunch of crap.

MR. DuPONT: That's why I'm saying that --

MR. THOMPSON: If you were in the room with us, which most lawyers are for most depositions, you would see the stack that's sitting to his immediate right and you could ask him. The complication is, you're trying to take this remotely, man. And he's also got six boxes --

MR. DuPONT: That's not a complication. He's already given his opinions.

MR. THOMPSON: It is.

MR. DuPONT: Now you're changing them.

MR. THOMPSON: It is a complication. They're sitting right here. Do you want him to identify them or not?

MR. DuPONT: He just answered the question. He just answered the question again.

MR. THOMPSON: All right. Well, I'm telling you, there's a stack of articles right here that you need to go through if you want to fully explore the doctor's opinions. There's also six boxes of stuff behind him that he's read and you're not touching any of that, so do whatever you want to do. But I'm telling you right now, we're prepared to continue to let you ask questions. If you don't want to, no problem. Go for it.

BY MR. DuPONT:

Q. Doctor, you have identified for me and marked as exhibits all of the documents and articles that you have relied upon for your opinions, your primary opinions in this case --

MR. CARR: No, because he's got --

BY MR. DuPONT:

Q. As opposed to your analysis of the plaintiffs' experts; correct?

A. I can't limit myself to the things that I brought because this is not a formal report. And there are articles that are in my reasonable background. I have told you about a bunch of CAPM articles that I have not done. There may be other articles in other areas that I have not brought. So I don't want to be limited to the articles that I have brought. But I brought those articles that support that. I can tell you that these --

Q. And to the extent that the --

MR. CARR: I'm sorry, I'm not going to allow you to cut this one off. Doctor, please finish your answer.

BY MR. DuPONT:

Q. To the extent --

MR. CARR: Doctor, please finish your answer.

BY MR. DuPONT:

Q. To the extent that you --

MR. CARR: The witness is talking, Andrew.

THE WITNESS: Of the pile that I have, I believe I have given you the ones that I would consider proactive rather than reactive. However, I would reserve the right to be reactive with the others. And I have brought --

BY MR. DuPONT:

Q. That's what I'm trying to sort out.

A. I have not brought all the articles that would be in the background of my opinions.

MR. THOMPSON: You also --

BY MR. DuPONT:

Q. That's what I'm trying to sort out. To the extent there's an article that supports and you rely upon for your opinions, your primary opinions, your affirmative opinion, we have marked them as exhibits. Or if you haven't brought it with you, you have identified it to the best of your ability;

correct?

A. May I have that again, please?

Q. To the extent that there is an article that you rely upon for support of your affirmative opinion, this would be your critique of the plaintiffs' experts, we have marked those as exhibits. Or to the extent you can identify the article and have not brought it with you, you have identified it for me to the best of your ability. Correct?

A. Yes. But I reserve the right to have other articles, which are the basis of my opinion that I have not presented to you because I have not filed a formal report. I have talked generally, I have done my best to quote the articles as specifically as I can. But there may be things that I have missed and if I'm able to identify them and they're part of the body of literature and they're a background to my opinion, I would want to be able to bring them forth.

Q. You understand that this is my opportunity to ask you what those articles are.

A. And I'm doing my best to answer. That's why I brought the pile and that's why I have answered the best that I can.

Q. Okay. And what you have with you in your pile of articles that has not yet been marked as an exhibit, or you did not identify when I asked you what your primary opinions are, that pile of articles are articles that you're using to address the plaintiffs' expert's opinion?

A. They would be reactive to the plaintiffs' opinion or to issues that could be brought up whether the plaintiffs' experts brought them up or not.

Q. Okay. And are your comments on those articles that you have brought with you and have not been marked yet, and what you are terming reactive or reactive opinions, are your comments on those articles set forth in your written notes and comments?

A. Not entirely, no.

Q. How many articles do you have left that we haven't discussed?

A. We have discussed Gunn, but it has not been made an exhibit. I have two articles that Dr. Williams quoted. Or one article that she quoted regarding psoriasis by ^Godelli. I've got a couple of articles on autoimmune disease related to psoriasis that I don't need. I've got statements as to OSHA and NIOSH levels, which are not important.

I've got an NPM1^ article, which is reactive to a comment by Dr. Shadduck, but not of critical value. It's just I disagree with him about an unrelated point.

I've got the original case control study by Rushton and by Schnatter. That would be the two other nested case control studies that will be part of the pooled analysis.

I've got Trangch^ report by Talbot. I've got ^Wynell report, which is reactive to a comment I think by Dr. Harrison. I've got the Zheng article, which is relevant to the psoriasis I believe and

was quoted by one of your doctors regarding obesity, but it doesn't talk about obesity.

I've got an article about DNA damage, DNA repair, which isn't directly related to it. But it talks about why you may have increased risk of leukemia with age because of decrease in DNA repair. I've got a ^Nielson article which I pulled because I thought that was the article your author went -- Dr. Harrison was referring to, but it wasn't. So it's not relevant. I've got another copy of Rushton. I've got Bonds because your author -- one of your experts pulled it, I think Harrison. I've got Klu^ because I think Harrison quote that in terms of effects on the --the only one that is worth discussing at least briefly would be Collins. Dr. Petty I think talked about peak levels that were low, but what he called peak. And I just pointed out that the only relationship that Collins found with peak levels was greater than 100 parts per million times 40 days, which certainly doesn't apply here.

Q. Okay. So out of those articles you just went through, the only ones you feel worth commenting on is Collins?

A. Right. I also got Irons in my pile. Another article that was quoted regarding psoriasis, but not relevant, and the Shell letter. And Dr. Harrison's notes. That's what I've got.

Q. Okay. The Irons study is the study you already told me about. Again, in the 24 percent of patients with benzene?

A. Yes. Those haven't been admitted, but some of them have discussed. The only one that hadn't been discussed that I would consider relevant was Collins, which I just told you about.

Q. So the ones you consider relevant are Ward, Lan, Irons and Collins?

A. Yes. But Ward is -- Ward is relevant, Lan -- yes, those are relevant.

Q. Okay. Let's mark Ward. And you have given me your comments on Ward; correct?

A. I have given you my comments on

Ward, yes. And two articles by Lan.

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(Whereupon the documents were marked, for identification purposes, as Sarna Exhibit Numbers 18, 19, 20 and 21.)

- - -

THE WITNESS: I don't know whether we ever marked the Martyn Smith article as one. I don't know whether you want that marked.

BY MR. DuPONT:

Q. All right, we'll mark Martyn Smith's article as 22. And you have given me your comments on that; correct?

A. Yes.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 22.)

- - -

BY MR. DuPONT:

Q. Does that exhaust the list of articles that you think are relevant and

worthy of discussing?

A. You had Triebig, we gave you that, I got a second copy of that, yes, in my pile. That would be it.

Q. The Triebig we'll make sure that's been marked, and if it hasn't been marked already we'll mark it.

Are we now done with the articles in your comments and articles that you believe are relevant and worthy of discussing?

A. Certainly we're done with the articles that I have identified to bring with me.

Q. Okay. How long does the hematotoxic effect of benzene exposure in the blood remain? In other words, how long do blood counts remain depressed after benzene exposure?

A. I don't have an answer from the literature on that. My expectation is that it lasts no more than several days to a week or two, but I don't know that.

Q. All right. You don't know one

way or the other?

A. I have not seen a study that defines it.

Q. All right. Those are all the questions I have.

I'm sorry, the boxes of documents that you have, are those discovery materials? In other words, deposition transcripts and documents from the defendants?

A. Largely they're medical records and transcripts. There are some gas chromatographs which I frankly didn't look at. And there are some benzene content measurements for various products that are there. Certificates of Analysis, and what I gleaned from them is in my written report. But those are there.

Q. Have all of your observations based on the defendant documents, I'll call them, the documents that are not transcripts and not medical records and not the articles that we have been going through, have all of your observations based on the defendant

documents been set forth in your notes and comments?

A. Well, I think the -- I did not mention when you asked me about my qualitative view of exposure, that the Certificates of Analysis showed low levels of contaminant level benzene. And that would be supportive that my exposure was -- that exposure particularly in the last 10 years was trivial.

Q. Do you have separated the documents from the defendants that you have reviewed and considered in coming to your opinion?

A. Have separated as to how or what?

Q. In other words, do you have the documents that you have reviewed and relied upon in coming to your opinions that have come from the defendants, that are not transcripts and are not medical records and are not articles, do you have them in one location, one folder, one stack that we can easily identify?

A. No. They're scattered to the winds, as all of my articles were. I got six boxes that are full of stuff in no particular order.

Q. Okay. To the extent that you considered information from the documents provided to you by the defendants, have you identified those documents in your report?

A. My document, as I mentioned, was notes with comments. And I took notes as I evaluated things. So if they were important to me or relevant that I thought they should have been put on paper they're in my notes. A lot of the things are not important, but were just relevant as note taking as I reviewed things.

Q. Okay. I'm not really concerned about whether we characterize Exhibit 4 as your report or your notes and comments. I just want to make sure that if you reviewed and relied upon and found important a document from one of the defendants, did you identify that document in your notes and comments?

A. Yes.

Q. Do you have any notes that you have made concerning this case that are not set forth in your notes and comments?

A. Again, I didn't -- I've quoted to you some articles that may not be listed in my notes. So those I have discussed with you, but they may not be in my notes. Other than that -- and articles that I have not been able to identify, my answer would be, no.

Q. Okay. So there's no other document where you have written down observations or made notes as you were going through transcripts or other documents?

A. I have some handwritten notes behind me that are chicken scratch and illegible and largely led to them being in my report if they were relevant. I think the accurate answer is that it would be a waste of your time to go through them. But if you want to try to decipher my handwriting, you're welcome to.

Q. Well, let's attach them as an

exhibit anyway.

- - -

(Whereupon the document was marked, for identification purposes, as Sarna Exhibit Number 23.)

- - -

BY MR. DuPONT:

Q. Have those handwritten notes been transcribed into your notes and comments that's Exhibit 4?

A. Anything that's important, yes.

Q. Those are all the questions I have.

(Discussion held off the record.)

MR. THOMPSON: I propose that we stipulate to relieve the court reporter of duties and responsibilities under the California Code.

The original of the transcript will be sent to Dr. Sarna as early as possible, early next week, Monday or Tuesday is the target. Dr. Sarna will

have until the 9th of August to read and sign, and we'll alert counsel by letter or notification of any changes that are made.

If either that does not happen or the transcript is unavailable at the time of trial, a certified copy can be used for any and all purposes.

MR. CARR: So stipulated.

MS. DIPPONG: So stipulated.

MR. DuPONT: So stipulated.

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