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IN THE DISTRICT COURT OF
GALVESTON COUNTY, TEXAS
56TH JUDICIAL DISTRICT

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JOHN RINGSTAFF and KAREN RINGSTAFF,

Plaintiffs, NO. 03CV-0551

-VS-

AMOCO CHEMICAL COMPANY, et al.,

Defendants.

-----X

March 13, 2006

10:45 a.m.

DEPOSITION OF JOHN WHYSNER, M.D. taken
pursuant to Notice and Subpoena, at the
Marriott Hotel, 670 White Plains Road,
Tarrytown, New York, before Anita Shemin, a
Notary Public and Shorthand Reporter within
and for the State of New York.

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A P P E A R A N C E S:

PROVOST UMPHREY, L.L.P.

Attorneys for the Plaintiffs

490 Park Street

Beaumont, Texas 77704

BY: RODNEY BARNWELL, ESQ.

Of Counsel

WERNER & KERRIGAN LLP

Attorneys for the Defendants Marathon Oil Company
and Crown Central Petroleum Company

2000 West Loop Street, Suite 1550

Houston, Texas 77027

BY: PHILIP A. WERNER, ESQ.

1

2 (Exhibit 1, Notice of Deposition and
3 Subpoena, marked for identification, as of this
4 date.)

5 (Exhibit 2, invoices of Washington
6 Occupational Health, marked for identification,
7 as of this date.)

8 (Exhibit 3, summary of medical
9 records, marked for identification, as of this
10 date.)

11 (Exhibit 4, testimony of Dr. Whysner
12 in the past four years, marked for
13 identification, as of this date.)

14 (Exhibit 5, curriculum vitae of Dr.
15 Whysner, marked for identification, as of this
16 date.)

17 (Exhibit 6, documents relating to
18 case, marked for identification, as of this
19 date.)

20 J O H N W H Y S N E R, M.D.,

21 residing at 166 Farrington Avenue, Sleepy
22 Hollow New York 10591, having been first
23 duly sworn, testified as follows:

24 EXAMINATION BY

25 MR. BARNWELL:

1 *J. Whysner, M.D.*

2 Q. Dr. Whysner, we have had a little
3 computer glitch. We are going to start over. I
4 am going to lead you back through these first few
5 exhibits and see if we can recapture what we have
6 already testified about; okay?

7 A. Yes.

8 Q. We have marked Exhibit 1 through 6 at
9 this time.

10 A. Yes.

11 Q. Exhibit No. 1 is the Notice and
12 Subpoena Duces Tecum for your deposition;
13 correct?

14 A. Yes.

15 Q. You have brought with you your file
16 records and some documents that you have
17 generated yourself in the development of this
18 case; correct?

19 A. Correct.

20 Q. Could you identify Exhibit No. 2 for
21 us?

22 A. These are the bills that were
23 generated by the company I work for, Washington
24 Occupational Health, both to Haines & Boone as
25 well as to Werner & Kerrigan to date.

1 *J. Whysner, M.D.*

2 Q. As I understand, you were first
3 contacted about this case at the end of 2004?

4 A. Yes.

5 Q. That was by Stan Perry?

6 A. Yes.

7 Q. You had worked with Mr. Perry one time
8 previously in the Lambert case?

9 A. Yes.

10 Q. You have also stated that since the
11 final bill in Exhibit No. 2, which runs through
12 February 2006, you have done, approximately, 20
13 to 40 hours of additional work?

14 A. Yes.

15 Q. Can you characterize what sort of
16 activities that have made up that 20 to 40 hours?

17 A. Going through my files, organizing
18 them, rereading some of the papers, rereading
19 depositions that I had already looked at and also
20 the medical records.

21 Q. Yes?

22 A. And I had received depositions of Drs.
23 Infante, Gardener, Nadelson since that time.

24 Q. Yes?

25 A. Or since the last billing.

1 *J. Whysner, M.D.*

2 Q. You have also brought with you a
3 document we have marked as Exhibit No. 3?

4 A. Yes.

5 Q. Could you identify Exhibit No. 3?

6 A. Yes. This is a summary that Dr.
7 Scott, Sam Scott, who is at Washington
8 Occupational Health, and I put together regarding
9 Mr. Ringstaff's medical records. It is a summary
10 of our review of the medical records.

11 Q. Do you recall, approximately, when you
12 prepared Exhibit No. 3?

13 A. Well, recently I went over the medical
14 records and made a few changes in this based upon
15 what I saw, but I think this was mostly prepared
16 in -- last year.

17 Q. Do you recall what changes or
18 additions you have made to Exhibit No. 3?

19 A. I think -- I think all I remember was
20 that there was a -- there was a mistake about --
21 oh, I remember the hypogammaglobulin anemia. He
22 had made it sound that he had that in the past
23 rather than he developed that since his
24 chemotherapy, and his disease had developed.

25 Q. And the accurate reading of the record

1 *J. Whysner, M.D.*

2 is that it occurred after chemotherapy?

3 A. That is my understanding when I look
4 back at the medical records, yes.

5 Q. Can you identify what has been marked
6 as Exhibit No. 4?

7 A. This is my testimony at deposition or
8 trial in the last four years.

9 Q. As I understand based upon our earlier
10 conversation, you have testified in,
11 approximately, three cases where there was some
12 allegations of Benzene in some capacity causing
13 an illness; correct?

14 A. Correct.

15 Q. The three cases where you testified
16 about Benzene were Flodin, Hopper and the IBM
17 case?

18 A. Correct.

19 Q. In all of the cases concerning Benzene
20 allegations that you have testified in, you have
21 testified on behalf of defendants; correct?

22 A. Correct.

23 Q. You have also identified a few cases
24 concerning leukemia that you have testified in?

25 A. Correct.

1 *J. Whysner, M.D.*

2 Q. I think one was Flodin.

3 A. Yes.

4 Q. Wasn't there another one?

5 A. There was a case that had to do with
6 alleged PCB exposure, and there were ten -- no, I
7 think there were 13 plaintiffs, and I believe one
8 of them was a leukemia case, but -- but it has
9 been a while, I can't remember.

10 Q. Which case was that, if you recall?

11 A. That was a case -- I don't remember
12 the caption, but it was a case of alleged
13 exposure of PCBs from ceiling tiles. Armstrong
14 World Industries was the defendant.

15 Q. In the two cases that you can recall
16 where you have testified concerning a form of
17 leukemia, you have testified on behalf of
18 defendants in both of those cases as well?

19 A. Correct.

20 Q. It is accurate to say that you have
21 never testified on behalf of a plaintiff in a
22 Benzene related lawsuit?

23 A. Correct.

24 Q. You have never testified on behalf of
25 a plaintiff in a case where the plaintiff had

1 *J. Whysner, M.D.*

2 some form of leukemia; is that correct?

3 A. That is correct.

4 Q. We talked earlier, to the best of your
5 recollection, Doctor, you have never testified in
6 the State of Texas?

7 A. That is correct.

8 Q. And you do not recall testifying in
9 any cases pending in Texas; do you?

10 A. Correct.

11 Q. Exhibit 5, could you identify that for
12 me?

13 A. This is my current CV.

14 Q. Are there any additions that need to
15 be made to make Exhibit 5 updated and complete?

16 A. No, no. There is one book that I
17 realized that I am supposedly -- I had a chapter
18 in it a long time ago, a risk assessment book,
19 which I have realized has been published, but I
20 can't get a hold of a copy of it. I have asked
21 my editor to send me a copy.

22 Q. To confirm that you were actually
23 included in the publication?

24 A. Correct.

25 Q. All right.

1 *J. Whysner, M.D.*

2 Is that listed in Exhibit 5?

3 A. No.

4 Q. What publication was that?

5 A. It was a book about cancer risk
6 assessment, or at least that was my part of it.
7 It was -- the editor was Peter Shields.

8 Q. We have gone through 1 through 6
9 fairly quickly. When we were interrupted on the
10 little hotel emergency, we were talking about
11 Exhibit No. 6 and the extending conversation from
12 that. Can you identify Exhibit No. 6 for us?

13 A. No. 6 includes the documents that we
14 received from Haines & Boone regarding this case.

15 Q. All right.

16 The documents that you received from
17 Haines & Boone, a complete list of those are
18 included in Exhibit No. 6; is that true?

19 A. That is correct, because it was sent
20 first to the office in Washington, and they put
21 this list together, and only later, then did I
22 get the documents from them, and all of the
23 Haines & Boone documents, as far as I know, went
24 through my office.

25 Q. When did you personally receive the

1 *J. Whysner, M.D.*

2 documents that are reflected in Exhibit No. 6?

3 A. It depends on which ones they are.

4 Q. Well, I saw what looked like about two
5 or three different dates.

6 A. Correct. Actually, there are only two
7 dates. There are documents that we got on the
8 25th of May, 2005, and there is one that we --
9 that we received on August 10th, 2005.

10 Q. After August 10, 2005, did you receive
11 any more documents from Haines & Boone?

12 A. Not to my recollection, no.

13 Q. Now, what did you receive -- strike
14 that. I will speed this up a little bit.

15 As I understand your testimony
16 earlier, you received after August 10, 2005,
17 documents from Mr. Werner's firm?

18 A. Correct.

19 Q. What you already told me you received
20 from Mr. Werner's firm are the deposition and
21 exhibits for Dr. Gardener and Dr. Infante?

22 A. I got the one exhibit from Dr.
23 Gardener that he said was in addition to what he
24 had already provided in the Hatton case.

25 Q. I will restate it.

1 *J. Whysner, M.D.*

2 You have received from Mr. Werner's
3 office the deposition and exhibits of Dr.
4 Infante's testimony?

5 A. Correct.

6 Q. You received the deposition of Dr.
7 Gardener, and along with that, you received the
8 Mundt report from Hatton and the Wong study?

9 A. Say that again, please. I am sorry.

10 Q. Sure.

11 You also received the deposition of
12 Dr. Gardener, and included in that was the Mundt
13 report from Hatton and the Wong study?

14 A. I am not sure where the Mundt report
15 is. I have it in my file with Gardener's
16 deposition because I believe he received it, too.
17 I don't remember when I received that, but -- but
18 I am trying to see if it is on this list already.

19 Q. Sure.

20 I will just tell you that the date on
21 there, I believe, was August 24th, 2005, so I
22 doubt --

23 A. Okay.

24 Q. -- I doubt it is on this list.

25 A. It may have been sent to me by Haines

1 *J. Whysner, M.D.*

2 & Boone earlier, and because, as I mentioned,
3 that was in the Hatton case. Now, I received
4 documents from Haines & Boone in the Hatton case
5 that aren't on this list, such as Dr. Gardener's
6 deposition and -- and articles that said Hatton
7 on it, so somehow it wound up in this file even
8 though it is -- it is part of the Hatton
9 documents.

10 So I don't remember when I received
11 that. It may be in this other -- I have a list
12 of documents, I don't know if I brought it with
13 me from the Hatton case.

14 Q. All right.

15 What else have you received from Mr.
16 Werner's office?

17 A. I have received depositions of Dr. --
18 you mentioned Infante and Gardener -- Nadelson,
19 which I don't have a hard copy of. I just
20 received it electronically and reviewed it.

21 Q. Did you print it out to review it?

22 A. No.

23 Q. You reviewed it on the computer?

24 A. Yes.

25 Q. All right.

1 **J. Whysner, M.D.**

2 A. There is one other thing. I received
3 this affidavit of Dr. Infante. I believe I
4 received this from Mr. Werner prior to Dr.
5 Infante's deposition.

6 Q. Anything else?

7 A. No, that is all.

8 Q. Did you receive the deposition of Dr.
9 Kirk?

10 A. No, not in this case.

11 Q. Did you receive it in the Hatton case?

12 A. Yes. Not his deposition, his report,
13 excuse me. Right.

14 **MR. WERNER:** He actually received it
15 on his computer. He may not have received
16 it, yet, but it hasn't been sent to you. I
17 thought it had.

18 **THE WITNESS:** Okay.

19 **BY MR. BARNWELL:**

20 Q. Sitting here today, you haven't had a
21 chance to review Dr. Kirk's testimony?

22 A. Correct.

23 Q. One other thing. We talked about
24 previously your rates. As I understand it, you
25 charge \$500 an hour for all of your work?

1 *J. Whysner, M.D.*

2 A. Currently, that is correct. This
3 year, yes.

4 Q. Did you have a different rate prior to
5 2006?

6 A. I think the company rate was 450.
7 Until this year, I think the bills were 450, and
8 before, for Haines & Boone.

9 Q. Obviously, we can add all of this up,
10 but do you have a recollection of what your total
11 charges have been in this case so far?

12 A. Dollar figure?

13 Q. Yes.

14 A. No.

15 Q. It looks like a little over \$29,000
16 prior to March 2006.

17 A. That is, approximately, correct. 25,
18 something like that.

19 Q. In the month of March, 20 to 40 hours
20 at \$500 an hour would be another 10 to \$20,000
21 this month?

22 A. Correct.

23 Those older bills represent Dr.
24 Scott's time and a little bit of Dr. Chase as
25 well.

1 *J. Whysner, M.D.*

2 Q. Yes?

3 A. As well as office assistants.

4 Q. Other than folks who perform clerical,
5 copying, that sort of type of work, who all has
6 been involved in this case from your firm?

7 A. Well, mostly Dr. Scott reviewing the
8 medical records, and Dr. Chase, his time is
9 probably primarily related to organizing, I
10 think, making sure things happen. Rather than
11 being substantively involved, that is more as his
12 role as President of the company.

13 Q. And Dr. Chase is the President of your
14 company?

15 A. Yes.

16 Q. What is your position there?

17 A. I am the Vice President.

18 Q. Are there other office holders?

19 A. I don't think so.

20 Q. Are you an owner of the company?

21 A. No.

22 Q. Who owns your company?

23 A. Dr. Chase.

24 Q. What is Dr. Scott's position there?

25 A. He is a physician. I forget his exact

1 *J. Whysner, M.D.*

2 title, but -- but he is -- he sees patients. We
3 do a lot of medical surveillance programs, a lot
4 for the federal government, so he does a lot of
5 examinations. He is a board certified internal
6 medicine and occupational medicine.

7 Q. What did Dr. Scott do in connection
8 with this case?

9 A. He was the first one to go over the
10 medical records, and he reviewed them. He wrote
11 a first draft of the -- of the summary of Mr.
12 Ringstaff's's medical condition.

13 Q. What we have marked as Exhibit 3?

14 A. Yes.

15 Q. What else did Dr. Scott do?

16 A. He read over Mr. Ringstaff's
17 deposition as well as it pertained to his medical
18 condition.

19 Q. Anything else?

20 A. No.

21 Q. Exhibit No. 3 was produced fairly
22 early in your work of the case?

23 A. A draft of it was.

24 Q. Did you all continue to review records
25 and update it on a frequent basis, or was it just

1 *J. Whysner, M.D.*

2 a one time review by you subsequent to that?

3 A. No, I reviewed it twice. I reviewed
4 it right after he had done the first part, and
5 then I re-reviewed it recently when I was going
6 over the medical records for the deposition.

7 Q. I think I asked you this a minute ago.
8 Do you recall, approximately, when Exhibit No. 3
9 was made?

10 A. No, I could not tell you exactly. It
11 was probably in the range of -- well, I could
12 tell you from looking at -- see when Dr. Scott's
13 main hours were.

14 Q. Sure.

15 A. I would have to say it was -- it would
16 be before the end of September.

17 Q. 2005?

18 A. Correct.

19 Q. Once you have a summary like in
20 Exhibit No. 3 prepared that Dr. Scott has worked
21 on, what sort of role does he play in the
22 development of the case from your standpoint?

23 A. None.

24 Q. Are there other professionals at
25 Washington Occupational Health Associates that

1 *J. Whysner, M.D.*

2 testify?

3 A. Yes.

4 Q. Who else testifies over there?

5 A. You mean in general?

6 Q. Yes.

7 A. Dr. Scott does, and Dr. Chase has,
8 also.

9 Q. How do you guys decide on who is going
10 to testify on what case?

11 A. Usually it depends upon the -- usually
12 it is not decided by us, it is usually decided
13 exclusively in conjunction with whoever has hired
14 us as to which of our expertise seems to fit in
15 with the case. I am a toxicologist, I am also a
16 physician. I am probably -- I am the most
17 familiar with the scientific literature on issues
18 regarding certain clinical issues. Treatment
19 matters, Dr. Scott would be the person who would
20 testify in those kind of cases.

21 Dr. Chase also, he has been involved
22 in some PCB. We both testified together on
23 certain PCB litigations, for example, where he
24 testifies from the standpoint of an occupational
25 medicine physician, and I have testified as a

1 J. Whysner, M.D.

2 toxicologist.

3 Q. Have you ever been in a private
4 medical practice?

5 A. No.

6 Q. In other words, have you ever had a
7 relationship where you had a group of patients
8 that came to see you on a regular and routine
9 basis as their treating doctor?

10 A. In two instances, I did. I was at the
11 National Institute of Health, and I got my
12 medical license, and when I was there, I began
13 treating people at a free clinic that was in a
14 church. So, I would go every -- once or twice a
15 week in the evenings and see a group of patients.
16 This was pediatrics.

17 I did the same in rural Pennsylvania,
18 I had a home there, and I also had my consulting
19 firm, which was Medical Research Applications in
20 Washington, but I would go up there every Friday,
21 and I would participate in pediatric clinics up
22 there, and I sort of had a group of my own
23 patients in that setting.

24 Q. When was the last time that you acted
25 as a doctor to a group of patients?

1 J. Whysner, M.D.

2 A. It has been 20, 30 -- 20 years, I have
3 been involved in doing medical surveillance work,
4 but we are not actually their physicians, we are
5 -- we are looking for something.

6 Q. The time period that you are talking
7 about would be the '71 to '74 time period?

8 A. Whenever my medical license -- I let
9 my medical license in Pennsylvania expire. That
10 would probably be when I ended. I think that is
11 on there.

12 Q. Okay.
13 1973 to 1976?

14 A. Correct.

15 Q. So, at the latest, 1976 would have
16 been the last time you were a treating doctor to
17 a group of patients?

18 A. Yes.

19 MR. BARNWELL: Off the record.

20 (Brief Recess)

21 BY MR. BARNWELL:

22 Q. Doctor, are you ready?

23 A. Yes.

24 Q. Did you bring some other documents
25 with you?

1 *J. Whysner, M.D.*

2 A. These are just the original letters
3 from Haines & Boone, retention letters that we --
4 that are routine materials.

5 A letter from --

6 Q. What else do you have?

7 A. A letter from Mr. Werner. I think
8 this is just -- this is unsigned version of the
9 letter that is part of this, the retention
10 letter.

11 Q. Do you recall whether or not you
12 personally spoke with Mr. Perry when he first
13 contacted your firm?

14 A. In this case?

15 Q. Yes, sir.

16 A. Yes, I think so.

17 Q. Were you retained in Ringstaff first
18 or Hatton first?

19 A. They were together.

20 Q. When you talked with Mr. Perry, what
21 did he tell you he envisioned your role to be in
22 the litigation?

23 A. I certainly know he wanted me to
24 testify whether or not Benzene exposure could
25 cause CLL.

1 *J. Whysner, M.D.*

2 Q. You think that is something he talked
3 to you about in your initial conversation?

4 A. Correct.

5 I -- I believe that he wanted us to
6 talk about whether or not Mr. Ringstaff's -- what
7 his other potential medical conditions were, what
8 other risk factors he might have related to CLL
9 and based on his medical records.

10 But beyond that, I don't specifically
11 -- in fact, I am -- I don't recall any specific
12 conversations, but I believe that is based upon
13 what I have seen in my files, that that is what
14 he wanted us to do.

15 Q. What is it that you have seen in your
16 file that makes you believe that?

17 A. The fact that we got all of the
18 medical records, and we reviewed the medical
19 records and -- and looked in the summary, we --
20 we summarized his -- looked for his body weight,
21 for example, to see if he was an obese
22 individual, looked at his smoking history, looked
23 at his other aspects of his history.

24 Q. I am going to mark as Exhibit No. 7,
25 the letter that you have given me.

1 *J. Whysner, M.D.*

2 A. This is the unsigned one, by the way.

3 Q. That is fine.

4 This is an exact copy of the --

5 A. I want to make sure that that wasn't
6 for some reason subsequently --

7 Q. Let's just mark as Exhibit No. 7 the
8 signed copy to make it complete.

9 (Exhibit 7, a letter, marked for
10 identification, as of this date.)

11 A. You want the whole package here?

12 Q. Yes, sir.

13 A. Okay.

14 Q. When you talked with Mr. Perry
15 initially in December 2004, did you express an
16 opinion to him about Benzene and CLL?

17 A. I don't recall.

18 Q. Do you know if Dr. Chase expressed an
19 opinion to Mr. Perry concerning Benzene and CLL?

20 A. No, I am not really sure that we were
21 together when we talked to Mr. Perry, and --

22 Q. Do you know if Dr. Scott had a
23 conversation with Mr. Perry in December 2004
24 where he expressed an opinion?

25 A. I don't think Dr. Scott ever spoke to

1 *J. Whysner, M.D.*

2 Mr. Perry. I think all of that communication was
3 between me and Dr. Scott.

4 Q. All right.

5 But now, Exhibit 7, the letter is from
6 Dr. Chase to Mr. Perry?

7 A. Correct.

8 Q. But you don't know if Dr. Chase and
9 Mr. Perry ever talked about the connection
10 between Benzene and CLL?

11 A. I don't know, but I -- I don't
12 actually think so because that is our routine and
13 our office's, if somebody contacts me, I ask Dr.
14 Chase to send a letter, which I usually review,
15 but because he is President of the company, he
16 sends the letter, gets the approval and then
17 initiates the paperwork, and the billing codes
18 and so forth.

19 Q. Do you have any other documents here
20 with you on the table that we haven't talked
21 about, yet?

22 A. This is the project initiation form
23 for when I was -- when I was hired by --
24 actually, Philip Werner called me, I must have
25 talked to him earlier than I already indicated.

1 *J. Whysner, M.D.*

2 This has a start date of October 20, 2005, but --
3 but I didn't really do any work on the case. He
4 just said that he wanted to -- this was right
5 after Mr. Perry wasn't involved in the case
6 anymore, and he called me and said I want you to
7 stay on the case. This is just a copy of this
8 list of things. This is your notification letter.
9 It was a letter that I received from Mr. Werner
10 about the dates of the --

11 Q. Of the trials?

12 A. These are the three sheets summarizing
13 the studies from my notes.

14 Q. Okay.

15 What else do you have?

16 A. This is just a page that for some
17 reason wound up in this general file. This is a
18 page from the ATSDR tox profile on Benzene
19 exposures, and this relates to the concentrations
20 in the air at which symptoms are felt.

21 Q. Can I see that, also?

22 Now, the documents that I just gave
23 back to you, the summary of the cases and then
24 the Benzene exposure health effects chart that
25 you did, those are documents that you put

1 J. Whysner, M.D.

2 together?

3 A. No. This one is from the ATSDR tox
4 profile, this page, Page 21. These are -- these
5 other three documents, I put together, and the --
6 the -- they represent some of the papers that are
7 in my files.

8 Q. You have a couple of boxes here. What
9 else do we have?

10 A. Those are all the medical records in
11 that box down there that I received.

12 Q. Have you personally reviewed these
13 medical records?

14 A. Yes.

15 Q. Is it part of your practice to make
16 notations on the medical records?

17 A. No. Usually I put those little
18 Post-Its on there for pages that I might want to
19 go back and take a look at again.

20 Q. Did you take any notes concerning Mr.
21 Ringstaff's medical records?

22 A. The only notes are the summary that I
23 already gave to you.

24 Q. All right.

25 There is no in between work product

1 J. Whysner, M.D.

2 that you create?

3 A. No.

4 Q. On the front of each of these
5 sections, there is a little notation here. It
6 looks like it is some sort of accounting system
7 or original system you have.

8 A. That should correspond to the
9 numbering on that sheet that you have called Our
10 Documents Status Sheet.

11 Q. What else do we have in that box?

12 A. EPA risk assessment guidelines.

13 Q. What else do you have?

14 A. Okay.

15 I have three files of research
16 articles, literature -- well, more than that,
17 actually. Three files of -- I will call them
18 research articles. One of them is A through H,
19 one of them is I through P, and one of them is R
20 through Z.

21 Then I have some IARC, International
22 Agency for Research on Cancer. These are
23 complete monographs or excerpts from -- I have --
24 I have a file that contains three Health Watch
25 documents, I have a file that contains some

1 *J. Whysner, M.D.*

2 general references on CLL.

3 And other than that, they are all
4 listed in -- they are depositions or legal papers
5 that we have already discussed.

6 Q. And there are no notations made on
7 those copies that you have there?

8 A. Of these articles?

9 Q. Yes.

10 A. Well, I have Post-Its.

11 Q. I see them, right.

12 A. On a couple of them, there is a -- a
13 notation, but --

14 Q. But, generally speaking, it is not
15 your practice to make notes on the documents of
16 the case?

17 A. No, no notes. I might underline
18 something.

19 Q. Having reviewed Mr. Ringstaff's
20 medical records, what do you believe his
21 diagnosis is?

22 A. Chronic lymphocytic leukemia.

23 Q. You don't question that diagnosis?

24 A. No.

25 Q. Have you had a chance to evaluate what

1 *J. Whysner, M.D.*

2 types of treatments he has received as a result
3 of his diagnosis?

4 A. That is part of that medical summary.

5 Q. As a physician, do you question any of
6 the treatments he has received?

7 A. No.

8 Q. Do you think his treatments were
9 necessary to battle the illness he had?

10 A. I don't have an opinion about this.

11 Q. Obviously, I don't think you are going
12 to be coming down to question the reasonableness
13 of the charges, I assume?

14 A. No.

15 Q. Do you consider yourself an
16 occupational medicine doctor?

17 A. Well, in certain aspects, I wouldn't
18 see patients, but I have done medical
19 surveillance work, I have participated in
20 meetings, I am part of the organization, I have
21 served on one of the committees, Environmental
22 Medicine Committee of the -- of the American
23 College of Occupational and Environmental
24 Medicine when it took on the environmental
25 medicine part.

1 **J. Whysner, M.D.**

2 So, in some regards, I wouldn't
3 consider myself to be the kind of occupational
4 medicine physician who would participate in -- in
5 a practice of people who are occupationally in an
6 occupational environment. I am a toxicologist as
7 that relates to occupational medicine, and that
8 is part of occupational medicine. That is the
9 part that I participate in.

10 Q. You are not a treating doctor of
11 occupational illnesses?

12 A. Correct.

13 Q. You have had a chance to read Mr.
14 Ringstaff's deposition. What have you assumed
15 his work history to be?

16 A. Well, he worked at a number of
17 different facilities. I -- I didn't assume, this
18 is what I have gathered from what I have seen.
19 He worked in a number of --

20 **MR. BARNWELL:** Let's go off the record
21 real quick. I am sorry.

22 (Brief Recess)

23 **BY MR. BARNWELL:**

24 Q. Doctor, do you recall which facilities
25 Mr. Ringstaff worked at based upon his testimony?

1 *J. Whysner, M.D.*

2 A. Vaguely. I mean, I know that he
3 worked at a Shell facility and a Marathon
4 facility, and I know he went to work eventually
5 for Amoco, I believe in 1978, but I don't
6 specifically recollect all of it.

7 Q. Sitting here today, do you have any
8 reason to dispute that Mr. Ringstaff worked as a
9 contractor at Marathon or Crown Central
10 Petroleum?

11 A. No.

12 Q. Have you made any assumptions about
13 Mr. Ringstaff's exposure levels to Benzene, or
14 does that really even matter, in your opinion?

15 A. Well, both of those are correct, I
16 haven't and it doesn't matter.

17 Q. You have not made any assumptions
18 about his exposure to Benzene; is that correct?

19 A. I mean, I have looked at his exposures
20 generally in terms of how they might relate to
21 things that have happened in -- in the scientific
22 literature. Like, for example, the Pliofilm
23 Cohort, based upon what I saw in his deposition
24 testimony, I would suspect that his exposures
25 would be less than many of the people who were in

1 *J. Whysner, M.D.*

2 that study based upon what I read about their
3 activities related to Benzene.

4 Q. You are not an industrial hygienist;
5 correct?

6 A. No, but I have to review industrial
7 hygiene data as a toxicologist because, of
8 course, exposure issues are very important in
9 toxicology.

10 Q. Other than forming the opinion that
11 Mr. Ringstaff's exposure was less than some of
12 the people in the Pliofilm, and I would assume
13 you would agree was possibly higher than some of
14 the people in the Pliofilm study, could you get
15 any more specific than that?

16 **MR. WERNER:** Object to the form.

17 A. Could you repeat that?

18 Q. I will reask it.

19 You stated a moment ago that you
20 believe Mr. Ringstaff's exposure levels would
21 have been less than some of the people in the
22 Pliofilm. I think that is what you said;
23 correct?

24 A. Yes.

25 Q. Would it be fair to say that his

1 *J. Whysner, M.D.*

2 exposure could have been higher than some of the
3 members of the Pliofilm Cohort, also?

4 A. I don't know.

5 Q. Have you made any calculations to
6 determine a range of exposure that you personally
7 would assign to Mr. Ringstaff concerning Benzene?

8 A. No.

9 Q. Have you made any conclusions about
10 Mr. Ringstaff's Benzene exposure other than what
11 you have already told me?

12 A. No conclusions.

13 Q. Do you intend to rely upon Mr. Spencer
14 to evaluate the overall exposure levels of Mr.
15 Ringstaff?

16 A. Yes.

17 Q. What risk factors do you believe exist
18 for the development of CLL?

19 A. Well, there -- there is -- there is
20 pretty clearly a risk factor of ethnicity. CLL
21 is one of the -- is probably the leukemia that
22 seems to differ in background rates around the
23 world the most.

24 Q. Can you explain which ethnic
25 backgrounds are more susceptible to CLL or less

1 J. Whysner, M.D.

2 susceptible --

3 A. Europeans are more susceptible, and
4 people in the Asian countries seem to be less
5 susceptible to CLL, at least Chinese, Japanese,
6 you know, that part of Asia.

7 Family history is important.
8 Certainly, if somebody has had CLL in their
9 family, that appears to be a risk factor. So,
10 there is a genetic component to CLL, which seems
11 to be maybe the most predominant risk factor that
12 has been established.

13 Q. Any other risk factors?

14 A. Some of the literature has reported
15 obesity for being a risk factor for CLL. There
16 has been literature reporting smoking as being a
17 risk factor for CLL. I think that for both of
18 those cases, it is not clear whether or not it is
19 truly a risk factor, but there is some literature
20 that makes some associations.

21 And other than that, most of the cases
22 of CLL are just not known. Of course, age is
23 another important risk factor, and -- and as
24 somebody gets older, and being a male versus a
25 female, so it tends to be a disease of older men.

1 *J. Whysner, M.D.*

2 Q. Is there an age where you believe that
3 the risk begins to substantially increase?

4 A. It seems to be maybe in the mid 40's
5 or starting around 50 that -- that -- I mean, the
6 curve starts to go up from what I remember.

7 Q. Are you relying upon any studies to
8 indicate age as a risk factor for CLL?

9 A. Well, no particular studies, but I
10 believe that some of the general articles that I
11 have about CLL discuss this matter.

12 Q. Can you identify any for me?

13 A. This is called "Chronic Lymphocytic
14 Leukemia," excuse me, and the authors are Rai and
15 Keating.

16 And this is from a publication of the
17 American Cancer Society called "Cancer Medicine."
18 It is Chapter 130. And on the first page, they
19 have Figure 130-1, which shows CLL age at
20 diagnosis and rate per 100,000, and this starts
21 to go up perceptibly from the zero line at about
22 45 to 49, and then it goes up higher at 50 to 54,
23 and then it gets even higher at 55 to 59 and then
24 it keeps on going up.

25 Q. If I am reading the chart correctly,

1 **J. Whysner, M.D.**

2 the study by Rai and Keating, it looks like it
3 elevates up to about five per 100,000 at the age
4 of 55 to 59; is that correct?

5 A. Yes, that is correct.

6 Q. What is the level at about 59 to 65?

7 A. Twelve per 100,000.

8 Q. It is more than twice the rate of 55
9 to 59; correct?

10 A. Correct.

11 Q. What is the highest reported rate on
12 the chart?

13 A. Thirty. Around 25 to 30.

14 Q. Per 100,000?

15 A. Correct.

16 Q. What age is that group?

17 A. 85 plus.

18 Q. All right.

19 A. Then there is an article, Chapter 92,
20 called "Chronic Lymphocytic Leukemia" by James
21 Johnston, Wintrobe. This is from him, and --

22 **MR. BARNWELL:** Let me go ahead and
23 mark this Rai and Keating article as Exhibit
24 No. 8.

25 (Exhibit 8, article by Rae and

1 *J. Whysner, M.D.*

2 Keating, marked for identification, as of
3 this date.)

4 A. This article has a Figure 92-1, and
5 this has age again, and it has percent, so it is
6 a little bit different, age distribution in
7 chronic lymphocytic leukemia, and this is talking
8 about various authors and so forth.

9 Q. Which volume of Wintrobe is that,
10 which edition?

11 A. Two.

12 Q. All right.

13 A. And this is 2004, so I guess that is
14 the 11th Edition.

15 Q. I hope I didn't interrupt you, but
16 were you going to say what -- well, that is fine,
17 the chart speaks for itself, I guess.

18 Do you know what the background rate
19 per 100,000 is for the development of CLL?

20 A. Again, it depends upon age. So, that
21 chart -- I am sorry, the one by Rai and Keating
22 is showing the background rate at different ages.

23 Q. Do you know what the overall average
24 rate per 100,000 is in the United States for the
25 development of CLL?

1 *J. Whysner, M.D.*

2 A. Not offhand.

3 Q. All right.

4 A. I don't remember whether any of these
5 other articles actually address that issue.

6 Q. Do you believe, within a reasonable
7 medical probability, that obesity contributes to
8 cause CLL?

9 A. I think it is -- it is -- I don't know
10 whether to say probable or possible. Like I say,
11 I have seen three articles now, and I know that
12 one of them has shown it. This one, I have in my
13 file, and two of them have not.

14 I would say it is possible, but I
15 would not conclude to a reasonable degree of
16 medical certainty that CLL is associated with
17 obesity, but like I say, I think there may be a
18 difference of opinion out there regarding that.

19 Q. I think my question was a little
20 muddled. No, I am okay.

21 I am not trying to mince words with
22 you, Doctor, but I have to understand what you
23 are or are not going to testify to at the time of
24 trial. And the test in Texas is reasonable
25 medical probability, so what I need to understand

1 *J. Whysner, M.D.*

2 is are you going to come down and testify that
3 obesity, within a reasonable medical probability,
4 caused or contributed to cause Mr. Ringstaff's
5 CLL?

6 A. No.

7 Q. Do you believe, within a reasonable
8 medical probability, that Mr. Ringstaff's smoking
9 history caused or contributed to his development
10 of CLL?

11 A. No.

12 Q. Have you seen any evidence in Mr.
13 Ringstaff's family history?

14 A. His father died of some cancer of some
15 unknown causes, but other than that, I don't see
16 anything. If that was CLL, that would have been
17 significant.

18 Q. But we don't have any evidence that
19 that cancer was CLL; do we?

20 A. I haven't seen any.

21 Q. Do you plan to assume that he died of
22 CLL?

23 A. No.

24 Q. So, based upon the evidence in this
25 case, do you believe, within a reasonable medical

1 *J. Whysner, M.D.*

2 probability, that a family history of CLL existed
3 in Mr. Ringstaff's family?

4 A. I don't know that. As I have
5 indicated, I don't know what his father died of,
6 so I cannot say one way or the other.

7 Q. Sitting here today, you are not
8 prepared to point to a family history of CLL as a
9 contributing factor for Mr. Ringstaff's
10 development of CLL; are you?

11 A. No.

12 Q. What risk factors do you believe were
13 present for Mr. Ringstaff for the development of
14 CLL?

15 A. Well, certainly the only -- well, two
16 risk factors. One is that he is -- I don't know
17 his exact ethnicity, lineage, I am assuming that
18 he is European, possibly Scandinavian descent,
19 and that, as I mentioned before, has a much
20 higher level of risk of CLL than do people who
21 are Asian.

22 His age contributes. He is at the --
23 at the -- as we saw in the diagram where people
24 begin to get CLL, but certainly people who are
25 below 40 don't seem to have any significant

1 *J. Whysner, M.D.*

2 incidence of CLL. So, in his case, I would say
3 age and ethnicity are the two major factors.

4 Q. Is there any specific European descent
5 within the continent that you can point to as
6 having a higher incidence of CLL?

7 A. I think there are some differences,
8 but I don't recall exactly what they are, but
9 they are not as big as the ones between Asians
10 and Europeans.

11 Q. You recognize that Benzene causes AML;
12 don't you?

13 A. Yes.

14 Q. Are there any other illnesses that you
15 associate with Benzene exposure?

16 A. With AML, it also depends on the dose
17 level, that is a factor.

18 Myelodysplastic syndrome, or MDS, is
19 also associated with Benzene, and especially as
20 it preceded the development of AML.

21 Q. What about aplastic anemia?

22 A. Yes, aplastic anemia. Pancytopenias,
23 I was before talking about -- well, yes, that is
24 correct.

25 Q. Anything else besides AML, aplastic

1 *J. Whysner, M.D.*

2 anemia and the pancytopenias that you talked
3 about?

4 A. Can you repeat your question?

5 Q. I want to make sure you have finished
6 your list of illnesses that you believe can be --

7 A. Symptoms that can be caused by Benzene
8 exposure, but I cannot think of any other
9 illnesses.

10 Q. What do you mean by that? I don't
11 fully follow it.

12 A. For example, if somebody breathed too
13 much Benzene, they could become light-headed, and
14 we looked at the chart that -- indicating where
15 light -- headaches, some things that a patient
16 would report, but -- but would not necessarily be
17 considered to be an illness.

18 Q. Have you looked into the issue of
19 chromosomal abnormalities as it is related to
20 Benzene exposure?

21 A. Yes.

22 Q. Do you believe there is required to be
23 some sort of cytogenetic abnormalities or
24 chromosomal damage, such as deletion of the five
25 or seven chromosomes in order to say that the

1 J. Whysner, M.D.

2 leukemia is Benzene related?

3 A. That is a multi-part question.

4 MR. WERNER: I am going to object to
5 form.

6 BY MR. BARNWELL:

7 Q. Mr. Werner has objected, but you said
8 what you said. Give us your best shot.

9 A. I think I have to say no to the whole
10 thing, but there are parts of it I might agree
11 with.

12 MR. WERNER: His answer is as clear as
13 your question.

14 MR. BARNWELL: You get what your ask
15 for.

16 BY MR. BARNWELL:

17 Q. Is there a specific chromosomal
18 abnormality that you relate to Benzene exposure?

19 A. Well, certainly some have been
20 described, but whether or not there is a specific
21 or certain one, or whether or not there is a --
22 it is necessarily related to Benzene exposure is
23 uncertain.

24 But people have described things like,
25 for example -- I hope I don't get it wrong -- I

1 *J. Whysner, M.D.*

2 believe it is an 11 to 23 translocation that has
3 been associated with Benzene exposure. Like I
4 say, there are a number of different cytogenetic
5 abnormalities that have been described in some
6 people, but trying to draw a complete linkage of
7 that to the AML has been difficult.

8 Q. Sitting here today, do you believe
9 that a detectable chromosomal abnormality is
10 required before someone can say this disease such
11 as AML is related to Benzene?

12 A. No. I would say no. There are so
13 many different abnormalities that are already
14 associated with AML, I think it becomes
15 difficult.

16 Q. Have you looked into the issue of --
17 would that answer be the same for CLL as well?

18 A. I would have to say I don't think
19 Benzene causes CLL, so that -- so, any
20 abnormalities that one might find in somebody
21 with CLL, I would not attribute to Benzene.

22 Q. When someone has CLL, what is the
23 disease process that is going on in their body?

24 A. Well, it is basically an
25 overproduction of lymphocytes by the bone marrow.

1 *J. Whysner, M.D.*

2 Q. In layman's terms, what does that
3 mean? What is the role of lymphocytes in the
4 body?

5 A. This is a -- there are T lymphocytes,
6 and there are B lymphocytes. B lymphocytes are
7 responsible for -- some of them are responsible
8 for producing immunoglobulins that are -- that
9 are sort of the antibodies which then will attack
10 infections. They are responsible for what is
11 called -- therefore, they are responsible for
12 what is called acquired immunity, which means
13 that when you give somebody, for example, a
14 vaccination or challenge allergy patients, they
15 are the ones that are responsible for producing
16 the antibodies that will neutralize the antigens.
17 They serve an immune function.

18 Q. With someone who has CLL, there are
19 too many of these being produced; is that what
20 you said?

21 A. Correct.

22 Q. Where are the lymphocytes produced in
23 the body?

24 A. They are produced in the bone marrow.
25 It depends on the age.

1 *J. Whysner, M.D.*

2 Lymphocytes are also produced in the
3 liver in fetuses. They are produced in the
4 thymus in young children and even in adults, but
5 much less so.

6 Q. In the average adult, 40, 50, 60 year
7 old person, what part of the body is producing
8 the lymphocytes then?

9 A. Primarily the bone marrow.

10 Q. When someone has AML, what is the
11 disease process that is going on at that time in
12 their body?

13 A. Well, with AML, there is a different
14 line of cells that are also produced by the bone
15 marrow. These are called myelogenous cells.
16 These are cells that have a different function.
17 There are cells that, for example, will act as
18 what are called macrophages cells that will
19 actually attack bacteria or viruses and engulf
20 them and destroy them. There are also the red
21 blood cells which carry oxygen that are
22 myelogenous cells. So, in AML it is the cell
23 lineage that produces those types of cells that
24 is involved in the leukemia process.

25 Q. Where are these blood cells made in

1 *J. Whysner, M.D.*

2 the body, the myelogenous cells?

3 A. Bone marrow.

4 Q. You said they are involved in the
5 leukemic process. Is that an excess number of
6 myelogenous cells?

7 A. Correct.

8 Q. Is it your opinion, Doctor, that it is
9 biologically implausible that Benzene could cause
10 CLL and AML? Strike that. I will reask that
11 question.

12 Do you believe, or is it your opinion
13 that it is biologically implausible for Benzene
14 to cause CLL?

15 A. Based on what I have seen in the
16 medical literature, I don't see any indication
17 that it causes CLL and that is biologically
18 implausible as far as I am concerned.

19 Q. I understand that you are looking at
20 the subsequent evidence of, or you are basing
21 your opinion on subsequent evidence studies that
22 have shown, in your opinion, an absence of CLL in
23 Benzene exposure, generally correct?

24 A. That is part of it, yes.

25 Q. But the part I am asking about is the

1 *J. Whysner, M.D.*

2 way that Benzene attacks the body and the disease
3 process that is involved in CLL. Do you believe
4 that it is biologically implausible for Benzene
5 to cause the disease process that is present
6 during CLL?

7 A. Yes.

8 Q. Why is that?

9 A. Well, because the best indication that
10 we have, the way in which Benzene causes acute
11 myelogenous leukemia is through a process called
12 topoisomerase inhibition. In fact, topoisomerase
13 II, Roman Numeral II inhibition.

14 Now, what we see also in people that
15 have secondary leukemias who have received
16 topoisomerase inhibitors, we see the development
17 of acute myelogenous leukemia, and we don't see,
18 at least in terms of any kind of substantial
19 numbers, any other forms of secondary leukemias
20 produced by that process.

21 So, right there, we know that there is
22 a -- some kind of a specificity, and these are
23 people who are treated by physicians, and so we
24 know what they are receiving and we know that
25 they are getting secondary leukemias.

1 J. Whysner, M.D.

2 So, there is clearly a big
3 differential and maybe an absolute differential
4 for some drugs that cause AML through this
5 mechanism that do not cause any -- don't cause
6 other forms of leukemias.

7 As I mentioned, at least from my
8 review of the genotoxicity literature of Benzene,
9 it fits the best with -- with the mechanism being
10 an inhibition of topoisomerase II. This is --
11 this is in laboratory studies, studies in animals
12 and in humans.

13 Q. Other than for litigation purposes,
14 what research have you done into toxicology
15 related to Benzene?

16 A. When I was at the American Health
17 Foundation, I had a research laboratory, I did a
18 pretty wide range of studies that had to do with
19 looking at things that caused lymphopoietic
20 neoplasms or cancer. Our work was in
21 experimental animals or in -- in tissue cultures.

22 During that time, I did some
23 laboratory work related to the issue of Benzene
24 exposure to see if it was directly reacting with
25 DNA, and, also, I did this review of 1,400

1 *J. Whysner, M.D.*

2 genotoxicity studies that were available from the
3 literature to see -- to see how they fit in terms
4 of what mechanism was actually causing the
5 genotoxicity of Benzene.

6 Q. It would be accurate to say, wouldn't
7 it, Doctor, that Benzene has not been a chemical
8 that you have specifically focussed on throughout
9 the duration of your career; is that correct?

10 A. At the last part of my career in the
11 laboratory, yes, I did focus on Benzene, along
12 with a couple of other chemicals.

13 Q. And how long was that?

14 A. I would say probably about in the last
15 five years.

16 Q. As a scientist, when you want to
17 evaluate whether or not a chemical is causing a
18 specific illness or disease, take me through the
19 types of literature that you would go out and
20 review in making your determination.

21 A. I assume that we are talking about
22 human disease, so the primary literature I would
23 be relying upon would be studies in humans.

24 Q. Okay.

25 For example, would you look at

1 J. Whysner, M.D.

2 epidemiology studies?

3 A. Correct.

4 Q. Would you look at case studies?

5 A. It depends if there are lots of
6 epidemiology studies around. One doesn't need to
7 look at case studies. That is usually what
8 prompted the epidemiology studies to be
9 developing, and case studies are not really
10 reliable.

11 Q. Anything else?

12 A. The issues -- some of the issues that
13 we are talking about, animal tests are used. I
14 use them primarily to try to determine what the
15 mechanism is, if indeed we have found a chemical
16 to cause a particular human disease.

17 There have been occasions where --
18 where animal tests have pointed to a particular
19 disease, and, therefore, then we usually try to
20 follow that up with human studies to see if that
21 is true or not. Of course, there are a lot of
22 times they will indicate a disease that isn't
23 found, and maybe a different disease is found.
24 So, there is a species to species reliability
25 issue.

1 *J. Whysner, M.D.*

2 Then we would also look to certain
3 test tube studies, like genotoxicity studies, to
4 give us some indication about whether or not some
5 -- something could be related to a disease
6 entity. But, again, these are all clues to try to
7 lead us up to what we should look for in humans.

8 Q. You have used the phrase human studies
9 a couple of times, and we specifically mentioned
10 epidemiology studies. What other human studies
11 would you look to?

12 A. Certainly to develop a causal
13 relationship between, say, a chemical exposure
14 and disease, one would need epidemiology studies
15 to be able to confirm that. One might look at
16 clinical studies, certainly if they are done in a
17 clinical setting.

18 Like, for example, I used to do
19 clinical trials where I would look for treatment
20 by pharmaceuticals for certain conditions. In
21 those studies, sometimes you would come up with
22 adverse reaction on clear toxicity.

23 Q. Obviously, there are no clinical
24 studies available for Benzene that we are talking
25 about?

1 *J. Whysner, M.D.*

2 A. That is true. I think you asked the
3 question in general, so --

4 Q. Yes, I did, and I am just wanting to
5 make sure I am clear about what you are talking
6 about.

7 A. Correct.

8 Q. Anything else?

9 A. That is it, yes.

10 Q. Let's look through the studies that
11 you brought; okay?

12 A. Yes.

13 Q. You have them -- you have some up here
14 right now?

15 A. These are general articles relating to
16 CLL. They are book chapters.

17 Sorry. You mentioned a risk factor.
18 We talked about risk factors for CLL, and I
19 forgot to mention that at least some people also
20 would put exposures to Agent Orange as a risk
21 factor for CLL, and the National -- the National
22 Academy of Sciences has made that -- talked about
23 that being a risk factor, Vietnam veterans, but
24 some other groups have not really come to that
25 conclusion, although there are studies indicating

1 *J. Whysner, M.D.*

2 that it is not relevant to this case.

3 Q. Do you have opinion as to Agent
4 Orange's ability to cause CLL?

5 A. I think that the evidence is -- again,
6 this is in the probable/possible category, and I
7 could not give you an opinion about that right
8 now, I would like to see what some of the other
9 groups -- other additional studies, what -- what
10 the IARC might do with it, or the EPA or -- but
11 they have not come to a conclusion regarding
12 that.

13 Q. All right.

14 This is what I am going to do. Let me
15 just start with the running question I am going
16 to have for you for your articles; that is going
17 to be, identify the article for me and tell me
18 why it is relevant to your opinions in this case.

19 A. Okay.

20 An article by Kipps, and this is
21 called "Chronic Lymphocytic Leukemia and Related
22 Diseases," and this is Chapter 98 in Williams
23 Hematology, 6th Edition.

24 The only real relevance with this, it
25 is describing all of the chromosomal

1 *J. Whysner, M.D.*

2 abnormalities that have been found with CLL to
3 show that they are really quite diverse.

4 There has been some testimony in the
5 literature regarding the relationship of CLL and
6 a subtype of NHL, and -- and let me just say this
7 about all of the articles that discuss this, just
8 to -- just to maybe -- some of these articles --
9 some of these general review articles, the author
10 clearly says that NHL, and a subtype of NHL and
11 CLL are a spectrum of the same entity or closely
12 related. Others are more circumspect about that
13 and talk about them as being separate disease
14 entities.

15 So, the literature seems to be
16 somewhat divided on whether or not indeed, and
17 people's opinions seem to be divided on whether
18 or not indeed this one group, this small cell
19 lymphoma is a subtype of CLL. That is the main
20 reason for this.

21 Q. Do you have an opinion on that issue?

22 A. I think the jury is still out on that
23 issue. I think that where it comes up in this
24 case is what to do with the NHL literature, but
25 the issue here is that the -- that that group of

1 J. Whysner, M.D.

2 NHL, that subgroup that could be related to CLL
3 is only like 6 to 10 percent of total NHL cases.
4 That is another thing in these articles.

5 The NHL literature, therefore, cannot
6 be used to make a determination of whether or not
7 CLL is associated with Benzene because the --
8 almost the vast majority of NHL cases are not
9 that particular type of NHL that even some people
10 believe is related to CLL.

11 So, that is the purpose for bringing
12 that up and -- and including that in my file.

13 Q. Obviously, in science and medicine,
14 oftentimes reasonable minds just disagree about
15 those types of issues; don't they?

16 A. Correct.

17 My own view, like I say, this is not
18 my major area of expertise. I don't think -- I
19 think that they are different, but -- but I
20 understand that other people could have a
21 different opinion who are --

22 Q. Yes?

23 A. -- who are reasonable scientists.

24 Q. Yes?

25 A. This file also contains -- because of

1 J. Whysner, M.D.

2 that, I didn't bring my whole NHL file, which I
3 have, but I do have a summary of some of the case
4 control studies of NHL and a couple of the
5 articles in general.

6 Also, let me just say that I don't
7 think that the NHL literature shows any
8 association with -- with Benzene exposure anyway,
9 so for those two reasons, the NHL literature
10 really doesn't -- well, NHL literature actually
11 is not really relevant to the -- to a CLL case
12 and certainly doesn't support any notion that CLL
13 is related to Benzene exposure.

14 And the articles -- you want the names
15 of the articles?

16 Q. Yes.

17 Let me ask you this first, though.
18 You are looking at a specific file that you
19 pulled out. Does that file have a name?

20 A. Yes.

21 Q. Do these all relate to -- you said
22 general?

23 A. CLL general refs.

24 Q. Go ahead.

25 A. There is -- there is the article by

1 *J. Whysner, M.D.*

2 Johnston that we previously talked about.

3 Q. From Wintrobe?

4 A. Yes.

5 There is the CLL article by Rai and
6 Keating or Rai and Keating. There are NHL
7 articles by Aaron Blair, 1992, "Comments on
8 Occupational and Environmental Factors and the
9 Origin of Non-Hodgkin's Lymphoma," which is from
10 Cancer Research, 1992.

11 There is an article by Gerin, and this
12 is in the American Journal Of Industrial
13 Medicine, Volume 34.

14 Q. You are looking at the non-Hodgkin's
15 lymphoma articles right now?

16 A. Yes. They are in this, okay.

17 Q. I mean, if you don't feel like they
18 are relevant to the case, then I don't feel
19 compelled to go through them with you.

20 A. Okay.

21 There is an article on the -- by -- by
22 -- well, I don't have any Post-Its in here, so I
23 can't remember why I have this in here.

24 Q. Yes?

25 A. This one has the same sort of chart

1 *J. Whysner, M.D.*

2 with incidence with age. This is Redaelli, et
3 al., and this is -- this is from European Journal
4 of Cancer Care, 2004, Volume 13, 279 to 287.

5 Q. And you are relying upon that article
6 for the age issue?

7 A. That is what you asked me about.

8 Q. I am asking you what you are relying
9 on for these articles, and you showed me a chart?

10 A. Yes.

11 This shows the world incidence of CLL
12 and showing the fact that people in Australia
13 have the highest incidence of CLL, people in
14 Denmark are sort of the same about as the United
15 States -- well, U.S.A. is the second highest
16 incidence of CLL.

17 This gives the overall incidence per
18 100,000 in the United States of five for men, and
19 it shows that -- the fact that there is five for
20 ethnicity variation between CLL.

21 Q. Can I see that report?

22 A. There is an article by Chiorazzi on
23 CLL, and this is from New England Journal of
24 Medicine, February 24, 2005.

25 There is the article by Kipps. We

1 J. Whysner, M.D.

2 already did Kipps.

3 There is this excerpt on Agent Orange
4 from the National Academy of Sciences. There is
5 16 pages from an article, "Leukemias," from
6 "Harrison's Principles Of Internal Medicine,"
7 12th Edition.

8 This is also true of some of the other
9 ones. It talks about whether there is no
10 relationship of CLL to -- to radiation exposure,
11 which is different from -- again, differentiates
12 it from AML, and from other leukemias and doesn't
13 have it as a risk factor. This is, again, the
14 Rai article, but this was from Cancer Medicine,
15 the 5th Edition, rather than the 6th Edition, but
16 it is the same Rai and Keating article.

17 Q. Does it have the same chart?

18 A. I think this is a downloaded version,
19 and I don't know if it has the figures in it as
20 part of it.

21 MR. BARNWELL: All right.

22 I do want to mark that article.

23 (Exhibit 9, 2004 article by Redaelli,
24 marked for identification, as of this date.)

25 BY MR. BARNWELL:

1 **J. Whysner, M.D.**

2 Q. I have marked Exhibit No. 9, one of
3 the studies you brought with you today. Would
4 you tell us what this is?

5 A. Redaelli from the European Journal of
6 Cancer Care, 2004.

7 Q. And you talked about that a few
8 minutes ago?

9 A. Yes.

10 **MR. BARNWELL:** Let's take a break.

11 (Luncheon Recess).

12 (Time noted: 12:17 p.m.)

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1 *J. Whysner, M.D.*

2 A F T E R N O O N S E S S I O N

3 (Time noted: 1:14 p.m.)

4 **BY MR. BARNWELL:**

5 Q. Dr. Whysner, we are back on the
6 record. Are you ready to proceed?

7 A. Yes.

8 Q. We were going through the references
9 that you brought with you today. I think you
10 have about three other folders of references?

11 A. Well, five.

12 Q. Okay.

13 A. One of them is the Health Watch, three
14 articles, and -- and then there is another one
15 that has the IARC documents in it.

16 Q. Let's get to the next folder, then.

17 A. Okay.

18 This is the References A through H.
19 The first is an article by Askoy.

20 Q. What is the significance of that
21 article?

22 A. Askoy analyzed the Turkish workers in
23 the shoe industry, and although he found a number
24 of cases of acute myelogenous leukemia, he didn't
25 have any cases of CLL in his study.

1 J. Whysner, M.D.

2 Q. All right.

3 A. And that one -- it is down here, I am
4 sorry, okay.

5 So, he had 51 cases of leukemia with
6 no cases of CLL.

7 ARP, and I believe that is 1983, and
8 the significance of this article is that this,
9 along with another article are the last two in
10 the series of articles that were done on the
11 rubber workers by University of North Carolina,
12 and this article talks about how the Benzene that
13 was used in the rubber industry before the 1950's
14 was -- came from coal rather than petroleum, and
15 it says one explanation for the observed
16 differences and the relative risks between coal
17 and petroleum derived solvents is the possibility
18 that there may have been contaminants in coal
19 based solvents that were not present in the
20 petroleum products.

21 So, they analyzed the difference and
22 relative risks for coal based versus petroleum
23 based solvents, and the difference was the odds
24 ratio was 6.67 for coal based solvents and 1.5
25 for petroleum based solvents. And the coal based

1 *J. Whysner, M.D.*

2 solvents included Benzene, which had an odds
3 ratio of 3.0.

4 So, the reason I am relying upon this
5 article is that it explains some of the elevated
6 risk of -- in the rubber workers as being to
7 contaminants in the Benzene that was used in the
8 earlier workers.

9 Q. Yes?

10 A. Which is the same thing that McMichael
11 looked at.

12 Q. Are you aware of any contaminant
13 related to coal that could be more likely than a
14 hydrocarbon to cause CLL?

15 A. There are other hydrocarbons, but they
16 mentioned, you know, polyaromatic hydrocarbons
17 and other kinds of things which -- let's see if
18 they mention it specifically. As coal is
19 aromatic in structure, while petroleum is more
20 aliphatic, it is possible that polycyclic
21 aromatic hydrocarbon PAH contaminants in the coal
22 based solvents may have been etiologically
23 important.

24 Q. Are you aware of any study that has
25 looked into that specific issue?

1 *J. Whysner, M.D.*

2 A. Three PAH components, they are long
3 names, have been shown to induce lymphocytic
4 leukemia in laboratory animals.

5 Now, that is in laboratory animals,
6 but I don't know that -- that -- I don't know
7 that anybody has concluded that polyaromatic
8 hydrocarbons are indeed a cause of CLL. This is
9 what these authors from North Carolina are
10 indicating.

11 Q. That is the theory that specific
12 article puts forth?

13 A. Yes.

14 Q. Have you seen that theory put forward
15 by any other article or any other group of
16 authors?

17 A. About the Benzene contamination issue?

18 Q. Yes.

19 A. No.

20 Q. You are not aware of any other studies
21 that would specifically support that contention
22 about polyaromatic hydrocarbons related to CLL;
23 are you?

24 A. I was thinking whether or not the
25 agent -- well, dioxin is possibly the reason for

1 *J. Whysner, M.D.*

2 an association between CLL and Agent Orange.
3 That is a polyaromatic hydrocarbon, but it is not
4 the same ones that they are talking about here.
5 These are coal derived polyaromatics, and in that
6 case, I don't know whether if they know if dioxin
7 was part of the contamination in the coal. So,
8 that is the caveat that I would put on that.

9 If we were talking about Agent Orange
10 being a cause of CLL, which I am not 100 percent
11 sure it is, one would look to -- was it the --
12 was it the 24D and the 245T that make up Agent
13 Orange or whether the dioxin contamination in the
14 Agent Orange that was -- that would be the
15 causative agent.

16 Q. Well, getting back to the theory about
17 coal contamination in the Benzene, is there any
18 way for the authors to specifically know what
19 polyaromatics contaminated the coal based
20 Benzene?

21 **MR. WERNER:** Object to the form.

22 A. They don't mention it, but I would
23 assume that this has been looked at in some of
24 the literature, but I am not aware of it. It may
25 be in the Benzene IARC monograph.

1 *J. Whysner, M.D.*

2 Q. ARP's Article makes a conclusion about
3 lymphocytic leukemias in general; doesn't it, or
4 is it specific to CLL?

5 A. If you mean by conclusion, you are
6 talking about the abstract, yes, they mentioned
7 15 cases of lymphatic leukemia.

8 Q. Have you attempted to evaluate how
9 many of those cases were CLL versus ALL?

10 A. I don't think that information is
11 available.

12 Q. What was the odds ratio concluded in
13 the ARP article related to lymphatic leukemias?

14 A. As I mentioned, for petroleum based
15 solvents, it was 1.5, and for coal based
16 solvents, it was 6.67, and of that coal based
17 solvents, Benzene was 3.0, and Xylene was 5.5.

18 Q. What was the overall ratio of Benzene
19 in lymphatic leukemias?

20 A. I believe it was 4.5.

21 But my point here is -- my point here
22 is there is a difference between the petroleum
23 based and the coal based. And also the fact that
24 these rubber worker studies seemed to indicate
25 that a lot of things may be associated with CLL

1 *J. Whysner, M.D.*

2 when we really know that they probably aren't
3 what you were talking about, the polyaromatics,
4 Xylene.

5 And another article that I will show
6 you later has to do with a whole bunch of things,
7 other things like carbon tetrachloride and carbon
8 disulfide in these rubber worker studies and
9 Benzene are associated, but now we know that they
10 aren't associated with CLL.

11 So, there are some real difficulties
12 in trying to figure out what the rubber worker
13 studies was really all about, which that was the
14 reason I didn't include them in my summary
15 tables, because to be quite honest, I really
16 don't know what the -- what the -- I don't think
17 anyone knows what the causative agent was in
18 those studies. Like I say, the indications are,
19 at least from those studies, that it was -- that
20 they are better candidates than Benzene from
21 those studies.

22 Q. What is your next article?

23 A. Okay. This is part of the
24 toxicological profile for gasoline, and -- and my
25 reason for including this is -- is that gasoline,

1 *J. Whysner, M.D.*

2 of course, is a solvent or a fuel, however you
3 want to call it, which contains an amount of
4 Benzene, which is probably about 2 to 3 percent
5 overall. There have been some that are lower,
6 some that are even higher than that, but in
7 general, 2 or 3 percent, and their conclusion is,
8 therefore, there is no conclusive evidence to
9 support or reflect the carcinogenic potential of
10 gasoline in humans or animals based upon the
11 carcinogenicity of one of the components,
12 Benzene.

13 What they are talking about is they
14 were looking at studies of people who have been
15 exposed to gasoline, and they couldn't find that
16 gasoline is a human carcinogen. That has been a
17 conclusion of the IARC as well and, I believe,
18 the EPA, also. So, the idea here is when Benzene
19 is part of a mixed solvent, that the evidence
20 does not support the carcinogenicity of that
21 solvent.

22 Q. What is next?

23 A. A study here by Bowditch and Elkins.

24 This is a -- this is an older study,
25 it is from 1939, and this along with another

1 *J. Whysner, M.D.*

2 study is the study that people have talked about
3 a statement by the API in 1948 that talked about
4 whether or not low levels of Benzene could be
5 associated with disease, and then they referenced
6 this and that other article as the basis for what
7 their statements are.

8 And the reason that I have brought
9 this is that to just indicate that the
10 concentrations of Benzene in the atmosphere, this
11 is the industrial hygiene part of the paper, were
12 150, 180, 180; this is PPM, 100, 200, 200, 200,
13 500, 500, and their statement is based upon two
14 cases out of 80 some cases where -- where what
15 they say is that the Benzene exposure was less
16 than 25 PPM or less than 10 PPM. These are just
17 air levels, these are not PPM/years.

18 And these people got disease, so they
19 are -- the basis of that API statement, actually
20 those deal with very high levels of Benzene, even
21 the people who they say are -- you know, when
22 they are making that statement about low levels
23 might not be safe, they are talking about levels
24 that would be considered way above the -- the TLV
25 that is used today or -- or the levels that would

1 *J. Whysner, M.D.*

2 be considered to be related to AML for Benzene.

3 Q. You bring up an interesting point. Do
4 you have an opinion about what threshold Benzene
5 exposure you need to have before you get into an
6 area where you can be at Benzene related illness?

7 A. Well, in terms of AML, I would say you
8 would have to have at least PPM/years. And
9 although I have seen other people who have
10 re-analyzed that, this is based upon the Rinsky
11 1987 data, and I have seen -- I have seen a
12 valuation of that where they just looked at the
13 AML cases, the leukemia cases in which they found
14 you had to have 200 PPM/years.

15 I have seen other analyses that are
16 somewhere in between, so I think that -- that 40
17 -- you have to have at least 40 from the data
18 that I have seen, and whether or not you need
19 more than 40 up to 200 is kind of a gray area.

20 Q. As it concerns Mr. Ringstaff's
21 exposure, you haven't calculated an estimate of
22 his PPM/year exposure; have you?

23 A. No.

24 This is an article by Calle, and this
25 is one of the articles that have to do with

1 *J. Whysner, M.D.*

2 obesity, and increase in leukemia. They don't
3 differentiate between various kinds of leukemias.

4 Q. What is your next one?

5 A. Chen and Eastman.

6 This is an article that actually talks
7 about topoisomerase inhibition, and this is the
8 -- one of the first studies that actually took a
9 look at this particular issue.

10 Q. What is the year on that?

11 A. 1995.

12 A study by Collingwood, and this is --
13 okay, I started out with Wong and Robb, which was
14 their big analysis of the 19 studies, so all of
15 these represent updates from studies that were
16 included in the 19 -- except there is a Health
17 Watch study here, also. But, so, this
18 Collingwood study is a competitive update study
19 where they found a relative risk of .71, okay,
20 from the Palsboro, New Jersey refinery.

21 Q. What is the update process?

22 A. They follow people for more years.
23 They may have modified somewhat their inclusion
24 or exclusion criteria. They may have, it depends
25 on the study.

1 *J. Whysner, M.D.*

2 If you want to talk specifically about
3 this one, but in general, they have refined their
4 methodology in some way. I have seen things
5 where -- where, for example, in the Health Watch
6 study, when they did an update, they changed the
7 category of some people based upon better
8 information.

9 Q. Changed the category of their work
10 history or changed the category of their illness?

11 A. Well, I think in both. In the Health
12 Watch study, they changed some of their exposure
13 metrics based upon better information. They
14 actually reclassified some cases when they had
15 better information about what the real diagnosis
16 was. That is in their method, the method's
17 section of the 2005 update. You can find it
18 there.

19 Q. Wouldn't you expect whenever you would
20 change the makeup of a cohort, to get a different
21 outcome?

22 A. Do you mean if you would get slightly
23 different numbers? Yes. Would you change the
24 overall conclusion of a study? Not usually. At
25 least I haven't seen any of these petroleum

1 *J. Whysner, M.D.*

2 studies where the numbers really went from
3 significantly increased to decreased or vice
4 versa.

5 There was one group, one -- one
6 petroleum, one refinery where the -- where the
7 numbers for CLL are elevated, but they are not
8 for the others, and I don't think that has
9 changed very much overall of the various updates
10 that have taken place.

11 Q. The next study?

12 A. A study by Decoufle. This is a
13 Benzene study, a chemical plant where Benzene has
14 been used in large -- in large quantities, and
15 there were four cases of leukemia and only one
16 case of CLL that was found.

17 There is a study by Delzell and
18 Monson, 1981. These are rubber workers, and in
19 this study, they did not find an increase in
20 leukemias in their rubber workers.

21 Devine, 1999. Again, this is another
22 update, Texaco update where the SMR was .8.

23 This is a document by the Food and
24 Drug Administration that I got from their
25 website, and it talks -- it is a report, Guidance

1 *J. Whysner, M.D.*

2 for Industry: Recommended Approaches to
3 Integration of Genetic Toxicology Study Results."
4 What they are talking about is how to deal with
5 certain genotoxicity for the Food and Drug
6 Administration.

7 And -- and it is -- it basically says
8 that agents that induce effects by indirect
9 mechanisms, such as -- and they talk about a
10 couple of them, and they say or inhibition of
11 topoisomerase may have thresholds for genotoxic
12 effects. So, this speaks to the issue of if
13 something produces its effects by inhibition of
14 topoisomerase, the FDA is saying that it would be
15 expected to have some threshold dose below which
16 that would not take place.

17 A study by Forni, 1971. This is a
18 chromosome study in workers exposed to Benzene,
19 or Toluene or both. This is one of a number of
20 studies I have in my files which -- which talks
21 to the fact if you have Benzene exposure, but you
22 have exposure to either Toluene or Xylene as
23 well, you don't see as great an effect as you do
24 with the Benzene itself. And the explanation for
25 this is that these other hydrocarbons inhibit the

1 *J. Whysner, M.D.*

2 metabolism of Benzene to its active metabolites,
3 so they interfere with that metabolism.

4 So, in a solvent mixture that has
5 Benzene as a component, the metabolism -- if one
6 were exposed to that solvent mixture, not only
7 might not one get any effect from the Benzene,
8 because -- because its mechanism is
9 topoisomerase, which has a threshold, but also
10 because there are other things in the mixture
11 which are interfering with its metabolism. So,
12 these are explanations of why one would not see a
13 low dose effect with Benzene containing solvents.

14 This is another one by an author named
15 G-A-D or Gad, I guess, Karim. This is from 1984.
16 This is, again, showing this kind of inhibitory
17 effect with Toluene.

18 Q. Okay.

19 A. This is a review that Dr. Gardener
20 participated in in 1987 from Texas Medicine, and
21 -- and in it, it says radiation, drugs, chemical
22 exposure and viruses have not been implicated as
23 risk factors in the etiology or pathogenesis of
24 CLL.

25 When I read his deposition, he said we

1 *J. Whysner, M.D.*

2 really weren't talking about Benzene because I
3 couldn't quite figure out why it was, but because
4 it was in the same target population that they
5 were addressing in this article.

6 But -- but they do talk about "Chronic
7 Benzene exposure as being a -- the etiology is
8 not known, it is almost certainly
9 multi-factorial. It is a risk factor which
10 includes prior exposure to ionizing radiation,
11 immunodeficiency, chronic Benzene exposure and
12 prior treatment with cancer chemotherapeutic
13 drugs, particularly alkylating agents." And this
14 is under -- under "Epidemiology: Etiology of
15 Acute Non-Lymphocytic Leukemia."

16 He is making a distinction between
17 what is AML, sort of the same as AML, Benzene
18 being a risk factor, but there being no risk
19 factors like that known for CLL.

20 Q. What is next?

21 A. Okay.

22 Deborah Glass, her article from 2003.
23 This is one article that is an analysis of
24 leukemia subtypes. The highest exposure group
25 has an increased -- I assume that these are

1 *J. Whysner, M.D.*

2 relative risks. It doesn't say in the table, but
3 the highest exposure has a relative risk that is
4 elevated, but not statistically significant.

5 And this is also part of the same data
6 that was in the Health Watch 2001.

7 Q. Is it your position when looking at
8 the epidemiology studies, that there is a
9 specific relative risk that has to be achieved
10 before that study can be said to go towards the
11 idea of causation?

12 A. Statistically increased risk.

13 Q. Let's set that aside.

14 First, let's talk about the relative
15 risk. Is there a number in your mind that you
16 have to see of a relative risk before you could
17 say that an epidemiology study can be any
18 evidence of a cause and effect?

19 MR. WERNER: Object to the form.

20 A. Well, usually I think we like to see
21 ones that are above two.

22 Q. Is there an epidemiology textbook or
23 anything like that that creates a concept of a
24 2.0 cutoff?

25 A. Most of it was actually -- excuse me,

1 *J. Whysner, M.D.*

2 I was at the American Health Foundation, and our
3 founding president was Ernst Wynder, who did the
4 original studies on cigarette smoking and lung
5 cancer in the United States. He was an
6 epidemiologist. It wasn't so much as reading it
7 in a text, but Dr. Wynder said one needed to see
8 something in or about that range, two to three at
9 least, in order to take a study seriously.

10 Q. What about a dose response
11 relationship, is that something that you are
12 looking for when you review the scientific
13 evidence?

14 A. Yes.

15 Q. If there is a dose response, even if
16 it doesn't get above two, do you consider that
17 relevant evidence?

18 A. Not if it doesn't get above two.

19 Q. What about a relative risk that has a
20 confidence interval that includes one of the
21 relative risks above two, do you consider that
22 evidence of causation?

23 A. No.

24 Q. Not under any circumstances?

25 A. No.

1 *J. Whysner, M.D.*

2 Q. Doctor, in the Glass article that you
3 were just citing me, you did see what the summary
4 conclusion was by the authors?

5 A. (No Response).

6 Q. And on Page 576, it says, "These data
7 demonstrate a strong association between Benzene
8 exposure and the risk of acute and chronic
9 leukemia."

10 Do you disagree with that conclusion?

11 A. Excuse me, where is that, again?

12 Q. 576.

13 A. Believe it or not, my copy does not
14 have a 576.

15 I see, there are pages that are
16 backwards here. Sorry. Yes, I see that here.

17 Q. And you just --

18 A. It says acute and chronic leukemia. I
19 don't see the -- the -- the lymphocytic -- did
20 you say lymphocytic --

21 Q. No, I didn't.

22 You just disagree with Glass'
23 conclusion there in terms of overall leukemia?

24 A. She is talking about overall leukemia,
25 acute and chronic, because there isn't anything

1 *J. Whysner, M.D.*

2 else. I agree with that in terms of all
3 leukemia.

4 Q. Okay.

5 What is your next study?

6 A. All right.

7 The next study is another study by
8 Glass, which is chemico-biological interactions,
9 2005.

10 MR. WERNER: May I see that last one?

11 BY MR. BARNWELL:

12 Q. All right.

13 A. This is a paper where she discusses
14 some of the parameters by which they did their
15 exposure assessments.

16 Q. In the Australian study?

17 A. Yes.

18 Q. That is fine.

19 A. But the other important thing, I
20 think, is, and she says this essentially in that
21 other paper as well, she says in this paper
22 something in our study, the odd's ratios for
23 leukemia were significantly raised, unlike those
24 reported from similar petroleum industry studies,
25 and she -- she -- she references studies by

1 *J. Whysner, M.D.*

2 Schnatter, which is Reference 14 in the paper,
3 and by Rushton, which is Reference No. 15.

4 This talks about at the exposure
5 levels that they were investigating, which are
6 quite a bit lower than the Pliofilm study, she is
7 pointing out that her paper is at odds with the
8 other papers reported in the literature.

9 Q. You do recognize that there are
10 epidemiological studies out there that have shown
11 excess CLLs in refinery workers at one time or
12 another?

13 A. There is one that I know of, and that
14 is the -- at least a cohort study, the Huebner
15 study, which is an update of Lewis. That is the
16 Baton Rouge study, which is 1 out of 19
17 refineries, one of the others found an increase.

18 Q. Are you aware of any others where
19 there has been an excess of CLL identified?

20 A. There is a discrepancy in this Health
21 Watch study. As I said, it is not really -- at
22 least by Glass, it really wasn't reported to be
23 statistically significant, but she found
24 increased risk levels in the -- in the case
25 control study, but they didn't find it in the

1 *J. Whysner, M.D.*

2 cohort study update, which they had found in the
3 previous cohort study in 2000, they didn't find
4 it in 2005.

5 So, it seems as though something --
6 something may have changed in the interim in
7 terms of either their number of cases of CLL or
8 something about the -- about the comparison
9 groups. It -- one of the things that is very
10 clear in the case control study, there are what
11 is called controls. The lower exposure group
12 have about one-half of the CLL that one would
13 have expected from that group. So, she is -- she
14 is comparing a group of people who have one-half
15 of the expected rate with -- with the other
16 exposure groups.

17 Q. All right.

18 What is next?

19 A. All right.

20 This is another general article on NHL
21 by Greiner, and this is in Cancer Supplement. It
22 is in Cancer -- that is the name of the journal,
23 but it is a supplement, Volume 75, and this is in
24 1995.

25 And, again, this is where they talk

1 *J. Whysner, M.D.*

2 about low grade lymphomas constitute almost 10
3 percent of the NHLs. Some people say 6. I have
4 seen anywhere between 6 and 10 percent for these
5 small lymphocytic leukemias, the small cell type.

6 Q. What is the next study?

7 A. Guenel, 2002.

8 Q. Is that the Swedish study?

9 A. No, this is French.

10 It is a case control study, and they
11 did not find a statistically significant trend
12 for chronic lymphocytic leukemia among Benzene
13 exposed people.

14 Q. What trait are they looking at in that
15 study?

16 A. I am sorry, nested cohort of gas and
17 electric utility workers.

18 Q. There was some elevated leukemia in
19 that study; wasn't there?

20 A. There wasn't in the group where they
21 had three cases, which was 0 to 5.5. There was
22 one case in the group above 5.5. They calculated
23 a -- an odd's ratio, but the fact that it is
24 based on one case makes the confidence limits
25 huge, so it doesn't really mean much.

1 *J. Whysner, M.D.*

2 A study by Hayes, which is one of the
3 Chinese studies, and -- and they didn't look
4 specifically at CLL in this case, they found an
5 increase for AML.

6 Q. Again, as you pointed out, the Asian
7 population has a fairly low rate of CLL?

8 A. Correct.

9 Q. Do you know what the incidence per
10 100,000 is for CLL in the Asian population?

11 A. It is in one of those papers we were
12 just looking at. Maybe you have it there.

13 Q. Yes?

14 A. It's male and females combined is
15 probably about .3 per 100,000, something like
16 that.

17 Q. All right.

18 A. Bradford Hill.

19 Q. That is fine.

20 A. Hotz and Lauwerys, "Critical Reviews
21 in Toxicology," 1997.

22 This is vehicle mechanics, and -- and
23 they looked at a lot of different groups, and --
24 and I think they only found CLL in aircraft
25 mechanics.

1 *J. Whysner, M.D.*

2 Q. Did they calculate an odd's ratio?

3 A. They found for leukemia, it was 3.34.

4 Let me see who is that. I think this
5 is a review of another study, Linos, we will get
6 to that, where they talk about four cases, three
7 of them being CLL. That is part of the
8 literature review. I think the bottom line from
9 this study is they looked at a lot of different
10 groups with various different kind of vehicle
11 mechanics and didn't find any relationship to CLL
12 exposure.

13 Q. What is next?

14 A. And Huebner. This is the -- this is
15 the incident study.

16 Q. That is actually a follow-up to the
17 Lewis study; isn't it?

18 A. No. This is an incident study. The
19 Huebner follow-up to the Lewis study is a worker
20 mortality study.

21 Q. I am sorry?

22 A. And in this study, the -- CLL is --
23 they have five cases of CLL where 4.1 would be
24 expected. They had an SIR of 1.22.

25 Q. Was that statistically significant?

1 *J. Whysner, M.D.*

2 A. No.

3 Q. Yes?

4 A. The confidence interval was from .4 to
5 2.85.

6 This is the -- this is Huebner, and
7 this is a 1997 study of Exxon workers, and in
8 this one, they found a 1.6 relative risk with a
9 confidence interval of .85 to 2.83. So, it is
10 not statistically significant.

11 2004, the update of Lewis by Huebner,
12 also where they found that 2.42 for Baton Rouge
13 and -- and found a 1.23 for Baytown, Texas.

14 This is an article by Hunter. This is
15 the other article that I was talking about with
16 that reference to the API 1948 document. This
17 actually goes through an analysis of all of the
18 cases.

19 Q. Okay.

20 A. Hunting, another vehicle mechanic
21 study. They had only -- this was a study
22 beginning with a cluster that they were
23 investigating, but they only found two CLL cases.
24 So, that is the first file.

25 Q. I just want to mark that whole group

1 J. Whysner, M.D.

2 as just one big exhibit.

3 A. Right. I don't really know that the
4 order matters. It is alphabetical.

5 Q. Then don't let me mess up your system.

6 A. We may have to get back to it.

7 Q. I am marking the group of studies that
8 you just discussed as Exhibit 10.

9 A. That is Benzene Refs A through H.

10 (Exhibit 10, Benzene Refs A through H,
11 marked for identification, as of this date.)

12 A. I through P.

13 Q. Go for it.

14 A. Infante, 1977. They found acute
15 myelogenous leukemia, chronic myelogenous,
16 monolymphocytic, but they didn't find any CLL
17 cases in the Pliofilm Cohort.

18 The next study is by Lamb and Bird.
19 This is review of the non-NHL literature where
20 they didn't find any increase related to Benzene
21 exposure.

22 Q. Again, you don't see a correlation
23 between CLL and non-Hodgkin's?

24 A. My reason for stating it is not really
25 relevant to the case. First of all, there is no

1 *J. Whysner, M.D.*

2 relationship except possibly a small crossover,
3 and that the -- that the NHL literature doesn't
4 support an association with Benzene anyway, so --

5 Q. Go ahead.

6 A. This is a Canadian update, Lewis.
7 This is different from the Lewis.

8 Q. Exxon?

9 A. Yes. This was .67 with a -- with a
10 range of -- or a confidence interval of .3 to
11 1.27.

12 This is an article by Linet, 1980,
13 which is a case control study, and -- and they
14 found four patients with leukemia and three in
15 control subjects with exposure to Benzene. They
16 did not find any association between Benzene
17 exposure and CLL.

18 They also showed an interesting
19 difference in case control studies between
20 whether or not just the people who had the
21 disease were answering the questionnaire or
22 whether it was family members included, so it
23 seems as though family members always tend to
24 overestimate exposures such as Benzene. And so
25 they were -- they didn't find anything with

1 *J. Whysner, M.D.*

2 Benzene, but they were also founded to be even
3 less when you just restricted your questions to
4 the people who actually had the disease, which
5 means that asking family members or friends can
6 be unreliable in case control studies.

7 Q. What is next?

8 A. This is Linet and Cartwright.

9 This is a chapter in "Cancer
10 Epidemiology and Prevention," 1996. This is a
11 literature review. They discuss this issue of
12 cigarette smoking and CLL, talking about the
13 studies that have been positive, and -- and they
14 also discuss Benzene exposures, and -- and their
15 conclusion is that there is sufficient evidence
16 that Benzene causes AML, but not the -- they
17 didn't make that conclusion of CLL.

18 Q. Does that author make the conclusion
19 about smoking and CLL?

20 A. I don't think so. They talk about
21 both sides of the --

22 Q. Okay.

23 A. -- issue.

24 Q. What is next?

25 A. Lunberg.

1 *J. Whysner, M.D.*

2 This has to do with paint industry
3 workers, and they found two cases of CLL overall
4 in their group. One -- about one would have been
5 expected, but it is not statistically
6 significant.

7 This is the article by Lynch, which is
8 a laboratory study that shows a threshold for
9 topoisomerase inhibition.

10 Lynch is a study of exposure to
11 gasoline vapors and service station workers, and
12 -- and they reported that -- that the SIR -- that
13 is an -- it is an incident study for CLL, was .8,
14 with a confidence range of .3 to 1.6.

15 Q. All right.

16 A. Malone is a study of chronic -- or CLL
17 in relation to chemical exposures, and they found
18 a relative risk of 1.1 with a confidence interval
19 of .6 to 2.0.

20 McMichael, 1974 -- I mean McMichael,
21 1975.

22 This one was in the tire industry, and
23 they found -- I don't know if they did a
24 statistical analysis, but they found 16 cases of
25 lymphocytic leukemia and 15 cases of myeloid

1 *J. Whysner, M.D.*

2 leukemia in their work.

3 Q. Did they calculate any relative risk?

4 A. Not really. Tire building, eight over
5 3.2. They didn't actually calculate it, but they
6 don't really specifically link it to Benzene
7 exposure, either.

8 Q. Did they identify any of the potential
9 carcinogens that could have caused CLL there or
10 could have caused the lymphocytic or myeloid
11 leukemias?

12 A. I don't see it.

13 **MR. BARNWELL:** I am going to mark this
14 folder as Exhibit 11.

15 (Exhibit 11, Benzene Revs I through P,
16 for identification, as of this date.)

17 A. R through Z.

18 Robb, 1998. This is Beaumont,
19 Texas..25 is the refinery study. .25 with
20 confidence range of .01 to 1.39.

21 Q. What is next?

22 A. Robb and Wong.

23 Q. What is next?

24 A. This is 1996, Rai and Keating. We
25 have already been through this.

1 *J. Whysner, M.D.*

2 Rinsky, 1987. Again, they don't show
3 any cases of CLL in their cohort.

4 Rinsky, 2000. I am not sure that
5 study -- Rinsky, 2002. And, again, they don't --
6 they don't find any cases of CLL in there. They
7 have increased the numbers, but no CLL cases.

8 Q. From a scientific standpoint, isn't it
9 possible that a chemical could cause a certain
10 group of diseases and not have every disease show
11 up in every cohort?

12 A. That is why -- do you mean on a random
13 basis?

14 Q. On any given cohort study.

15 **MR. WERNER:** Object to the form.

16 A. I don't know if that is an expert
17 opinion question. It is a question like can
18 anything happen. I guess I don't have an opinion
19 about that if you are asking me --

20 Q. Aren't there --

21 A. -- in that sense.

22 Q. I will ask you this: There is no real
23 dispute in the scientific community that Benzene
24 causes AML at some level?

25 **MR. WERNER:** Object to the form.

1 *J. Whysner, M.D.*

2 A. It can.

3 Q. Can cause AML at some level?

4 A. May cause, yes. Not necessarily, but
5 it -- in some individuals, it may cause.

6 Q. But there are epidemiological studies,
7 cohort studies, that show Benzene exposed
8 populations that don't show an excess of AML;
9 aren't there?

10 A. Correct.

11 Q. At doses where you realistically could
12 expect to find excesses of AML?

13 A. I don't know of any. Because the
14 Turkish studies, the Chinese studies, the Rinsky
15 studies, at those levels that one would expect, I
16 think they are all pretty consistent.

17 Q. All right.

18 What is next?

19 A. There is just the earlier Rinsky 1981
20 reports.

21 Study by Roh, 1987. It is another one
22 shows interference of Toluene with Benzene
23 effects.

24 Case control studies by Rushton. This
25 was the one that was quoted by Dr. Glass as being

1 *J. Whysner, M.D.*

2 not consistent with her study.

3 Q. What year is that study?

4 A. 1997.

5 Q. Are you aware of any earlier Rushton
6 studies that do show excess CLL?

7 A. It says, "There is no evidence in this
8 study of an association between exposure to
9 Benzene and lymphoid leukemia, either acute or
10 chronic."

11 Satin, 2002. These are two -- these
12 are two petroleum refineries cohort studies with
13 relative risings of .97 and .86.

14 Q. Okay.

15 A. Satin, 1996. And this is the Port
16 Arthur that shows -- this is the one that
17 actually showed a statistically significant
18 decrease of .3 with confidence intervals of .06
19 to .87.

20 Schnatter, 1996. This is the other
21 study that Dr. Glass quoted as being inconsistent
22 with hers.

23 This is another Schnatter. This is a
24 review of the literature on Benzene exposure and
25 leukemia subtypes --

1 J. Whysner, M.D.

2 Q. All right.

3 A. -- where he -- where he basically
4 says that it is -- CLL is -- is -- he says a few
5 of the case control studies show elevations
6 related to Benzene, but cohort studies did not
7 show such risks.

8 Sorahan and Dole, 2005.

9 S-O-R-A-H-A-N. I realize I misspelled it on my
10 sheet here.

11 CLL is 1.24 with confidence intervals
12 of .02 to .89, Benzene exposed workers in the U.K.

13 Tice, I cannot give you a date right
14 now. This is another one with the interference
15 with Benzene effects.

16 Q. Okay?

17 A. Tunick, 1982, ditto.

18 Q. The low level Benzene interference
19 with the effects, that kind of stuff?

20 A. Well, it is one milligram Benzene per
21 kilogram -- I don't know that I would call that
22 low, but --

23 Q. When you said ditto, I thought you
24 were referencing the previous comment that you
25 made about --

1 *J. Whysner, M.D.*

2 A. Benzene interferes with the toxicity.
3 It says effects of Benzene and Benzene
4 metabolites on bone marrow solidarity (sic)
5 number of granulopoietic stem cells and frequency
6 of micronuclei in mice.

7 This is by Revol and Girard, it is in
8 French, from 1969. They found two lymphatic
9 leukemias, no statistical analysis.

10 Here is another article on obesity
11 that found among white veterans, statistically
12 elevated for several cancer, including CLL and
13 AML.

14 Wisner, "Genotoxicity of Benzene,"
15 2000.

16 Q. Okay.

17 A. Wisner, "Genotoxicity of Benzene and
18 its Metabolites," 2004.

19 Wilkowsky, which is 1984, and
20 discussed before. This shows increased odd's
21 ratio for a bunch of other things, including
22 leukemia. We don't know what the cause of rubber
23 worker is of the increased relative risk.

24 Wong and Robb, "The Meta-Analysis."
25 Wong, this is the update of Morgan, and also

1 *J. Whysner, M.D.*

2 Robb. This is 2001, Beaumont, Texas update. So
3 there are two of them, and this shows .18 going
4 from .01 to 1.03.

5 A general article by Wong and Robb
6 about NHL among petroleum workers. This is a
7 study by Wong, 1986. This is actually the
8 published version of that paper that Dr. Gardener
9 -- that is in my file had with his recent
10 deposition.

11 Q. Okay.

12 A. Chinese papers, three of them, Yin,
13 Yin, and Yin, and they go through all of their
14 patients. As far as I can tell, they have one
15 case of CLL among the whole group, which totalled
16 42 cases. Even though they have a low incidence
17 of spontaneous CLL, I don't think this study
18 shows certainly no increased levels of CLL in
19 these exposed workers.

20 **MR. BARNWELL:** All right.

21 Let me mark the folder that you just
22 finished talking about Exhibit No. 12.

23 (Exhibit 12, Benzene Refs R through Z,
24 marked for identification, as of this date.)

25 **BY MR. BARNWELL:**

1 *J. Whysner, M.D.*

2 Q. You have a couple of more folders of
3 reference material, and we are going to mark one
4 of those, and could you tell us what that is,
5 Exhibit 13?

6 (Exhibit 13, various references from
7 the IARC, marked for identification, as of this
8 date.)

9 A. It is various references from the
10 IARC.

11 Q. One folder that you brought that we
12 are not going to mark is the Health Watch
13 studies.

14 A. Correct.

15 Q. Could you tell us which ones you
16 bought with you today?

17 A. 12th report, 2005, 11th report, 2000,
18 and then a -- a report from -- a technical report
19 on lymphohaematopoietic cancer and exposure to
20 Benzene in the Australian petroleum industry,
21 dated June 2001.

22 Q. Do you understand there to be any
23 difference -- strike that.

24 Are you aware of there being any
25 difference in the manner that the data is

1 *J. Whysner, M.D.*

2 analyzed in the 2001 study versus 2000, 2005?

3 A. Yes.

4 Q. What would that difference be?

5 A. 2001 is a nested case control study of
6 the cohort study from -- from the others.

7 Q. The 2000 study is not an update from
8 the nested control study from 2001; is it?

9 A. That is correct.

10 Q. How many cases have you worked on
11 where you were giving an opinion concerning a
12 plaintiff who had actually worked in a refinery.

13 A. Could I see that list?

14 None.

15 Q. I am not limiting it to the four years
16 represented on the list, I am asking about at any
17 time in your career.

18 A. Not that I recall. I mean, giving a
19 deposition or testified at trial.

20 Q. I will break this down, then.

21 Have you ever given testimony either
22 at deposition or at trial in a case involving a
23 plaintiff who worked in a refinery?

24 A. No.

25 Q. Have you ever been retained as an

1 *J. Whysner, M.D.*

2 expert in a case where a plaintiff has worked at
3 a refinery?

4 A. Yes.

5 Q. Have you had an opportunity to
6 evaluate the type of work people do in
7 refineries?

8 A. Yes.

9 Q. Have you become familiar with the
10 turnaround process in refineries?

11 A. To a limited extent.

12 Q. You recognize in that process, units
13 are being shut down, equipment is being opened
14 up, workers are going into vessels and things
15 such as that? Do you understand that to be the
16 case?

17 A. I have seen that in deposition
18 testimony.

19 Q. You recognize turnaround work in
20 refineries where contractors were performing work
21 in vessels, and pulling exchangers and opening
22 equipment could have a potential for Benzene
23 exposure?

24 A. Potential, yes.

25 Q. I saw on your CV, Doctor, that for

1 *J. Whysner, M.D.*

2 about 13 years, you worked at the American Health
3 Foundation?

4 A. Yes.

5 Q. What is the American Health
6 Foundation?

7 A. It's a research institute. Most of
8 its money came from the National Cancer Institute
9 studying cancer prevention. So, all of the
10 research -- almost all of the research related to
11 how to find what the causes of cancer were and
12 how to prevent them.

13 Q. Other than the National Cancer
14 Institute, what were some of the other sources of
15 funding for the Foundation you were a member of?

16 A. The American Cancer Society was one.
17 And we were also funded by -- at least the things
18 I knew about were funded by food companies,
19 chemical companies, pharmaceutical companies.

20 Q. Do you have a percentage of funding --
21 are you aware of the breakdown in percentage
22 terms of the funding that the American Health
23 Foundation received from the private companies
24 versus the American Cancer Society and the
25 National Cancer Institute?

1 *J. Whysner, M.D.*

2 A. I know that the National Cancer
3 Institute was about 80 percent.

4 Q. Do you know any of the experts that
5 have been retained on the Plaintiffs' behalf in
6 this case?

7 A. Know in what sense?

8 Q. Do you have professional dealings with
9 any of them?

10 A. No. Other than -- other than in a
11 couple of other cases, they are also expert
12 witnesses. Other than that, no.

13 Q. Do you have any opinion on whether or
14 not Dr. Infante, Dr. Gardener, Dr. Kirk or Fred
15 Parker (sic), Plaintiffs' experts, are in some
16 way unqualified to render the opinions in this
17 case?

18 A. I don't have an opinion on that.

19 Q. You recognize Dr. Infante's name from
20 Benzene literature?

21 A. Yes.

22 Q. Do you recognize Dr. Infante as a long
23 standing researcher into the long term health
24 effects of Benzene?

25 A. I don't have an opinion on that.

1 *J. Whysner, M.D.*

2 Q. Do you have an opinion on what the
3 odor threshold of Benzene is?

4 A. No.

5 Q. Based upon your review of Mr.
6 Ringstaff's medical records, what complications
7 has he experienced as a result of his CLL
8 diagnosis?

9 A. I think they are listed in the -- in
10 my -- can I refer to that?

11 Q. Sure.

12 A. It says "Additional medical problems,"
13 and I believe that most of these are subsequent
14 to his treatment, depression, hypogammaglobulin
15 anemia, infections, gout, hypothyroidism,
16 asthenia and possibly sleep apnea.

17 Q. Which of those would you say are
18 related to either his treatment or illness?

19 A. I don't have an opinion on that.

20 Q. Have you ever been hired by a refinery
21 or chemical company to come in and help them
22 evaluate potential steps to take to protect their
23 workers?

24 A. Yes.

25 Q. What chemical companies or refineries

1 *J. Whysner, M.D.*

2 were those? What can you tell me about that?

3 A. The only one that I -- well, the one
4 that I -- that I remember off the top of my head
5 is a vinyl chloride plant where I was asked to
6 help design a medical surveillance program to
7 look at potential liver effects, and for
8 scheduling when to shift workers out of the
9 facility and then back into the facility if they
10 showed any effect based upon chemical tests and
11 which chemical tests to use.

12 Q. You agree that the worker has the
13 right to know the hazards that they are working
14 in; right?

15 A. Well, I know -- I agree with the
16 worker right to know law.

17 Q. Even before it was a law, it was an
18 idea that you would support?

19 A. I don't know if I have an opinion on
20 that.

21 (Continued on next page to allow for
22 signature line and jurat.)

23

24

25

1 J. Whysner, M.D.

2 MR. BARNWELL: Those are all the
3 questions I have. I will pass the witness.

4 MR. WERNER: I have an hour and a
5 half.

6 Off the record.

7 (Time noted: 2:47 p.m.)

8

9

10 _____
JOHN WHYSNER, M.D.

11

12 Subscribed and sworn to before me

13 this ____ day of _____, 2006.

14 _____

15 Notary Public

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I, ANITA SHEMIN, a Shorthand
(Stenotype) Reporter and Notary Public of the
State of New York, do hereby certify that the
foregoing examination of the witness, JOHN
WHYSNER, M.D., taken at the time and place
aforesaid, is a true and correct transcription of
my shorthand notes.

I further certify that I am neither
counsel for nor related to any party to said
action, nor in any wise interested in the result
or outcome thereof.

IN WITNESS WHEREOF, I have hereunto
set my hand this 15th day of March, 2006.

ANITA SHEMIN

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March 13, 2006

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JOHN WHYSNER, M.D.	Mr. Barnwell	3
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----- INFORMATION REQUESTS -----

DIRECTIONS (DI):	None.
INSERT:	None.
RULINGS (RL):	None.
REQUESTS (RQ):	None.
CERTIFIED (CE):	None.
MOTIONS (MO):	None.

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