

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

- - -

DAVID GERCHMAN and
THERESA GERCHMAN,
Plaintiffs,

vs.

BERRYMAN PRODUCTS, INC.;
CRC INDUSTRIES, INC.,
which will do business
in California as
Pennsylvania CRC
INDUSTRIES, INC.;
(Continued)

: CASE NUMBER
: VC 060 106
:

: Assigned to:
: Hon. Raul A.
: Sahagun

: Dept. SE "F"

: Complaint
: Filed: December
: 12, 2012
:

- - -

Thursday, July 26, 2012

- - -

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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Philadelphia, Pennsylvania 19103

1 Continued:

2 FORD MOTOR COMPANY; :
3 LOCTITE CORPORATION :
4 n/k/a HENKEL CORPORATION :
5 ILLINOIS TOOL WORKS, INC. :
6 Solely as successor-in-interest :
7 to Permatex, Inc. :
8 RADIATOR SPECIALTY COMPANY :
9 UNITED STATES STEEL CORPORATION :
10 SAFETY-KLEEN CORP. :
11 SAFETY-KLEEN SYSTEMS, INC. :
12 3M COMPANY :
13 E.I. DUPONT DE NEMOURS & COMPANY :
14 SEA FOAM SALES COMPANY :
15 JUSTICE BROTHERS, INC. :
16 HANSON MERRILL CORPORATION :
17 a/k/a ERNIE'S AUTO PARTS :
18 HENKEL CORPORATION :
19 Individually and as :
20 Successor-in-Interest to :
21 LOCTITE CORPORATION and :
22 HENKEL LOCTITE CORPORATION :
23 and DOES I THROUGH 100 :
24 Inclusive :
25 Defendants :

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I N D E X

- - -

Testimony of: Gregory Sarna

By Mr. DuPont.....

- - -

E X H I B I T S

- - -

EXHIBIT NUMBER	DESCRIPTION	PAGE MARKED
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Sarna-1	Invoices of Dr. Sarna	
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Sarna-2	E-mail from Mr. Skaar To Dr. Sarna	
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Sarna-3		
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Sarna-4	Notes and Comments of Dr. Sarna	
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Sarna-5	Scientific Articles	
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Sarna-6	Article - Chelghoum, Kane	
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Sarna-7	Article - Scarselli	
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Sarna-8	Articles - Pukkala, Ji, Guo, Costantini, Hotz, McLean	
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Sarna-9	Articles - Pedersen-Bjergaard, Mauritzson, Sonali Smith, Martyn Smith, Mitelman, Brownson, Doll, Adami, Thomas, Chelghoum, Kane, Sandler	
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1	Exhibits (continued):		
2	EXHIBIT NUMBER	DESCRIPTION	PAGE MARKED
3	Sarna-10	Article - Triebig, McLean	
4	Sarna-11	Article - Wester and Abstract	
5	Sarna-12	Article - Miller	
6	Sarna-13	Research Report, Miller	
7		Article - Williams	
8	Sarna-14	Article - Glass - Health Watch	
9		Exposure Estimates	
10	Sarna-15	Article - Glass - Leukemia Risk	
11	Sarna-16	Article - Hayes	
12	Sarna-17	Article - Ward	
13	Sarna-18	Article - Qing Lan, Polymorphisms	
14	Sarna-19	Article - Qing Lan, Hematotoxicity	
15		In Workers Exposed to Low Levels	
16		Of Benzene	
17	Sarna-20	Chemico-Biological Interactions	
18	Sarna-21	Article - Collins	
19	Sarna-22	Article, Martyn Smith	
20	Sarna-23	Legal pad of handwritten notes	
21			
22			
23			
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1	DEPOSITION SUPPORT INDEX		
2	DIRECTION TO WITNESS NOT TO ANSWER		
3	Page	Line	
4			
5	REQUEST FOR PRODUCTION OF DOCUMENTS		
6	Page	Line	Description
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9	STIPULATIONS		
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1 GREGORY PAUL SARNA, M.D. - ROUGH

2 - - -

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

6 - - -

7 BY MR. DUPONT:

8 Q. Good morning, Doctor. How are
9 you?

10 A. I'm doing well, thank you.

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

14 A. I understand that, yes.

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

23 A. Yes, I will.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 me if you think I'm going off track. But
3 it's usually not black and white when an
4 answer stops and when explaining an answer
5 goes on.

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

8 A. I never got a Notice of
9 Deposition personally.

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 would not limit myself to these. I generally
3 would say that these reinforce the body of
4 knowledge and will be able to give me precise
5 data in terms of that body of knowledge. But
6 I will be answering based upon my general
7 knowledge, not limited to these reports.

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

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

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

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

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

25 A. I have copies of that, yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. Did you bring your references?

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

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

9 A. Pretty much.

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

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

15 Q. Okay. I have a copy that was
16 provided to me earlier in the case. The most
17 recent publication I see on it appears to be
18 an abstract, and it looks like it is dated
19 2011. The primary author was Jahanzeb,
20 ^J-A-H-A-N-Z-E-B?

21 A. That would be a current CV.

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

25 A. To my knowledge, yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

25 A. Yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. which conferences were they?

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

12 Q. Have you spoken at Defense
13 Research Institute conferences?

14 A. Defense Research Institute?
15 No.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 records for my review. And that was based
3 upon a phone call that day, and the medical
4 records were by e-mail.

5 Then there -- in terms of
6 billing, on May 4th there was a bill for
7 \$7,375. On June 9th there was a bill for
8 \$3,250. On July 4th, there was a bill for
9 \$3,625.

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

12 A. \$500 an hour.

13 Q. what is your rate for
14 deposition testimony?

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

18 Q. And which rate are you charging
19 today?

20 A. I'm away from my office.

21 Q. All right. Is that something
22 new?

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

25 Q. So since 2009, you have charged

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 \$1,000 an hour for a deposition away from
3 your office?

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

12 Q. Okay. But that wasn't my
13 question. My question was, is it your
14 testimony that since 2009 you have charged
15 \$1,000 per hour for depositions outside of
16 your office?

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 please.

3 - - -
4 (Whereupon the documents were
5 marked, for identification purposes,
6 as Sarna Exhibit Number 1.)

7 - - -

8 BY MR. DUPONT:

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

12 - - -

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

16 - - -

17 BY MR. DUPONT:

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

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

22 THE WITNESS: Probably not
23 engagement, but good enough.

24 BY MR. DUPONT:

25 Q. Do you have a separate

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 correspondence that's your engagement in this
3 case?

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

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

10 A. No.

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

13 A. Yes.

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

16 A. When he was at Reed Smith, yes.

17 Q. On how many occasions?

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

25 Q. Your prior work with Barry

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

4 A. Yes.

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

8 A. Not in a benzene litigation.

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

12 A. Yes.

13 Q. Which type of case was that?

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

18 Q. I'm sorry, contaminated?

19 A. Heparin.

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

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

25 Q. Was it a specific causation

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

5 A. Both.

6 Q. Tobacco and how it causes
7 certain conditions?

8 A. Both specifically and general.

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

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

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

20 A. No.

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

23 A. No.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

11 - - -

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

15 - - -

16 BY MR. DUPONT:

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

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

25 Q. And how much have you earned

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 per year in your work in benzene exposure
3 cases?

4 A. I haven't kept track of that.

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

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

9 Q. Yes.

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

13 MR. DuPONT: Yes, from July 4th,
14 2012 to the present.

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

19 BY MR. DuPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

5 A. No.

6 Q. Is it more or less?

7 A. More.

8 Q. More?

9 A. More.

10 Q. How much have you typically
11 billed per case?

12 A. There isn't a typical, but some
13 cases are initial consult only, which is
14 \$1,500. Some cases are quickly settled and
15 don't require reviewing six boxes of records.
16 Some cases require an extensive detailed
17 report. So it will vary tremendously.

18 A case that goes to court where
19 I have to testify in trial, and has a 30 or
20 40 or 50 page report, is going to be a lot
21 more expensive and a lot more work than a
22 case where I give an initial consultation and
23 nothing more. It all ranges between those.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 outside of the benzene litigation realm?

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

10 Q. When did you begin to practice
11 medicine?

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

14 Q. Yes, sir.

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

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

22 A. I don't recall such a patient.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 caused by exposure to any chemical product?

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 of smoking.

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

6 A. I can't recall such.

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

11 A. No.

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

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

21 Q. Have you written on the
22 epidemiology of benzene?

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 taking the gist of your question and
3 answering that, the answer is, I have not
4 published on benzene's role as a leukemogen
5 or carcinogen or inducer of other
6 malignancies.

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

5 A. For what purpose?

6 Q. For any purpose.

7 A. Yes. Adavita's Oncology
8 textbook is one that's authoritative for
9 guiding clinical practice. In hematology,
10 probably Williams and Winthrop are useful
11 textbooks.

12 Q. I have been provided with a 25
13 page report. Would you call this your -- a
14 25 page document?

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

20 Q. What can we refer to this as?

21 A. Notes with comments.

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

24 - - -

25 (Whereupon the document was

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 marked, for identification purposes,
3 as Sarna Exhibit Number 4.)

4 - - -

5 BY MR. DuPONT:

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

10 MR. THOMPSON: Do you understand
11 the question?

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

14 BY MR. DuPONT:

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

19 MR. THOMPSON: If you agree with
20 that characterization.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 principle it's the issue of whether
3 benzene caused the disease. So the
4 strict answer to your question is,
5 yes. And the explanation is in
6 workers' Compensation cases.

7 - - -
8 (Discussion held off the
9 record.)

10 - - -

11 BY MR. DUPONT:

12 Q. Doctor, you were discussing
13 workers' Compensation being your total
14 workers' Compensation matters. Was that
15 always on behalf of the defendants in the
16 workers' Compensation matters?

17 A. Yes.

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

23 A. Yes.

24 Q. Yes?

25 A. Yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. In what respect?

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

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

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

14 Q. When did that occur?

15 MR. THOMPSON: Did you say when?

16 MR. DuPONT: Yes.

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

19 BY MR. DuPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 with in such a matter.

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

5 A. No.

6 Q. Which agency?

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

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

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

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

18 A. No.

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

21 A. Would you like me to count
22 them?

23 MR. THOMPSON: Maybe you can
24 estimate.

25 THE WITNESS: I'll count. A

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 quick estimate is 44, plus there are
3 some other things that are not
4 articles that are relevant. One is a
5 little thing on benzene toxicity
6 standards and regulations from ATSDR.
7 One is the letter that came out of Dr.
8 Shadduck's deposition from Shell.
9 Another is Dr. Harrison's addendum to
10 his deposition or report, whatever it
11 was, review of records and opinions.

12 BY MR. DUPONT:

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

16 A. Can you define industrial
17 hygiene?

18 Q. Do you consider yourself an
19 industrial hygienist?

20 A. No.

21 Q. Did you hear my question?

22 A. Yes. My answer was no.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

16 A. Correct.

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

20 A. Yes.

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

25 A. I think that's one portion --

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 piece of information that goes into a
3 conclusion or decision. I don't think it's
4 the sum of information or the totality. I
5 think that it's part of the equation.

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

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

12 Q. What do you consider minuscule?

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

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

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

25 Q. How do you used the term

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 cumulative exposure on a qualitative basis?

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 leukemia in that profession. And I would
3 believe that I am qualified as a
4 hematologist/oncologist to look at that
5 literature and come to a conclusion.

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

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

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

21 A. Yes.

22 Q. I'm sorry?

23 A. Yes.

24 Q. What is your opinion?

25 A. If one puts aside his current

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 condition, which I do not have up-to-date
3 information on, but if one puts that issue
4 aside and just deals with the issue of
5 prognosis for someone who has had a second
6 stem cell transplant from an unrelated donor
7 for acute myelogenous leukemia that has
8 relapsed after a first transplant, the
9 figures that his physicians support him have
10 been a 20 to 30 percent success rate and a
11 comparable risk of dying due to the
12 procedure, and I would agree with those
13 figures.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

10 Q. Do you have any opinion?

11 A. I think he was well treated.

12 Q. Do you have any concern?

13 A. No.

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

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

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

22 A. Not a formal opinion.

23 Q. Do you have an opinion?

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 He had psoriasis. He was otherwise
3 reasonably healthy. He was at the time of
4 the diagnosis, I think, 51, if I recall
5 correctly. And the average life expectancy
6 for someone in good health at 51 is probably
7 to about 85. Morbid obesity would probably
8 lower that to about 75 to 80. He had a
9 history of smoking, but it was distant. So
10 probably if I had to opine at this point, off
11 the top of my head, it would have been a 25
12 to 30 year estimate of survival.

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 BY MR. DuPONT:

3 Q. What's your opinion?

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

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

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

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

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

19 MR. THOMPSON: Notes and
20 comments.

21 MR. DuPONT: Notes and comments.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 or depositions. And they may have
3 opinions that come as a result of
4 doing -- reviewing that material. So
5 if there is given further material,
6 either in terms of Mr. Gerchman's
7 clinical course or other people's
8 opinions or other people's reports or
9 other people's depositions, that may
10 require new work and may or may not
11 evoke new opinions.

12 BY MR. DUPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 review of Dr. Shadduck's deposition and Dr.
3 Harrison's handout. And also there's some
4 comments on Dr. Petty's and Dr. Williams'
5 assessments of exposure.

6 Q. What is your experience in
7 industrial hygiene?

8 A. I am not an industrial
9 hygienist.

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

12 A. Hands-on experience, no.

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

15 A. No.

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

18 A. Hold on. Yes, I do.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 Because as he's said, he's got
3 opinions scattered throughout the
4 thing. So I'm certainly not going to
5 want to limit him to only the ones he
6 just identified in a cursory manner by
7 leafing through the thing. I mean, we
8 can use that as a starting point. But
9 I'm just telling you now, this is the
10 -- as with the other experts, those
11 who have provided some kind of report
12 or notes or whatever, like Harrison
13 did, you know we had to go through and
14 kind of suss^ it out, and the same is
15 probably going to be the case here.

16 BY MR. DUPONT:

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 290 pounds was 41.6, which would be morbidly
3 obese. And that on March 7th, which was --
4 March 7, 2011, he weighed 220 pounds and was
5 still obese. Although with his bariatric
6 surgery he had lost 70 pounds over two years.
7 But he still was obese.

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

12 MR. THOMPSON: Take your time,
13 Greg.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 mentioned before in my deposition, is
3 a risk factor for leukemia. But I
4 also said that given the lag time from
5 when he stopped smoking, I did not
6 believe it was a cause of Mr.
7 Gerchman's leukemia.

8 BY MR. DUPONT:

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

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

14 Q. All right.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 AML. But a third of the patients are less
3 than 54 years of age at diagnosis by ^SEIR
4 data, so it is certainly quite consistent
5 with de novo AML. De novo meaning without an
6 identifiable cause.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 relevant issue in terms of the discussion of
3 causation.

4 Q. what is your next opinion?

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

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

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

25 Q. we will get to that, but the

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 way I'm trying to organize this, I would like
3 to first ask what your opinions are.

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

7 Andrew, is that what you have in
8 mind?

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 latency from the time of first
3 exposure to the time of development of
4 leukemia. In the first place, that
5 presupposes that exposure was the
6 cause. And then it presupposes that
7 if exposure were a cause, that it was
8 caused by the first drop of exposure
9 on the first day, both of which are
10 premises that I do not accept.

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

16 BY MR. DuPONT:

17 Q. Doctor --

18 A. I'm sorry.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

15 Q. All right. What is your next
16 opinion?

17 MR. THOMPSON: Keep looking.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 doesn't fit under the umbrella as I
3 see it may still be relevant. So I do
4 not want to be restricted to only what
5 I just listed as opposed to what's in
6 my notes.

7 BY MR. DUPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

15 BY MR. DuPONT:

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

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

20 A. Go ahead.

21 Q. Agreed?

22 A. Yes.

23 Q. Do you agree with that?

24 A. Yes.

25 Q. Number two, smoking, a risk

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

10 A. Yes.

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

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

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

25 A. Very few people are going to

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

17 BY MR. DuPONT:

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

22 A. Yes.

23 Q. That's your fifth opinion?

24 A. Yes.

25 Q. Sixth opinion, the literature

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 on mechanics do not show in the -- in the
3 modern era do not show an increased risk of
4 leukemia or AML?

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

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

12 A. Yes.

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 five years?

3 A. Yes.

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

7 A. Yes.

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

16 BY MR. DuPONT:

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

20 A. Spontaneous mutation, yes.

21 Q. Now, you're going to look
22 through your report and tell me what your
23 10th opinion is.

24 A. Will do.

25 One of the issues that may not

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 have been implicit in the previous relative
3 to Mr. Gerchman was that his exposure was
4 qualitatively small over the ten years
5 preceding his diagnosis.

6 Another opinion that may not
7 have been clear is the issue of looking at
8 part per million year to try to determine
9 which group of benzene exposed people are at
10 increased risk of leukemia. And one uses
11 that metric to determine risk. I would say
12 40 to 60 part per million year would be the
13 low range for an increased risk.

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 of opinions.

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

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

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

23 MR. DuPONT: Well --

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 he's going to be testifying about.
3 You asked what he's going to testify
4 about and I'm telling you.

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

7 MR. THOMPSON: Excuse me?

8 MR. DuPONT: I asked what his
9 opinions are.

10 MR. THOMPSON: Right.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 are in there. So we told you at the
3 start that his report contains all the
4 information and opinions, either
5 direct or indirect, and they're all in
6 there. And I'm telling you now,
7 putting you on notice, those are going
8 to be topic areas he's going to cover.
9 So feel free to ask about them
10 or not, this is your shot.

11 BY MR. DUPONT:

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

6 THE WITNESS: One by one,
7 please.

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

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

17 BY MR. DuPONT:

18 Q. Okay.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 don't have reports out here, he told
3 you his notes --

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

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

11 BY MR. DuPONT:

12 Q. Okay. What are the topic
13 areas that your counsel has indicated he
14 wants you to discuss that are not your
15 opinions here that you have listed so far?

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

21 BY MR. DuPONT:

22 Q. Have you listed all the
23 opinions you have?

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 expectation is, should this come to trial, I
3 will be asked a variety of questions by both
4 yourself and Mr. Thompson, and unless there
5 is an objection to my answering the question
6 which is sustained, I will answer those
7 questions. The questions which he's raised
8 are relevant and I have opinions. Those
9 opinions are intrinsic to some of the
10 opinions and overlapping the opinions I've --

11 Q. What are those opinions?

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

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

17 A. Can the court reporter give us
18 back?

19 - - -

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

22 - - -

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 Dr. Harrison. And my answer is that I
3 do not believe in the general
4 causation belief that low level
5 exposures of benzene is the cause of
6 leukemia. And I do not believe in the
7 specific causation that Mr. Gerchman's
8 cause -- leukemia was caused by
9 benzene exposure.

10 BY MR. DUPONT:

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

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

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

24 BY MR. DUPONT:

25 Q. Okay. Are we done with your

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 opinions?

3 MR. THOMPSON: No, we're not.

4 THE WITNESS: Third issue?

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

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

25 MR. THOMPSON: And you will also

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 be asked whether that can be ruled in
3 or ruled out in Mr. Gerchman's case.

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

6 MR. THOMPSON: Okay.

7 MR. DuPONT: Now are we done?

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

12 MR. DuPONT: Gee, thanks.

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

16 MR. THOMPSON: Right.

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

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

25 MR. THOMPSON: Correct.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 MR. DuPONT: Okay.

3 BY MR. DuPONT:

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

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

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

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

20 Q. Which was 2008 and which was
21 this year?

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

25 Q. So Larson was 2008. And

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Castillo was 2011.

3 A. 2012.

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

13 BY MR. DuPONT:

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

4 Yes.

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

7 - - -

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

11 - - -

12 BY MR. DuPONT:

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. The Adame one is not?

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

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

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

11 MR. DuPONT: I understand that,
12 I apologize.

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

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

25 BY MR. DuPONT:

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

4 - - -

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

8 - - -

9 BY MR. DuPONT:

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

13 A. Yes. Approximately 1.3.

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

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

25 MR. THOMPSON: Andrew, this is

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 the last time I'll bring it up, but
3 when can we expect those checks for
4 the three experts?

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 cases. That's your expert's article.
3 I expect he could provide it for you,
4 but that's the reference.

5 BY MR. DuPONT:

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

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

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

15 A. There are three articles that I
16 just mentioned.

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

19 A. Correct.

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

22 A. No.

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

25 A. None of those three. I have

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 other psoriasis articles which deal with the
3 issue of barrier and absorption. Which adds
4 another opinion. That is, the presence of
5 psoriasis did not increase dermal absorption
6 of benzene. And that -- that will be enough
7 for that.

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

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

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

19 A. May be.

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

25 A. There are two issues. One is

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

25 A. The data are too scanty for me

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 to reach a firm conclusion about whether
3 people with psoriasis are at risk for AML.
4 They may be, based upon the articles that
5 have been quoted. If they are, which may or
6 may not be the case, that may be related to
7 methotrexate, Imuran or other types of
8 issues, and that's not analyzed in these
9 studies.

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

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

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

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

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

25 BY MR. DUPONT:

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

12 THE WITNESS: Okay.

13 BY MR. DUPONT:

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

16 A. Correct.

17 Q. What do you mean by an
18 appreciable cause?

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 pattern of chromosomes that suggest benzene,
3 no reason to think that benzene is in the
4 discussion of such a situation for sitting in
5 this room or pumping gasoline for your car.
6 So there's no appreciable cause for leukemia
7 in that setting and that would be considered
8 de novo.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 studies could have been related to benzene.
3 But that was the data from the sixties,
4 that's not current situation.

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

8 A. It's hard to determine that
9 because someone who's exposed may not have
10 been caused. But when I look at Mitelman's
11 chromosomal data, he had something like
12 20 percent of people had a history of
13 solvents. And some of those had normal
14 karyotypes and certainly not all of them were
15 caused. So that would be about 15 percent
16 from that figure.

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

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

25 THE WITNESS: I'm okay, but

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 other people may need a break in the
3 room.

4 MR. THOMPSON: Go ahead, Andrew.

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

7 - - -

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

11 - - -

12 MR. THOMPSON: Okay.

13 BY MR. DuPONT:

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

16 A. Yes, I did, thank you.

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

22 A. Yes.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 caused by benzene exposure?

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

16 Let me see if I can find you
17 the article.

18 I do not have a printout of the
19 article, but the reference is, "[^]Benzene, The
20 Exosome and Future Investigations of Leukemia
21 Etiology", published in [^]Chembile
22 Interactions, volume 192, pages 155 through
23 159, 2011. And that information is in Table
24 1. And where he gets that figure is totally
25 unclear to me.

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

4 A. Yes.

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

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

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

25 A. They tell us that --

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. Do they give you that figure?

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

15 A. Correct.

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. Do you know the percentage for
3 51 and older?

4 A. No.

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

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

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 cleaners and parts washing solvents contain
3 benzene?

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

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

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

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 generally that you're saying are de novo?

3 A. AML.

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

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

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

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

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

25 A. Yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. Is that correct?

3 A. Yes.

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

8 A. Yes.

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

11 A. Yes.

12 Q. How did you come across those
13 articles?

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

16 Q. Do you have those two articles
17 with you?

18 A. I believe I do. Let me check.
19 Yes, I have them.

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

22 - - -

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 - - -

3 BY MR. DuPONT:

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

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

10 (Reviewing articles.)

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

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

17 A. No.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

11 - - -

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

14 - - -

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 depending upon the details.

3 BY MR. DuPONT:

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

9 MR. THOMPSON: Objection,
10 incomplete hypothetical.

11 THE WITNESS: Valid for what
12 purpose?

13 BY MR. DuPONT:

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

17 MR. THOMPSON: Same objection.

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

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

25 MR. DuPONT: Yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

5 MR. DuPONT: Yes.

6 MR. CARR: Thank you.

7 BY MR. DuPONT:

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

13 MR. THOMPSON: Objection, vague
14 and ambiguous. Incomplete
15 hypothetical.

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

19 BY MR. DuPONT:

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 MR. THOMPSON: Objection. Same
3 objections.

4 Do you understand the question,
5 Dr. Sarna.

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

11 BY MR. DUPONT:

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

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

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

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

21 MR. DUPONT: The anecdotal
22 information that he characterized.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 MR. DuPONT: Correct.

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

21 BY MR. DuPONT:

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

25 MR. THOMPSON: Do you know what

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 he means by bias of the doctor?

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

11 BY MR. DUPONT:

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

16 A. Yes.

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

19 A. Yes.

20 Q. And that's a 1994 article?

21 A. 1997.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 through nine of your notes and comments. And
3 this is a section where you address this
4 opinion. And there are -- the articles your
5 cite are Hotz and Lauwerys, McLean, it's
6 dated 2009. ^Costantini, dated 2001, Guo,
7 G-U-O, dated 2004, Ji, J-I, dated 2005, and
8 Pukkala, P-U-K-K-A-L-A, dated 2009. Are
9 those all of the references that you rely
10 upon for the opinions that the literature on
11 modern mechanics do not show an increased
12 risk of leukemia or AML, they show no
13 increased risk?

14 A. Yes.

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

18 - - -
19 (Whereupon the document was
20 marked, for identification purposes,
21 as Sarna Exhibit Number 8.)

22 - - -
23 THE WITNESS: I'll take a moment
24 to gather what I have. Hopefully I
25 have them all. I will tell you that I

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 did not printout the entire Pukkala
3 article because it's roughly a 150
4 pages. And I also printed out
5 material which was relevant that was
6 from the material on the Internet part
7 of the article, but an appendix or an
8 addendum to the article which was
9 relevant to AML. So of the pages that
10 I will provide are the pertinent
11 pages, but not all 150 pages, plus
12 everything else in the addendum. So
13 hold on, let me see what I can get.

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

19 BY MR. DuPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 opinion?

3 A. I have first cited Hotz, which
4 I have. I have next cited McLean, which I
5 have. I next cited Costantini, which I have.
6 I next cited Guo, which I have. I next cited
7 Ji, which I have. And then I cited Pukkala,
8 which I have the pertinent pages of.

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

14 A. Correct.

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

19 A. To my recollection, no.

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

24 A. To my knowledge, no.

25 Q. Do those articles provide

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

6 A. May I hear that question again,
7 please?

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

14 A. No.

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

19 A. No.

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

23 A. To my knowledge, no.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 karyotype and the absence of what you called
3 typical benzene induced karyotype, which
4 argues against benzene being the cause of his
5 AML.

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

9 THE WITNESS: Run that by me
10 again, please.

11 BY MR. DUPONT:

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

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

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

24 THE WITNESS: Okay.

25 BY MR. DUPONT:

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 discussion about that.

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

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

19 Q. That would be part of it.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 Smith article cited by Dr. Shadduck?

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

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

9 A. Off the top of my head, no.

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

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

15 BY MR. DUPONT:

16 Q. Do you know --

17 A. May I correct my answer to your
18 question?

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

24 Q. Do you have those with you here
25 today?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. I believe so. Let me get
3 those.

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

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

12 - - -

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

16 - - -

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

21 BY MR. DUPONT:

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. Not specifically, no.

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

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

15 Q. Would that be [^]Ethan Natelson,
16 one of the other defense experts in this
17 case?

18 A. This would be Dr. Natelson,
19 yes.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 defense, as he says? Do you know
3 anything about that, Dr. Sarna?

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

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

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

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

24 BY MR. DuPONT:

25 Q. Dr. Sarna, following your

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 attorney's instructions on how you can answer
3 questions, can you tell me --

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

8 BY MR. DUPONT:

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

16 A. The data from the --

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

25 THE WITNESS: May I have the

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 question again, please?

3 - - -

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

6 - - -

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

12 BY MR. DUPONT:

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

22 MR. THOMPSON: Same objections.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 BY MR. DUPONT:

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

5 A. May I have that back, please?

6 MR. THOMPSON: '81.

7 BY MR. DUPONT:

8 Q. Excuse me, '81.

9 A. Mitelman is 1981. Go ahead.

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

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

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

19 A. They looked at the chromosome
20 genetics, meaning karyotype, on 110 subjects
21 with no history of exposure to solvents or
22 pesticides or petrol products. Of these 110,
23 75 had normal karyotypes. They also looked
24 at, it looks like 28 -- 27 subjects who had a
25 history of solvent exposure.

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

9 of the 27 subjects who were
10 solvent exposed, three had normal karyotypes.
11 So there is quite a difference there. 24 of
12 27 had abnormal karyotypes, as opposed to I
13 think it was like 30 percent or so of the
14 people who were not exposed, which is a
15 rather dramatic difference. When you look at
16 the karyotype abnormalities, they tend to
17 have repetitive themes; minus five, 5q minus,
18 minus seven, 7q minus. To some degree
19 trisomy 8, and complex karyotypes meaning
20 three or more different abnormalities.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 that are a subset of the therapy related
3 leukemias.

4 One of your experts, I think
5 Shadduck --

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

8 THE WITNESS: Fine.

9 BY MR. DUPONT:

10 Q. Is there anything that you
11 wanted to add?

12 A. That will do it.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. well, the articles state that
3 they were benzene exposed workers.

4 A. The article states they were
5 solvent exposed workers.

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

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

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

16 A. No.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 normal karyotypes in this group. And perhaps
3 those three did not have benzene associated
4 leukemia -- benzene caused leukemia. But I
5 think there is a dramatic difference in
6 karyotype, which tells me that something is
7 different between these two. And what I'm
8 told is different is solvents.

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

15 A. The timeframe of exposure is
16 not defined.

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

22 A. May I have Exhibit 9 back.
23 The article by Mauritzson,
24 M-A-U-R-I-T-Z-S-O-N, published in Leukemia,
25 2002.

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

5 A. Yes. And myelodysplasia as
6 well.

7 Q. What are the findings of that
8 article?

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

17 Q. And these were not benzene
18 exposed individuals?

19 A. No, these were treatment
20 related.

21 Q. Do we know exactly how benzene
22 causes AML?

23 A. No.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

23 Q. What's the date of that
24 article?

25 A. 2003.

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

5 A. Yes.

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

8 A. Correct.

9 Q. What do you take from this
10 article?

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 model. And as a valid model I think that it
3 is appropriate to look at it and understand
4 the difference in chromosomal patterns.

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

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

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

19 BY MR. DUPONT:

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. I'm unaware of any such
3 occurrence.

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

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

10 Q. What do you take from that
11 article?

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 is a valid model, just as Dr. Shadduck
3 apparently accepted from Dr. Smith.
4 But my take on it is that you look at
5 the frequency, not whether it shows up
6 ever. And there is a marked
7 difference in frequency from the data
8 that I have shown you.

9 BY MR. DUPONT:

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

14 A. My take is that there are
15 differences in frequency between the 5, 7 and
16 8 abnormalities. I also take from it that
17 Dr. Smith believes, based upon the models
18 that he uses, which are labeled in Figure 1
19 through 8, that benzene causes leukemia
20 through chromosomal abnormalities of 5 and 7
21 particularly. And that de novo cases
22 typically have a normal karyotype.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 7, deletion 7q, loss of 5, deletion 5q, and
3 then at the bottom he has de novo cases which
4 are listed as de novo, meaning no cause. So
5 they start with the normal karyotype.

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

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

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

13 MR. THOMPSON: Objection.

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

18 BY MR. DUPONT:

19 Q. What opinions would those be?

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

23 MR. DUPONT: I don't know, the
24 doctor can answer my question.

25 THE WITNESS: He's published a

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 Meta-analysis on lymphoma, or what
3 he's called a Meta-analysis. The
4 first part of that is a listing of
5 anecdotal positive odds ratios out of
6 studies. These include studies that
7 may have 10 different odds ratios,
8 some of which are positive, some of
9 which are negative.

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 my judgment, healthy worker effect
3 does not decrease the risk of
4 lymphoma. A healthy worker effect is
5 largely due to people who have smoking
6 and other types of problems being
7 excluded and I don't --

8 BY MR. DUPONT:

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

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

13 BY MR. DUPONT:

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

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

18 BY MR. DUPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 that's what you want to do. Because
3 we're not on the video record, the
4 record would reflect that he's been
5 interrupted and cut off before his
6 answer is complete. So the record is
7 going to look like that's all he had
8 to say, and it's not. So I think you
9 either need to withdraw the question
10 or do something else other than cut
11 him off and tell him to do something
12 else.

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

19 MR. CARR: You asked him --

20 BY MR. DuPONT:

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

25 MR. CARR: Andrew, I'm okay with

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 that, so long as you make the record
3 clear that you stopped him in his last
4 answer.

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

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

12 BY MR. DuPONT:

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 going off on a tangent that had
3 nothing to do with the question.

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

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

13 MR. THOMPSON: Stop what?

14 BY MR. DuPONT:

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 today as it was not part of my charge.

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

9 BY MR. DUPONT:

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

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

17 Q. Again, these are not benzene
18 exposed workers?

19 A. Correct.

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

8 A. Okay. That appears to be all.

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

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

18 THE WITNESS: Say it again,
19 please?

20 BY MR. DUPONT:

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

25 A. Yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 references that come to that conclusion.
3 Series of alkylating agent related
4 chemotherapy -- alkylating agent related
5 leukemias.

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 there is one of the articles that I gave you
3 on smoking that talked about the risk of
4 smoking related leukemia disappeared after
5 about five years, which implies that the
6 latency period for smoking related leukemia
7 is five years or less. So you would still
8 have a continued increased risk.

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

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

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

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

22 Q. I believe it's Number 6.

23 A. (Reviewing document.)

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 acute leukemia in smokers. Current smokers
3 have a relevant risk of 1.4. Ex-smokers, who
4 have stopped smoking within one to ten years,
5 have a relative risk of 1.1. So that would
6 be the article.

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 benzene has been stopped." So basically they
3 looked at people who were benzene exposed and
4 looked at what their risk was after they
5 stopped the exposure. And by 10 to 15 years
6 it was probably gone.

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

10

- - -

11

12

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

13

- - -

14

15

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

16

BY MR. DuPONT:

17

18

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

19

20

21

22

23

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.

24

25

Q. Do you consider yourself to be
an epidemiologist?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. By trade, no.

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

6 A. That is correct.

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

9 A. I don't recall doing so.

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

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

19 Q. And that was not benzene
20 epidemiology; correct?

21 A. It was disease --

22 Q. Epidemiology of benzene induced
23 malignancies --

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 not conclude that benzene was a cause of
3 lymphoma when I discussed the epidemiology of
4 lymphoma in chapters.

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

8 A. No.

9 Q. No, you do not?

10 A. No, I do not.

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

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

20 Q. That wasn't my question.

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

25 A. Under some circumstances, yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

14 BY MR. DUPONT:

15 Q. Okay.

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

20 A. Are you talking about to the
21 second?

22 Q. Any metric. Year?

23 MR. THOMPSON: With reasonable
24 precision.

25 THE WITNESS: Yes. The answer

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 is, yes.

3 BY MR. DuPONT:

4 Q. Okay. And who has done that?

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

8 Q. Okay.

9 A. When someone --

10 Q. Has anyone done that --

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 it was still in the body for two or three
3 days and that's when the genetic insult
4 occurred, or else you would not have had
5 leukemia.

6 Q. Does every genetic insult lead
7 to leukemia?

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

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 has.

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

5 MR. THOMPSON: Okay.

6 - - -

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

10 - - -

11 BY MR. DUPONT:

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

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

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

24 MR. THOMPSON: If you know.

25 THE WITNESS: My belief is that

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 most of them who use the word latency
3 use that, but a rose is a rose is a
4 rose.

5 BY MR. DUPONT:

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 after the five minute infusion rather than
3 during the five minute infusion.

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

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

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

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

20 BY MR. DuPONT:

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 prior to their chemotherapy?

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

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

14 A. No, I don't know that.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 average of five years prior to diagnosis, and
3 the great majority within ten years. There
4 might be some that are 11 years or 12 years.
5 I don't think there would be any that would
6 be 20 years.

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

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

17 Q. And you would say that no
18 exposure that occurred more than ten years
19 before diagnosis, even if there was a
20 cumulative and ongoing exposure over
21 40 years, that ends within five years from
22 diagnosis, you would say that none of the
23 exposures more than ten years prior to
24 diagnosis played any role in causing the AML?

25 A. I think cumulative exposure as

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 a metric can predict who's at risk for
3 benzene related to acute leukemia. But
4 largely that's because the risk goes up with
5 age and because the risk normally occurs at
6 the tail end of a high -- long career of
7 exposure rather than someone who's only had
8 brief exposure.

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

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

21 A. I would --

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 exceedingly unlikely 15 years or more.
3 I think it very unlikely 10 years or
4 more.

5 BY MR. DuPONT:

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

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

14 Q. All right.

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

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

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

25 Q. Well, if the exposures are more

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

4 A. No.

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

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

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

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

25 Q. Has anybody studied the issue

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 and determined that there are spontaneous
3 AMLs amongst somebody who has never been
4 exposed to any leukemogenic agent?

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

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

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

25 Q. Doctor, my question was

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 different. My question was, is it your
3 opinion that people who have de novo leukemia
4 never had any exposure to leukemogens?

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

8 MR. DuPONT: Yes.

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

12 BY MR. DuPONT:

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 maybe it was not DNA molecule, maybe it was
3 DNA repair enzymes were the molecule of the
4 year. And at that time there was a
5 measurement of spontaneous mutations
6 occurring within the genome. And the author
7 concluded that there was something like ten
8 to the 27th, which is 10 with 27 zeros after
9 it, spontaneous mutations. And made the
10 point that these are much more dangerous than
11 causing cancer than any external carcinogen.

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 something like that. One over 10 to the 25th
3 maybe.

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

7 A. I am concluding it based upon
8 there being, to my recollection, 10 to the
9 27th spontaneous mutations in normal
10 lifetime. I may be off on my number, but
11 it's a big number, whatever it is.

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

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

25 So based upon that, and based

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 upon the fact that the typical mechanic's
3 exposure based upon Scarselli's article is
4 small. And the typical exposure based on
5 williams' article in terms of modern
6 chemicals is small. I think that it was
7 small exposure.

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

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

15 MR. DuPONT: Yes.

16 THE WITNESS: Just asked me
17 about ten years.

18 BY MR. DuPONT:

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

21 MR. THOMPSON: Did you also look
22 at Petty and williams' reports in this
23 case, Doctor?

24 THE WITNESS: The reports from
25 Dr. Petty and williams are very

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 different. Dr. Petty's report claims
3 a lot of dermal exposure, which is, to
4 my knowledge, largely unprecedented in
5 the literature. Doctor Williams
6 differs.

7 BY MR. DUPONT:

8 Q. Dermal exposure is
9 unprecedented in the literature?

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 to be in association -- those associated with
3 leukemia would have to be revised
4 considerably upward based upon new exposure
5 analysis.

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

9 A. What is your question?

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

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

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

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

18 A. A bit better. A bit greater.

19 Q. A bit greater?

20 A. Yes.

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

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

25 THE WITNESS: I don't have a

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 quantitative analysis. I have
3 qualitative. And I can say it's a bit
4 greater, but that's where it ends.

5 BY MR. DUPONT:

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

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

11 Q. Okay.

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

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

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

21 Q. How much of an increased risk?

22 A. It depends upon the exposure.

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

25 A. As a rough estimate, 50 to 100

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 percent increased risk.

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

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

7 Q. The question was 40 part
8 million years.

9 A. Well, I'm answering between 40
10 to 60. I can't give you a precise answer for
11 40 exactly.

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

14 A. 40 to 60 would include that
15 range.

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

18 A. A tiny bit higher than 40 to
19 60.

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. How about 80 part per million
3 years --

4 A. Probably --

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

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

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

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

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

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

21 Q. What do you mean by a marker?

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 some degree the earlier exposure is a marker
3 of risk, rather than a determinant of risk.
4 People who have enough exposure in the ten
5 years prior to it generally have a long
6 history of exposure because exposure is less
7 now than it used to be. And so that is a
8 marker of risk.

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

12 A. In the United States it's
13 unlikely.

14 Q. Is it possible?

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH

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

8 BY MR. DUPONT:

9 Q. Doctor, that's not my question.

10 A. That's my answer.

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

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 other scenarios, so I won't say that's
3 the only way. But in the modern
4 workplace, the evidence is, there's no
5 increase of leukemia and my
6 expectation is that it doesn't occur.

7 BY MR. DUPONT:

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

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

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

20 BY MR. DUPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 and that would be consistent, yes. I think
3 that if I recall correctly --

4 Q. Are the --

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

7 BY MR. DuPONT:

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

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

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

19 BY MR. DuPONT:

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

22 MR. THOMPSON: In what regard?

23 BY MR. DuPONT:

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 whether or not benzene exposure will cause
3 leukemia; correct?

4 MR. THOMPSON: If you agree with
5 that.

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

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

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

25 MR. DuPONT: Whether you agree

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 with that or not, okay.

3 MR. THOMPSON: What's wrong with
4 that?

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

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

11 BY MR. DuPONT:

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

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

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 as a marker or a risk factor.

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

5 A. ^Rinsky is one.

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

11 MR. THOMPSON: Just go ahead and
12 give him the names of the articles you
13 recall.

14 THE WITNESS: The Rinsky
15 article. The Wong analysis of the
16 pliofilm cohort. There are several
17 other analyses of the pliofilm cohort.
18 The Pukkala article. The article by
19 Glass is worth discussion because it
20 is the only one that I know of that
21 claims two part per million as a cause
22 and because there are serious problems
23 with that article that make it not
24 credible.

25 BY MR. DUPONT:

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. So Glass 2003, Pukkala, the
3 pliofilm studies are the articles you rely
4 upon for that opinion?

5 A. Yes.

6 Q. And those are the articles that
7 you considered for that opinion in this case?

8 A. Those are the article I brought
9 with me. On all of my opinions I'm
10 considering the body of information I have,
11 which may not be linked to a specific
12 article, but linked to what I have read in
13 general.

14 I would also add Wong and
15 Rothman's Meta-analysis on petroleum
16 industry. Brown and ^Steinman's various
17 articles on painters. There's a
18 Meta-analysis on diesel exposed workers. All
19 of which show no increased risk of leukemia.

20 Q. Okay. What is the diesel
21 exposed worker study you're referring to?

22 A. I don't have the author off the
23 top of my head.

24 Q. Do you have that article with
25 you?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. No.

3 Q. What's your basis for your
4 disagreement with Dr. Shadduck and Harrison
5 on their opinions on general causation of AML
6 and specific causation in David Gerchman?

7 A. You're going to have to tell me
8 specifically what you want me to opine on.

9 Q. I don't know, your attorney
10 says you're going to opine on that, so I want
11 to know what you're going to say. If you
12 don't have an opinion on that, then tell me
13 you don't have an opinion on that.

14 A. In terms of general --

15 MR. THOMPSON: Hold on a second,
16 Doctor. Take a look at your notes,
17 where you made some notes about
18 Harrison and Shadduck.

19 MR. DuPONT: Counsel, please do
20 not direct the witness how to answer
21 my question.

22 MR. THOMPSON: I'm just telling
23 you, he's going to be asked questions
24 on --

25 MR. DuPONT: No. No. It's not

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 your place to tell me anything.

3 MR. THOMPSON: Whatever.

4 MR. DuPONT: It's not your place
5 to coach the witness how to answer
6 questions. I'm entitled to get an
7 answer from the witness, not an
8 attorney for the witness.

9 MR. THOMPSON: Fine.

10 THE WITNESS: Okay. Regarding
11 Dr. Shadduck.

12 MR. THOMPSON: Andrew, you're
13 ignoring the fact that the guy made 25
14 pages of notes in preparation for
15 coming here and giving his opinions.
16 And you haven't looked through them
17 once. All right? And so he is going
18 to rely on those notes for his
19 opinions in this case. The facts that
20 he wrote down there were those that he
21 found to be salient, as he told you
22 earlier. So I'm telling you now, he's
23 going to be asked questions about
24 Shadduck and Harrison. He made
25 notations there that were important to

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 him that he doesn't have committed to
3 memory. So we're going to go through
4 those now so you have a chance to ask
5 the questions about the things he
6 might comment on. So, go ahead,
7 Doctor.

8 THE WITNESS: In terms of Dr.
9 Shadduck, he cited the threshold for
10 AML was 4.5 to 45 part per million
11 year. That was based on the letter
12 from Shell Oil to EPA regarding the
13 pooled analysis of the Canadian, UK
14 and Australian nested case control
15 studies. And that draft report said,
16 "Preliminary analyses indicate that
17 the risk of MDS was statistically
18 significant at cumulative exposure
19 levels, i.e., 4.5 to less than 45 part
20 per million years, below that
21 previously reported in the literature.

22 Number one is the preliminary
23 analysis. Number two is unpublished.
24 Number three is not --

25 BY MR. DUPONT:

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. Doctor, are you basically going
3 to read your -- however this report was
4 written, are you just going to read this in
5 response to my question about your opinions
6 on Dr. Shadduck and Harrison?

7 A. Parts of it will be taken from
8 that, parts of it will be elaborated on and
9 expanded.

10 Do you want me to continue my
11 answer?

12 Q. All right.

13 A. It's also --

14 MR. THOMPSON: Do you want him
15 to keep going?

16 BY MR. DUPONT:

17 Q. Is it your understanding that
18 it was Dr. Shadduck's opinion that the
19 threshold is 4.5 part per million years?

20 MR. CARR: Hang on a second,
21 he's in the middle of an answer again
22 and you've cut him off. So you either
23 need to withdraw the question, make
24 clear on the record that you stopped
25 him, or let him finish his answer.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 BY MR. DUPONT:

3 Q. Doctor, my question is: Is it
4 your understanding that it was Dr. Shadduck's
5 opinion that the threshold for AML being
6 caused by benzene exposure is 4.5 part per
7 million years?

8 MR. CARR: Okay. I'm going to
9 object to you cutting off the witness
10 again when he's in the middle of an
11 answer. And I want the record to be
12 clear that if you take the position at
13 trial that his concerns with Dr.
14 Shadduck's opinions or Dr. Harrison's
15 opinions were not disclosed to you in
16 this deposition, that he was in the
17 middle of telling you what they were
18 and you stopped him.

19 BY MR. DUPONT:

20 Q. Doctor, can you please answer
21 my question? Is it your understanding that
22 it's Dr. Shadduck's opinion that 4.5 part per
23 million years is the lowest dose, or a
24 threshold above which benzene causes AML?

25 A. In the course of his

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 deposition, one of the things he cited for
3 exposure was the threshold for AML in 4.5 to
4 45 ppm per million year range based upon the
5 letter from Shell. That's my understanding.
6 I believe he also cited different data as
7 well.

8 Q. And is your analysis concerning
9 Dr. Shadduck's opinion based on the letter
10 from Shell to the EPA that which are set
11 forth in these comments and notes?

12 A. There may be beyond that. I
13 have not read to see whether he has done
14 anything else. I can certainly go over it
15 verbally, but I expect most of it is in the
16 notes.

17 Q. All right. Well, why don't you
18 read it to yourself and then tell me if there
19 is anything in addition.

20 A. Comments one and two and three
21 and four on page 60 would encompass my
22 comments on the Shell letter being cited by
23 Dr. Shadduck.

24 Q. All right. Do you have any
25 other criticism of Dr. Shadduck that is not

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 set forth in writing in your notes and
3 comments that we have attached as Exhibit 4?

4 A. My notes and comments would
5 encompass my comments on which would include
6 at times criticisms of Dr. Shadduck's
7 opinions.

8 Q. Okay. Are your comments and
9 criticisms of Dr. Shadduck's opinions fully
10 set forth in this writing?

11 A. Fully? They're not have full
12 elaborations, no. They're set forth in
13 principle.

14 Q. Okay. Is there anything that
15 you want to add that is not written in this
16 notes and comments regarding Dr. Shadduck?

17 MR. THOMPSON: Remembering that
18 you're going to be asked about whether
19 you agree with their opinions on
20 general and specific causation.

21 MR. DUPONT: Counsel.

22 MR. THOMPSON: Andrew, this
23 isn't a federal court report, man.
24 This isn't how we do it in California.
25 We come here and this is the time when

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 he discloses his opinions.

3 MR. DuPONT: I don't care what
4 state it's in. I'm entitled to get an
5 answer from the witness, not the
6 attorney.

7 MR. THOMPSON: That's not an
8 answer. I'm telling you, he's going
9 to be asked that series of questions.
10 I'm not giving him any answer. I'm
11 telling him he's going to be asked to
12 comment on that.

13 THE WITNESS: I disagree with
14 Dr. Shadduck's conclusion that low
15 dose benzene causes leukemia. And I
16 disagree with the specific causation
17 opinion that Mr. Gerchman's leukemia
18 was caused by benzene exposure in the
19 workplace.

20 The reasons for that are all
21 elaborated in my notes and in my
22 deposition. But as a blanket answer
23 to the question, do I agree or
24 disagree with this general or specific
25 causation, I disagree with both.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 BY MR. DUPONT:

3 Q. All right. And if I want to
4 know why you disagree with it, it's set forth
5 in this written report and this written notes
6 and comments?

7 A. Certainly there are additional
8 materials that I haven't documented that
9 bolster the opinions, but the crux of my
10 opinions are in this report. I'm not sure
11 whether I have all the issues about psoriasis
12 in here or not, but I mentioned that before
13 as one of the opinions and I expect we'll get
14 to that.

15 MR. THOMPSON: And we have been
16 here talking for five hours.

17 BY MR. DUPONT:

18 Q. And are you able to add
19 anything, as we sit here today, right now,
20 that bolsters your opinions, as you said,
21 that bolsters your comments and criticisms?

22 A. On Dr. Shadduck?

23 Q. Correct.

24 A. I don't have anything at this
25 point to add.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. Now, moving on to Dr. Harrison.
3 Are all of your points of disagreement and
4 criticism with Dr. Harrison's opinions and
5 testimony set forth in this written notes and
6 comments?

7 MR. THOMPSON: Andrew, let me
8 just clarify. He doesn't have the
9 deposition transcript. Did you have
10 Harrison's deposition transcript?

11 THE WITNESS: The only thing I
12 had was his handout.

13 MR. THOMPSON: So he had only
14 his report, so that's all he can
15 comment on at this time.

16 MR. DuPONT: Okay.

17 BY MR. DuPONT:

18 Q. You have criticized and
19 critiqued Dr. Harrison without reading his
20 deposition testimony?

21 MR. THOMPSON: He hasn't gotten
22 -- we don't have Dr. Harrison's
23 deposition yet, buddy.

24 MR. DuPONT: I'm asking a
25 question, Barry, okay.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 BY MR. DuPONT:

3 Q. Am I correct that you
4 criticized and critiqued Dr. Harrison's
5 opinions without reading the deposition
6 testimony?

7 MR. THOMPSON: That is just such
8 an unfair question, I can't believe
9 it. We don't have his deposition yet.

10 MR. DuPONT: I didn't suggest
11 you did. Okay. I'm entitled to ask
12 what he did. If you would stop
13 interrupting and stop interfering, I
14 would appreciate it. Stop it.

15 MR. THOMPSON: Don't answer that
16 question, Dr. Sarna. That's an
17 unfair, ridiculous, argumentative
18 question. He knows we don't have the
19 transcript yet and we have told him
20 you're going to read the transcripts
21 of any witness testifying on
22 causation. So don't answer that
23 question, that's a terrible question.

24 BY MR. DuPONT:

25 Q. Doctor, are your critiques and

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 and criticism of Dr. Harrison and his
3 opinions based only on his written report and
4 not what he stated during his testimony?

5 A. My written comments in my notes
6 are based upon the roughly 10 page report
7 that I got.

8 Q. Okay.

9 MR. THOMPSON: He is going to
10 read the doctor's testimony and
11 comment, just so you know. Because --
12 BY MR. DUPONT:

13 Q. And are all of your opinions
14 and criticisms in this written report, as
15 they relate to Dr. Harrison?

16 A. What I did was, I commented on
17 what was written there. That does not
18 include all my proactive comments, but it
19 includes all my reactive comments.

20 Q. Okay. Are there any comments
21 or opinions or analyses that you need to add
22 to your review of Dr. Harrison's report and
23 opinions that are not set forth in here,
24 including the basis for them?

25 MR. THOMPSON: I guess we're

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 going to go through the same exercise,
3 in that he's going to be asked to
4 comment on, agree with, disagree with,
5 or expand on Dr. Harrison's opinions
6 on general and specific causation. So
7 you are going to be asked that, Dr.
8 Sarna.

9 THE WITNESS: I expect to see
10 his deposition. I expect I will react
11 to that deposition. There may be
12 things that I agree with or disagree
13 with. There may be things to change
14 what I have written, or there may be
15 things to add to what I've written.

16 BY MR. DUPONT:

17 Q. Okay. Based on information you
18 have from Dr. Harrison at this point in time,
19 is there anything you have to add to your
20 opinions, your analysis and your criticism of
21 Dr. Harrison, beyond what's stated in your
22 written notes and comments?

23 A. My written notes and comments
24 reactive to the handout. It was not a
25 criticism of Dr. Harrison, it was -- it may

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 be a criticism of his comments, but it was
3 not criticism of him personally.

4 There may -- that was reactive
5 comments, not proactive comments. And so
6 it's reactively reasonably complete.
7 Proactively there may be things lacking
8 because this is not a formal report. And
9 it's incomplete from the point of view of
10 evaluating his opinions because I haven't
11 seen his deposition.

12 Q. Do you have anything to add to
13 your analysis, your criticisms or your
14 opinions pertaining to Dr. Harrison's written
15 report?

16 A. Not at this time.

17 Q. Or written notes?

18 A. Not at this time.

19 Q. Have you reviewed the most
20 recent Tran group studies?

21 A. I think it's 2010 or 2011.

22 Q. How many cases of AML were
23 found in the most recent Tran group study?

24 A. It was either three or four. I
25 believe three.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. And have you discussed with any
3 of the other doctors hired by defendants in
4 this case what their views on the Tran group
5 study are?

6 A. No.

7 Q. All right. Do you have an
8 opinion that you cannot rule in or rule out
9 de novo AML? First of all, is de novo a
10 cause of AML or is it just the absence of
11 knowledge of what caused AML?

12 A. Leukemia -- an AML would be
13 considered a de novo in the absence of a
14 known cause.

15 Q. Okay. So de novo in and of
16 itself is not a cause of leukemia; correct?

17 A. De novo means it occurred
18 without a known cause. It is not a cause.

19 Q. Okay. Without a known cause;
20 correct?

21 A. Yes.

22 Q. There could be a cause, but we
23 just don't know what it is; correct?

24 A. Everything has a cause. Most
25 of these presumably are spontaneous

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 mutations.

3 Q. But when we talk about de novo
4 and idiopathic we're talking about something
5 that we just don't know what caused it?

6 A. Idiopathic certainly means we
7 don't know. De novo means it occurred
8 without a known cause. And we can surmise
9 that most of those are spontaneous mutations,
10 but that's a surmise. There's no way of
11 proving or disproving it.

12 - - -
13 (Discussion held off the
14 record.)

15 - - -
16 (Whereupon there was a recess in
17 the proceeding from 3:34 p.m. to 3:41
18 p.m.)

19 - - -

20 BY MR. DUPONT:

21 Q. Doctor, do you hold the opinion
22 that you cannot rule in or out de novo AML?

23 A. There is no specific test to
24 prove that something is from a spontaneous
25 mutation. De novo simply is a term meaning

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 you don't know the cause and it appears to be
3 without a cause. So you can rule that in or
4 rule that out by saying there's no known
5 cause.

6 Q. So your basis -- have you ruled
7 in de novo leukemia?

8 A. In the absence of a known
9 cause, I can consider this de novo.

10 Q. Okay. So you're going to tell
11 the jury that it's more likely that David
12 Gerchman's AML arose for no reason whatsoever
13 than it is that his 40 year history of
14 exposure to benzene caused his AML?

15 A. I'm going to say that David
16 Gerchman's AML falls in the 80 percent of
17 AMLs with no identifiable cause.

18 Q. And is it your opinion that
19 that 80 percent of AMLs had a 40 year
20 exposure history with benzene?

21 MR. THOMPSON: Objection, vague
22 and ambiguous.

23 THE WITNESS: You're asking
24 whether everybody in that 80 percent
25 has had 40 years of benzene? The

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 answer to that is, no.

3 BY MR. DuPONT:

4 Q. Is it your opinion that anyone
5 within those 80 percent of de novo leukemias
6 has a 40 year occupational history of
7 exposure to benzene?

8 A. There are many, many, many
9 people who work with contaminant level
10 benzene. These people are not immune to
11 de novo leukemia. And I'm sure there are
12 plenty of people who have a history of
13 contaminant level of benzene who have
14 developed de novo leukemia.

15 Q. So you would include people
16 with a history of almost 40 years of exposure
17 to a known leukemia causing agent --

18 A. The people who --

19 MR. THOMPSON: Let him finish
20 his question, Doctor.

21 BY MR. DuPONT:

22 Q. Let me finish my question,
23 Doctor.

24 You would consider people with
25 a 40 year occupational exposure history of

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 exposure to an agent known to cause AML as
3 being within the 80 percent of individuals
4 who you claim develop AML as a result of no
5 known cause?

6 MR. THOMPSON: Objection,
7 incomplete hypothetical.

8 THE WITNESS: The people who
9 have a history of benzene exposure who
10 get de novo leukemia will generally
11 get it at an average age of about 65,
12 and will generally have started having
13 their contaminant level exposure
14 40 years, 45 years earlier. And they
15 will all have that history. They are
16 not immune to getting de novo
17 leukemia.

18 If these people all were having
19 leukemia due to benzene, you would see
20 a marked increase in leukemia. But
21 you do not. The studies that I have
22 shown to mechanics show that there is
23 no increase in AML. Therefore, the
24 cases that occur are spontaneous and
25 de novo rather than benzene caused.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 These people are not immune to de novo
3 leukemias. They will get it just like
4 anybody else.

5 BY MR. DuPONT:

6 Q. But in your opinion you can get
7 leukemia from no known cause, even though you
8 have exposure to a known cause of leukemia?
9 That's your opinion?

10 A. Of course.

11 Q. Okay. And it's your opinion
12 that you can get leukemia from no known cause
13 even though you have an occupational exposure
14 for 40 years to a known cause of leukemia?

15 A. Of course.

16 Q. And it's your opinion that some
17 of those 80 percent of the individuals with
18 AML that you attribute to no known cause
19 leukemia had 40 year occupational exposure
20 history to benzene?

21 A. Yes.

22 Q. Have you now described for me
23 your opinions on de novo AML generally and
24 specifically?

25 A. Yes. With the understanding

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 that I have already previously discussed the
3 genetic differences between de novo AML and
4 solvent related AML, and that plays into the
5 -- my overall opinions.

6 Q. All right. Are there any
7 articles that conclude or state or opine that
8 you cannot rule in or out de novo leukemia as
9 a cause of AML?

10 A. I know of no article that has a
11 test for de novo leukemia. It's a diagnosis
12 of exclusion. That rules it in or out. You
13 rule it out if you have another cause. So
14 someone who has chemotherapy with Cytosan at
15 age 18 and two years later gets a 5 minus, 7
16 minus leukemia, you've ruled out de novo
17 leukemia.

18 Q. You rule out de novo leukemia
19 if you have a 40 part per million cumulative
20 dose of exposure to benzene?

21 A. If you had 40 part per million
22 chemo dose exposure to benzene that ended
23 20 years ago and you have chromosomes that
24 are normal now, I would rule out that
25 benzene, yes.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. If you have a 40 part per
3 million cumulative dose of exposure to
4 benzene that ended less than 20 years ago, 20
5 years from diagnosis, do you rule de novo
6 leukemia as the cause of the AML, you rule
7 that out?

8 A. No, I don't rule out de novo
9 leukemia. I look at the chromosomes, I look
10 at the exposure in the last 10 years and I
11 would reach a judgment.

12 Q. Can you opine that somebody's
13 leukemia and AML specifically was caused by
14 benzene exposure solely based on their
15 chromosomes?

16 A. No.

17 Q. All right. Your last opinion
18 is that the presence of psoriasis did not
19 increase David Gerchman's dermal absorption
20 of benzene. Is that correct?

21 A. Yes.

22 Q. Do you hold that opinion?

23 A. Yes.

24 Q. Let me back up. I don't think
25 I got an answer. Are there any articles that

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 conclude or opine that you cannot rule in or
3 out de novo AML as a cause of AML?

4 A. Every article that has an
5 article about AML with a known cause opines
6 inferentially that de novo leukemia was ruled
7 out in that particular case.

8 Q. well, inferentially, that's
9 your interpretation of the article. Are
10 there any articles where the authors have
11 written that you can or you cannot rule in or
12 out de novo AML?

13 A. I have no -- know of no article
14 that says, in this case of leukemia which was
15 caused by Cytosan, we have ruled out de novo
16 AML.

17 Q. Has anyone written an article
18 that says that there is a method for ruling
19 in or out de novo leukemia as a cause of AML?

20 A. I don't know whether someone
21 has said that it's a diagnosis of exclusion,
22 but it's relatively obvious.

23 Q. Is there any article that you
24 can point me to that shares the opinion that
25 you cannot rule in or out de novo AML?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. I have answered the question.

3 Q. Can you cite for me an article
4 that says you can or cannot rule in or out de
5 novo AML?

6 A. I know of no specific article
7 to tell you.

8 Q. Okay. Presence of psoriasis
9 did not increase dermal absorption. What's
10 your basis for that opinion?

11 A. I think it likely that his
12 psoriasis was plaque psoriasis, chronic
13 plaque. I don't have pictures. But the fact
14 that he was self managing and presumably able
15 to have skin exposed to solvents or chemicals
16 argues against having ulcers or other types
17 of psoriasis.

18 Assuming it is chronic plaque,
19 that is thickened skin, which in principle
20 should be a variable to absorption rather
21 than. There are studies that have looked at
22 absorption of steroids and salicylates when
23 applied topically to psoriasis and shown no
24 increased absorption or evidence of
25 substantial absorption. And it is relevant

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 that there is a long history of treating
3 people with psoriasis topically with coal
4 tar, which has contaminant level benzene in
5 it. And that coal tar is not known or felt
6 to be a carcinogen or leukemogen and is still
7 approved by FDA for use in psoriasis.

8 Q. And how long has coal tar been
9 approved by the FDA for use in psoriasis?

10 A. I don't know. 30, 40 years
11 maybe.

12 Q. And is it still approved by the
13 FDA for use in psoriasis?

14 A. As far as I know, yes.

15 Q. Do doctors still prescribe coal
16 tar for use in treating psoriasis?

17 A. I'm not a dermatologist, I
18 can't answer how often it's still prescribed.

19 Q. I didn't ask how often, I just
20 asked is it still prescribed?

21 A. To my knowledge, the answer is
22 yes, but I'm not a dermatologist.

23 Q. Have you looked at any articles
24 that addressed the rate or amount or manner
25 of benzene exposure through the skin in

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 individuals with psoriasis?

3 A. No. I have not been able to
4 find such articles.

5 Q. What articles do you rely upon
6 for your opinions that the presence of
7 psoriasis did not increase dermal exposure of
8 David Gerchman to benzene?

9 A. I have the article by Wester,
10 which was previously discussed, that talks
11 about plaque having very little effect, but
12 some, on water loss, which is not the same as
13 absorption. In terms of absorption, there is
14 a -- sorry, the Wester article talks about --
15 I misspoke. The Wester talks about
16 absorption of hydrocortisone and there is no
17 difference in absorption when they applied
18 hydrocortisone to a psoriatic area versus a
19 normal area.

20 And there is an article I have
21 only in an abstract by Gip that talks about
22 absorption of betamethasone and salicylic
23 acid and found that there was no change in
24 plasma cortisol levels, which argues against
25 any absorption of betamethasone. And there

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 was no detectable salicylate levels in the
3 plasma, which tells you that that was not
4 absorbed. So those are the two articles.

5 Q. Is it your opinion that
6 hydrocortisone and benzene are absorbed
7 through the human skin the same way and at
8 the same rate?

9 A. I don't know whether that's
10 true or false. I have no data to answer that
11 question.

12 Q. Is it your opinion that benzene
13 and betamethasone are absorbed through the
14 human skin in the same way and at the same
15 rate?

16 A. I have no data to answer that
17 question.

18 Q. Is it your opinion that
19 salicylic --

20 A. Salicylic, like aspirin, but
21 it's not acetylsalicylic acid, it's just
22 salicylic acid.

23 Q. Okay. Salicylic acid, is it
24 your opinion that salicylic acid and benzene
25 are absorbed through the human skin in the

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 same way and at the same rate?

3 A. I have no data to answer that
4 question.

5 Q. Have you seen any testimony
6 that there were, in fact, open sores on David
7 Gerchman's skin from his psoriasis --

8 A. No.

9 Q. -- or is it your assumption --
10 I'm sorry, you have not seen that testimony?

11 A. I have not seen testimony about
12 open sores. I have seen testimony about
13 discolored lesions which sound like plaque,
14 but I have not seen open sores.

15 Q. If there were open sores on
16 David Gerchman's skin from psoriasis, would
17 that change your opinion that the psoriasis
18 did not increase his dermal absorption of
19 benzene?

20 A. If there were open, weeping
21 ulcers, what I said would not apply. That
22 would not mean that absorption was increased.
23 It would mean that it would not apply.

24 Q. If there were open sores on
25 David Gerchman's skin from psoriasis, would

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 his dermal absorption to benzene have been
3 increased?

4 A. Unknown.

5 Q. Unknown?

6 A. Unknown to me.

7 Q. Okay. Do you agree that
8 benzene has the same capability of causing
9 AML if it is absorbed through the human skin
10 as it does as if it's inhaled through the
11 lungs?

12 MR. THOMPSON: Hold on a second.
13 Objection, form and foundation.

14 THE WITNESS: I think if a
15 benzene molecule gets to the bone
16 marrow, it doesn't matter how it was
17 absorbed. Whether or not there is a
18 different handling of benzene that
19 goes -- comes from the skin,
20 putatively absorbed from the skin as
21 opposed to benzene that's inhaled, I
22 don't know.

23 BY MR. DuPONT:

24 Q. Okay. Will you take your
25 articles on psoriasis and we will mark those

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 as Exhibit 11, I think. Triebig should have
3 been 10. I want to mark your latency
4 article, if it's not already marked, as
5 Exhibit 10.

6 - - -
7 (Whereupon the documents were
8 marked, for identification purposes,
9 as Sarna Exhibit Numbers 10 and 11.)

10 - - -

11 BY MR. DUPONT:

12 Q. Exhibit Number 11 should be the
13 Wester and Gip, which are all the articles
14 that you relied upon and considered for your
15 opinions that the presence of psoriasis did
16 not increase the dermal absorption of benzene
17 by David Gerchman. Is that fair?

18 A. The articles, yes, but I did
19 comment on the coal tar issue.

20 Q. Have you brought any articles
21 with you that we have not marked as exhibits?

22 A. Yes, but I'm not sure they're
23 relevant. Some of them were dealing with the
24 comments of your other doctors. Some of them
25 were dealing with the issue of low level

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 exposure. There is Hayes from the CAPM
3 study. There's the Collins article. There's
4 some articles on autoimmune diseases, which I
5 guess relate to psoriasis that I can pull.
6 There's an IOM report by Miller and a letter
7 by Miller regarding the three case controlled
8 studies which are going to be nested on dose.
9 There's some articles that --

10 Q. Let me ask you this: Have we
11 marked as exhibits all the articles you are
12 relying upon for your opinions?

13 A. Yes. But some of them are
14 relevant to the comments on Dr. Shadduck or
15 Dr. Harrison in terms of dose response. So
16 some of these are issues in terms of the
17 question of dose level. And I'm not citing
18 them to support me, but if someone cites them
19 to reach another conclusion I have them for
20 discussion.

21 Q. Okay.

22 A. So I'm not citing CAPM data,
23 but if someone wants to discuss why CAPM data
24 is not a valid tool to say that low level
25 benzene exposure causes leukemia, I have such

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 data available.

3 Q. All right. So what articles
4 did you bring with you on the CAPM issue and
5 what are your opinions on the CAPM study?

6 A. I brought with me Hayes, but
7 the opinions depends upon more than Hayes.
8 The Hayes article would claim, I think, less
9 than 10 part per million year and it has an
10 increased risk. But the Hayes article in the
11 control group does not consider leukemia NOS,
12 or not otherwise specified. And that makes
13 the control group very uncertain. And the
14 numbers are small enough that that could make
15 a difference. And the Hayes group is not
16 properly controlled for age. When you take a
17 look at Yin's article about the breakdown of
18 CAPM, those people who are over 30 and entry
19 on the study make up 16 percent of the
20 exposed group rather than 10 percent of the
21 control group. So one has to correct for
22 that, not just the size of the control group
23 in general and the size of the overall group.
24 And that means that the overall risk factors
25 that they come up with are inflated. And how

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 those risk factors fit into the dose
3 dependent risk is uncertain.

4 MR. CARR: Andrew, this is Carr,
5 just for the record. Some of the
6 studies that he brought with him have
7 some relevance to Glass and some other
8 issues. I do intend to ask him
9 questions about those studies in this
10 case. I would get copies --

11 BY MR. DUPONT:

12 Q. Have we exhausted your comments
13 on the CAPM data? And for CAPM, we're going
14 to abbreviate that C-A-P-M, and that stands
15 for the Chinese Academy of Preventative
16 Medicine.

17 A. Yes. It's actually CAPM NCI,
18 but that's fine.

19 Q. Right. Have you given us all
20 of your analysis on the CAPM data that you
21 find relevant to this case?

22 A. Well, certainly I have not
23 given the details, but I have given you the
24 framework.

25 Q. Okay. Where do we find the

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 details?

3 A. I would have to generate them.

4 Q. Can you tell me what the
5 details are now?

6 A. No. Because they are the
7 specific numbers. Taking a look at the
8 specific numbers of people in each group with
9 specific odds ratios they came to. What the
10 odds ratio is grossly, just taking 75,000 and
11 35,000, or whatever the numbers are for the
12 two groups. How they allegedly controlled
13 for age, but came out with a higher odds
14 ratio rather than a lower one. Looking at
15 the age distribution of the leukemic patients
16 that are described by Travis in a different
17 population, where something like 80 percent
18 of them were in their forties or older,
19 meaning that they were entered in the cohort
20 in their thirties. And that's where the
21 leukemia linked. So it would require going
22 through those details, which I don't have in
23 front of me. But that's the framework.

24 Q. Okay. So anything beyond that,
25 you're not able to provide me at this time?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. I have told you those -- I
3 don't have the details to provide you at this
4 time. I have the framework, which I have
5 provided you.

6 Q. Okay. And do you have all the
7 articles with you that you used for your
8 analysis on the --

9 A. No, I didn't bring every CAPM
10 article with me. You have to go to the
11 Travis article, and one of the Yin articles.
12 You have to break down by age. And the
13 Travis article, to get the break down of the
14 age of the leukemic patients, they weren't
15 analyzed. It's not all in one article. You
16 have to look widely.

17 Q. So I can't discuss those
18 articles with you in detail today?

19 A. I did not provide a detailed
20 part of that. Again, that's more of a
21 rebuttal issue than a proactive issue. I
22 don't claim to support that, it's not a
23 proactive statement that tells me that the
24 risk is only at high levels. It is a
25 reactive statement that tells me if your

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 experts argue the CAPM data, and I did not
3 see that argued by them in their deposition
4 or sheets. So I did not provide that
5 rebuttal data. But should they make that
6 argument beyond that, I have those comments.

7 Q. All right. What are the
8 comments you can give me today on the Health
9 Watch study series?

10 A. Glass in 2003 and then 2005
11 came out with a nested case control of
12 something like 32 or 33 leukemics. Something
13 like 11 of which were AML. As to relative
14 risk function of exposure, it came to the
15 conclusion that at two parts per million
16 exposure and higher there is increased risk.

17 At face value, when you look at
18 how many people have that exposure, this
19 would suggest that the relative risk as
20 compared to a control group, SIR or SMR,
21 should be roughly three. But the Gunn
22 article shows that there's no increase in
23 leukemia and in AML there's no increase in
24 SMR or SIR in leukemia. So that at face
25 value doesn't make any sense.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Then when you go on to look at
3 how many people who got leukemia had all time
4 high exposure with greater than 32 part per
5 million years, an exposure of concentrated
6 benzene, they had -- were in the highest
7 exposure group, and the odds ratio was
8 something like eight or nine to one or
9 higher. And there was something like nine
10 patients in that group where one was
11 expected.

12 That means in the lower group,
13 which is everything below the high exposure
14 group, there was 23 patients expected. And
15 based upon the odds ratios, that was double
16 or triple what you expected, but that has to
17 be less than what you expected because there
18 was no increased risk.

19 So the whole thing makes no
20 sense unless the control group, which is low
21 exposure, had much fewer cases.

22 Apparently in the Millers
23 article, IOM report of the plans for the
24 pooled article between the Canadian, UK and
25 Australian case control studies, the authors

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 of the Australian study, which I presume are
3 Deborah Glass and others, admit there's a
4 problem with their data and it is uncertain.

5 Q. Is it your understanding that
6 the Gunn study that you referred to and the
7 Glass 2005 were nested case control studies,
8 the same nested case control study as the
9 Glass 2003?

10 A. Gunn is a cohort study. I
11 never said it was a nested case control. It
12 gives you SMR and SIR. It's the same Health
13 Watch population, but it's the cohort study.

14 Q. All right. And the Glass 2005,
15 the cohort study, is a nested case control
16 study?

17 A. Nested case control. The major
18 publication is 2003, but in 2005, they go
19 into the high exposure group.

20 Q. Have you seen any data or
21 information concerning the study that
22 combined the three petroleum distribution
23 cohorts --

24 A. Only the Shell oil letter --

25 Q. I haven't finished my question.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 -- beyond the letter that Shell
3 sent to the EPA reporting statistically
4 significant increased risks of MDS at
5 cumulative doses of 4.5 to 45 part per
6 million years? Have you seen any data or
7 commentary for inclusions from that study
8 beyond the letter that was sent to the EPA?

9 A. No. Although I will comment
10 that had there been a low risk of leukemia, I
11 would have expected that they would have been
12 duty bound to report that. And the absence
13 of such a statement makes me wonder whether
14 no such finding occurred.

15 Q. Do you know if they looked at
16 the risk of leukemia?

17 A. Well, the original case
18 controls, as far as I know, looked only at
19 leukemia and didn't look at MDS. So I was
20 surprised to see information come out on MDS.
21 If they did not look at leukemia, they failed
22 the intent of pulling the data.

23 Q. Okay. So my question to you
24 was, do you know if they looked at leukemia?

25 A. I know they were supposed to

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 look at leukemia. I don't know whether they
3 did what they were supposed to do.

4 Q. Okay. So you don't know if
5 they looked at leukemia; correct?

6 A. As a matter of fact I do not
7 know. As a matter of conjecture, I would be
8 shocked if they didn't do it.

9 Q. Have you seen any document that
10 sets forth the intent or the goal or the
11 design of that study?

12 A. Yes.

13 Q. What document is that?

14 A. An article by Miller I believe
15 talks about that.

16 Q. So you believe Miller's IOM
17 report sets forth the objectives and the
18 study design of that combined cohort study
19 that Shell reported to the EPA?

20 A. There was an article by Miller
21 separate from the IOM report too that was
22 shorter.

23 Q. Okay. So which article do you
24 contend provided the goal and the design for
25 the combined petroleum distribution marker

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 cohort study that was reported to Shell?

3 A. There's more than one, but the
4 information I have now is published, Miller,
5 et al., International Archives, Occupational
6 and Environmental Health, 2010, Volume 83,
7 page 69 through 76.

8 Q. All right. Let's make that the
9 next exhibit, please.

10 - - -

11 (Whereupon the document was
12 marked, for identification purposes,
13 as Sarna Exhibit Number 12.)

14 - - -

15 THE WITNESS: This was talking
16 about the exposure assessments, which
17 were a prelude to that. There may be
18 another article that talks about the
19 plan that I don't have with me. But
20 this talks about the visits to the
21 assure exposure measurements were the
22 same in preparation for that study.

23 Let me take a look at the IOM
24 report.

25 (Reviewing document.)

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 The IOM report talks about the
3 advantages of a pooled analysis. It
4 talks about how it should be done and
5 talks about pathology review. I'm not
6 sure it exactly sets out what it is,
7 but it's preliminary to it and fairly
8 talks about leukemia.

9 - - -

10 (Whereupon the document was
11 marked, for identification purposes,
12 as Sarna Exhibit Number 13.)

13 - - -

14 BY MR. DUPONT:

15 Q. Is it your opinion that
16 Exhibit 13, the IOM report, sets forth the
17 study design and objectives for the combined
18 case control study that Shell reported to the
19 EPA?

20 A. The IOM report audited the
21 three in preparation for the study.

22 Q. So it does not set forth the
23 plan or the design of the objectives?

24 A. I don't see a clear distinct
25 plan. I believe there is another article,

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 which I did not bring, that has that. But
3 the IOM report and the Miller report
4 elsewhere talk about preparation for this
5 article rather than the specific study.

6 Q. Can you identify for me any
7 other article in which you believe the plan
8 and the objective and the goal of the
9 combined case control study of petroleum
10 distribution workers at Shell published to
11 the EPA has set forth?

12 A. I can tell you I believe there
13 is such an article, but I can't give you the
14 details of it now.

15 Q. Are there any other articles
16 that you brought with you that are relevant
17 to your opinions in this case, including your
18 analysis of Dr. Shadduck, Dr. Harrison and
19 Mr. Petty, that we have not marked as
20 exhibits?

21 A. Did you want the Glass exhibit
22 marked?

23 Q. Glass 2003?

24 A. Yeah.

25 Q. Okay.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. And do you want Glass 2005
3 marked?

4 Q. Might as well.

5 - - -

6 (Whereupon the documents were
7 marked, for identification purposes,
8 as Sarna Exhibit Numbers 14 and 15.)

9 - - -

10 THE WITNESS: And did you want
11 Hayes marked?

12 BY MR. DuPONT:

13 Q. Yes.

14 - - -

15 (Whereupon the document was
16 marked, for identification purposes,
17 as Sarna Exhibit Number 16.)

18 - - -

19 THE WITNESS: The other issue as
20 long as I'm -- well, one of the issues
21 that we have not discussed that you
22 asked about my comments on your other
23 experts would be the issue of
24 hematoxicity at low dose benzene.
25 These were cited as some as if they

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 indicated a low dose benzene caused
3 leukemia. There were -- article by
4 Ward that was cited I think by Dr.
5 Harrison in this regard. Ward was the
6 pliofilm study and did show that
7 people with low exposure may have some
8 changes in their blood counts. But
9 those people did not get leukemia.

10 We know from the pliofilm study
11 that however you want to use the
12 metric, at least it was 40 part per
13 million years, if not 200 part per
14 million years, that in that study the
15 threshold -- so, yes, there was
16 hematologic toxicity, but that doesn't
17 mean that there was leukemogenesis.
18 That's a totally separate issue. One
19 does not relate to the other.

20 And there was articles by I
21 think Land, which were quoted as
22 showing hematoxicity at low levels of
23 benzene. And I would just point out
24 that those were in the later article
25 by Land, found to be dependent upon

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 specific genotypes. And this is
3 limited to people who are Chinese. So
4 how that applies here is unclear.

5 And, again, that doesn't mean
6 that there was an increased risk of
7 leukemia. If you choose to use
8 abnormal blood counts as a marker of
9 benzene exposure, it should be noted
10 that there was no abnormal blood count
11 in June of 2007 when Mr. Gerchman had
12 a blood count.

13 Then there was something else
14 that I wanted to discuss. Yes. The
15 Iron study was quoted as evidence that
16 only 24 percent of patients with
17 benzene associated -- benzene caused
18 leukemias have chromosomal
19 abnormalities. This was, one,
20 myelodysplasia, not leukemia. Two,
21 the people with high benzene exposure
22 had abnormal eosinophil precursors,
23 which are not normally found. And if
24 that is a unique syndrome to China
25 with a specific genetic abnormality

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 that doesn't show up on a karyotype,
3 that's fine. If I see someone with
4 MDS, and abnormal eosinophils, I would
5 not think that abnormal karyotype in
6 that particular syndrome is necessary.
7 But that syndrome is not relevant
8 here.

9 The people with lower exposure
10 may or may not have benzene caused
11 MDS. We have no information on
12 whether this population was at
13 increased risk or not. So as we
14 discussed before, people who have been
15 exposed to benzene don't mean that
16 their leukemia was caused by benzene.
17 But let me take my --

18 - - -

19 (Discussion held off the
20 record.)

21 - - -

22 BY MR. DUPONT:

23 Q. Are all of the studies that you
24 rely upon for your own opinions, as opposed
25 to your analysis of the opinions of Dr.

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 Shadduck and Dr. Harrison, the ones you
3 already identified for us when I asked you
4 about your opinions? Are the remaining
5 articles that you have with you those which
6 are -- those that are identified in your
7 critique of Dr. Harrison and Dr. Shadduck?

8 A. There's the Collins article,
9 which deals with peak exposure, so I don't
10 know whether you want to consider that. I
11 have the Gunn article which you haven't
12 marked. I don't know whether you want to
13 mark Gunn.

14 Q. Can you answer my question,
15 please. Are the articles that you have left
16 in your stack that we have not marked as
17 exhibits, articles that you have brought
18 because of your critique of Doctors Harrison
19 and Shadduck, or articles that you are
20 relying upon for your primary opinions in the
21 case?

22 A. I would say Collins would be
23 supportive of my primary opinions since it
24 showed little increased risk of leukemia with
25 cumulative dose.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 MR. CARR: Dr. Sarna, and I hate
3 to interrupt. I think he's asking you
4 a more basic question, and that is,
5 you have a stack left. Do you either
6 rely on everything in the stack for
7 the criticisms of his experts or are
8 you relying on it in your opinions in
9 this case? He just wants to know
10 whether --

11 THE WITNESS: Criticism of your
12 experts.

13 MR. CARR: Okay. For everything
14 in the --

15 BY MR. DUPONT:

16 Q. And those criticisms and your
17 analysis of those articles are set forth in
18 your notes and comments that we have marked
19 as Exhibit 4; correct?

20 A. If -- my expectation is that
21 the articles that I cited and for my position
22 are in the notes and comments. Again, I will
23 report, this was not a report and thus it is
24 not complete with every article that supports
25 my opinion. There are general articles that

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 may support my opinion that I have not
3 provided. But these are the ones that I felt
4 were appropriate to provide with my notes.
5 But it is not a report.

6 MR. CARR: Andrew, can I take a
7 shot at this?

8 BY MR. DUPONT:

9 Q. All I want to know, Doctor, is:
10 Have you told me about all the articles that
11 you're relying upon in support of your
12 primary opinions in this case?

13 A. My best answer is, yes. I'm
14 not sure that every article --

15 Q. Okay. Thank you.

16 MR. CARR: Wait a minute, you
17 cut him off.

18 MR. THOMPSON: He's got a
19 qualifier there, sorry.

20 THE WITNESS: I'm not sure that
21 every article has been provided you
22 and there are some times that I have
23 discussed things that I don't have the
24 reference for, but are in my general
25 knowledge. But I have discussed them.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 BY MR. DuPONT:

3 Q. All right. To the extent that
4 you have been able to identify an article
5 that has not been provided, you have
6 identified it for me; correct?

7 A. Yes.

8 MR. THOMPSON: Other than that
9 he has a whole stack right here, dude.

10 MR. DuPONT: No, no, no, no.

11 MR. THOMPSON: These are
12 articles you looked at. What do you
13 mean, no? You know, Andrew, the
14 problem is, like I said, you're in
15 Philly and we are here. And he has a
16 stack of stuff --

17 MR. DuPONT: The problem is I
18 have spent five and a half -- five
19 hours and 45 minutes going through the
20 Doctor's opinion, having the doctor
21 identify what he relies upon for those
22 opinions. And now I have an attorney
23 saying, oh, by the way, let's add
24 these other articles which the doctor
25 on his couldn't identify and now I as

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 the attorney want to identify and help
3 him. That's the problem.

4 MR. THOMPSON: That's just a
5 bunch of crap.

6 MR. DuPONT: That's why I'm
7 saying that --

8 MR. THOMPSON: If you were in
9 the room with us, which most lawyers
10 are for most depositions, you would
11 see the stack that's sitting to his
12 immediate right and you could ask him.
13 The complication is, you're trying to
14 take this remotely, man. And he's
15 also got six boxes --

16 MR. DuPONT: That's not a
17 complication. He's already given his
18 opinions.

19 MR. THOMPSON: It is.

20 MR. DuPONT: Now you're changing
21 them.

22 MR. THOMPSON: It is a
23 complication. They're sitting right
24 here. Do you want him to identify
25 them or not?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 MR. DuPONT: He just answered
3 the question. He just answered the
4 question again.

5 MR. THOMPSON: All right. Well,
6 I'm telling you, there's a stack of
7 articles right here that you need to
8 go through if you want to fully
9 explore the doctor's opinions.
10 There's also six boxes of stuff behind
11 him that he's read and you're not
12 touching any of that, so do whatever
13 you want to do. But I'm telling you
14 right now, we're prepared to continue
15 to let you ask questions. If you
16 don't want to, no problem. Go for it.

17 BY MR. DuPONT:

18 Q. Doctor, you have identified for
19 me and marked as exhibits all of the
20 documents and articles that you have relied
21 upon for your opinions, your primary opinions
22 in this case --

23 MR. CARR: No, because he's got

24 --

25 BY MR. DuPONT:

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. As opposed to your analysis of
3 the plaintiffs' experts; correct?

4 A. I can't limit myself to the
5 things that I brought because this is not a
6 formal report. And there are articles that
7 are in my reasonable background. I have told
8 you about a bunch of CAPM articles that I
9 have not done. There may be other articles
10 in other areas that I have not brought. So I
11 don't want to be limited to the articles that
12 I have brought. But I brought those articles
13 that support that. I can tell you that these
14 --

15 Q. And to the extent that the --
16 MR. CARR: I'm sorry, I'm not
17 going to allow you to cut this one
18 off. Doctor, please finish your
19 answer.

20 BY MR. DuPONT:

21 Q. To the extent --

22 MR. CARR: Doctor, please finish
23 your answer.

24 BY MR. DuPONT:

25 Q. To the extent that you --

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 MR. CARR: The witness is
3 talking, Andrew.

4 THE WITNESS: Of the pile that I
5 have, I believe I have given you the
6 ones that I would consider proactive
7 rather than reactive. However, I
8 would reserve the right to be reactive
9 with the others. And I have
10 brought --

11 BY MR. DUPONT:

12 Q. That's what I'm trying to sort
13 out.

14 A. I have not brought all the
15 articles that would be in the background of
16 my opinions.

17 MR. THOMPSON: You also --

18 BY MR. DUPONT:

19 Q. That's what I'm trying to sort
20 out. To the extent there's an article that
21 supports and you rely upon for your opinions,
22 your primary opinions, your affirmative
23 opinion, we have marked them as exhibits. Or
24 if you haven't brought it with you, you have
25 identified it to the best of your ability;

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 correct?

3 A. May I have that again, please?

4 Q. To the extent that there is an
5 article that you rely upon for support of
6 your affirmative opinion, this would be your
7 critique of the plaintiffs' experts, we have
8 marked those as exhibits. Or to the extent
9 you can identify the article and have not
10 brought it with you, you have identified it
11 for me to the best of your ability. Correct?

12 A. Yes. But I reserve the right
13 to have other articles, which are the basis
14 of my opinion that I have not presented to
15 you because I have not filed a formal report.
16 I have talked generally, I have done my best
17 to quote the articles as specifically as I
18 can. But there may be things that I have
19 missed and if I'm able to identify them and
20 they're part of the body of literature and
21 they're a background to my opinion, I would
22 want to be able to bring them forth.

23 Q. You understand that this is my
24 opportunity to ask you what those articles
25 are.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. And I'm doing my best to
3 answer. That's why I brought the pile and
4 that's why I have answered the best that I
5 can.

6 Q. Okay. And what you have with
7 you in your pile of articles that has not yet
8 been marked as an exhibit, or you did not
9 identify when I asked you what your primary
10 opinions are, that pile of articles are
11 articles that you're using to address the
12 plaintiffs' expert's opinion?

13 A. They would be reactive to the
14 plaintiffs' opinion or to issues that could
15 be brought up whether the plaintiffs' experts
16 brought them up or not.

17 Q. Okay. And are your comments on
18 those articles that you have brought with you
19 and have not been marked yet, and what you
20 are terming reactive or reactive opinions,
21 are your comments on those articles set forth
22 in your written notes and comments?

23 A. Not entirely, no.

24 Q. How many articles do you have
25 left that we haven't discussed?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. We have discussed Gunn, but it
3 has not been made an exhibit. I have two
4 articles that Dr. Williams quoted. Or one
5 article that she quoted regarding psoriasis
6 by ^Godelli. I've got a couple of articles
7 on autoimmune disease related to psoriasis
8 that I don't need. I've got statements as to
9 OSHA and NIOSH levels, which are not
10 important.

11 I've got an NPM1^ article,
12 which is reactive to a comment by Dr.
13 Shadduck, but not of critical value. It's
14 just I disagree with him about an unrelated
15 point.

16 I've got the original case
17 control study by Rushton and by Schnatter.
18 That would be the two other nested case
19 control studies that will be part of the
20 pooled analysis.

21 I've got Trangch^ report by
22 Talbot. I've got ^wynell report, which is
23 reactive to a comment I think by Dr.
24 Harrison. I've got the Zheng article, which
25 is relevant to the psoriasis I believe and

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 was quoted by one of your doctors regarding
3 obesity, but it doesn't talk about obesity.
4 I've got an article about DNA
5 damage, DNA repair, which isn't directly
6 related to it. But it talks about why you
7 may have increased risk of leukemia with age
8 because of decrease in DNA repair. I've got
9 a ^Nielson article which I pulled because I
10 thought that was the article your author went
11 -- Dr. Harrison was referring to, but it
12 wasn't. So it's not relevant. I've got
13 another copy of Rushton. I've got Bonds
14 because your author -- one of your experts
15 pulled it, I think Harrison. I've got Klu^
16 because I think Harrison quote that in terms
17 of effects on the --the only one that is
18 worth discussing at least briefly would be
19 Collins. Dr. Petty I think talked about peak
20 levels that were low, but what he called
21 peak. And I just pointed out that the only
22 relationship that Collins found with peak
23 levels was greater than 100 parts per million
24 times 40 days, which certainly doesn't apply
25 here.

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 Q. Okay. So out of those articles
3 you just went through, the only ones you feel
4 worth commenting on is Collins?

5 A. Right. I also got Irons in my
6 pile. Another article that was quoted
7 regarding psoriasis, but not relevant, and
8 the Shell letter. And Dr. Harrison's notes.
9 That's what I've got.

10 Q. Okay. The Irons study is the
11 study you already told me about. Again, in
12 the 24 percent of patients with benzene?

13 A. Yes. Those haven't been
14 admitted, but some of them have discussed.
15 The only one that hadn't been discussed that
16 I would consider relevant was Collins, which
17 I just told you about.

18 Q. So the ones you consider
19 relevant are Ward, Lan, Irons and Collins?

20 A. Yes. But Ward is -- Ward is
21 relevant, Lan -- yes, those are relevant.

22 Q. Okay. Let's mark Ward. And
23 you have given me your comments on Ward;
24 correct?

25 A. I have given you my comments on

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 ward, yes. And two articles by Lan.

3 - - -

4 (Whereupon the documents were
5 marked, for identification purposes,
6 as Sarna Exhibit Numbers 18, 19, 20
7 and 21.)

8 - - -

9 THE WITNESS: I don't know
10 whether we ever marked the Martyn
11 Smith article as one. I don't know
12 whether you want that marked.

13 BY MR. DuPONT:

14 Q. All right, we'll mark Martyn
15 Smith's article as 22. And you have given me
16 your comments on that; correct?

17 A. Yes.

18 - - -

19 (Whereupon the document was
20 marked, for identification purposes,
21 as Sarna Exhibit Number 22.)

22 - - -

23 BY MR. DuPONT:

24 Q. Does that exhaust the list of
25 articles that you think are relevant and

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 worthy of discussing?

3 A. You had Triebig, we gave you
4 that, I got a second copy of that, yes, in my
5 pile. That would be it.

6 Q. The Triebig we'll make sure
7 that's been marked, and if it hasn't been
8 marked already we'll mark it.

9 Are we now done with the
10 articles in your comments and articles that
11 you believe are relevant and worthy of
12 discussing?

13 A. Certainly we're done with the
14 articles that I have identified to bring with
15 me.

16 Q. Okay. How long does the
17 hematotoxic effect of benzene exposure in the
18 blood remain? In other words, how long do
19 blood counts remain depressed after benzene
20 exposure?

21 A. I don't have an answer from the
22 literature on that. My expectation is that
23 it lasts no more than several days to a week
24 or two, but I don't know that.

25 Q. All right. You don't know one

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 way or the other?

3 A. I have not seen a study that
4 defines it.

5 Q. All right. Those are all the
6 questions I have.

7 I'm sorry, the boxes of
8 documents that you have, are those discovery
9 materials? In other words, deposition
10 transcripts and documents from the
11 defendants?

12 A. Largely they're medical records
13 and transcripts. There are some gas
14 chromatographs which I frankly didn't look
15 at. And there are some benzene content
16 measurements for various products that are
17 there. Certificates of Analysis, and what I
18 gleaned from them is in my written report.
19 But those are there.

20 Q. Have all of your observations
21 based on the defendant documents, I'll call
22 them, the documents that are not transcripts
23 and not medical records and not the articles
24 that we have been going through, have all of
25 your observations based on the defendant

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 documents been set forth in your notes and
3 comments?

4 A. Well, I think the -- I did not
5 mention when you asked me about my
6 qualitative view of exposure, that the
7 Certificates of Analysis showed low levels of
8 contaminant level benzene. And that would be
9 supportive that my exposure was -- that
10 exposure particularly in the last 10 years
11 was trivial.

12 Q. Do you have separated the
13 documents from the defendants that you have
14 reviewed and considered in coming to your
15 opinion?

16 A. Have separated as to how or
17 what?

18 Q. In other words, do you have the
19 documents that you have reviewed and relied
20 upon in coming to your opinions that have
21 come from the defendants, that are not
22 transcripts and are not medical records and
23 are not articles, do you have them in one
24 location, one folder, one stack that we can
25 easily identify?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. No. They're scattered to the
3 winds, as all of my articles were. I got six
4 boxes that are full of stuff in no particular
5 order.

6 Q. Okay. To the extent that you
7 considered information from the documents
8 provided to you by the defendants, have you
9 identified those documents in your report?

10 A. My document, as I mentioned,
11 was notes with comments. And I took notes as
12 I evaluated things. So if they were
13 important to me or relevant that I thought
14 they should have been put on paper they're in
15 my notes. A lot of the things are not
16 important, but were just relevant as note
17 taking as I reviewed things.

18 Q. Okay. I'm not really concerned
19 about whether we characterize Exhibit 4 as
20 your report or your notes and comments. I
21 just want to make sure that if you reviewed
22 and relied upon and found important a
23 document from one of the defendants, did you
24 identify that document in your notes and
25 comments?

1 GREGORY PAUL SARNA, M.D. - ROUGH

2 A. Yes.

3 Q. Do you have any notes that you
4 have made concerning this case that are not
5 set forth in your notes and comments?

6 A. Again, I didn't -- I've quoted
7 to you some articles that may not be listed
8 in my notes. So those I have discussed with
9 you, but they may not be in my notes. Other
10 than that -- and articles that I have not
11 been able to identify, my answer would be,
12 no.

13 Q. Okay. So there's no other
14 document where you have written down
15 observations or made notes as you were going
16 through transcripts or other documents?

17 A. I have some handwritten notes
18 behind me that are chicken scratch and
19 illegible and largely led to them being in my
20 report if they were relevant. I think the
21 accurate answer is that it would be a waste
22 of your time to go through them. But if you
23 want to try to decipher my handwriting,
24 you're welcome to.

25 Q. Well, let's attach them as an

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 exhibit anyway.

3 - - -

4 (Whereupon the document was
5 marked, for identification purposes,
6 as Sarna Exhibit Number 23.)

7 - - -

8 BY MR. DUPONT:

9 Q. Have those handwritten notes
10 been transcribed into your notes and comments
11 that's Exhibit 4?

12 A. Anything that's important, yes.

13 Q. Those are all the questions I
14 have.

15 (Discussion held off the
16 record.)

17 MR. THOMPSON: I propose that we
18 stipulate to relieve the court
19 reporter of duties and
20 responsibilities under the California
21 Code.

22 The original of the transcript
23 will be sent to Dr. Sarna as early as
24 possible, early next week, Monday or
25 Tuesday is the target. Dr. Sarna will

1 GREGORY PAUL SARNA, M.D. - ROUGH
2 have until the 9th of August to read
3 and sign, and we'll alert counsel by
4 letter or notification of any changes
5 that are made.

6 If either that does not happen
7 or the transcript is unavailable at
8 the time of trial, a certified copy
9 can be used for any and all purposes.

10 MR. CARR: So stipulated.

11 MS. DIPONG: So stipulated.

12 MR. DuPONT: So stipulated.

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