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UNITED STATES DISTRICT COURT
FOR THE DISTRICT OF MASSACHUSETTS

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Brian K. Milward and *

Linda J. Milward *

*

v. * Civil Action

* No. 1:07-cv-11944-GAO

Acuity Specialty Products *

Group, Inc., et al. *

*

* * * * *

Deposition of David H. Garabrant, M.D., M.P.H.

Wednesday, February 18, 2009

Nixon Peabody LLP

100 Summer Street

Boston, Massachusetts 02110

----- J. Edward Varallo, RMR, CRR -----

Registered Professional Reporter

HG LITIGATION SERVICES ~ DALLAS, TEXAS

1-888-656-3376

1 Present for Plaintiffs:

Steve Baughman Jensen, Esq.

2 Allen Stewart, P.C.

Republic Center

3 325 North St. Paul Street - Suite 2750

Dallas, Texas 75201

4 214.965.8700 ~ Fax 214.965.8701

sjensen@allenstewart.com

5
6 Present for Radiator Specialty Company:

7 Brian R. Birke, Esq.

Adler Pollock & Sheehan, P.C.

8 175 Federal Street - 10th Floor

Boston, Massachusetts 02110

9 617.482.0600 ~ Fax 617.482.0604

bbirke@apslaw.com

10
11 Present for The Clorox Company:

12 Charles K. Mone, Esq.

Campbell Campbell Edwards & Conroy, P.C.

13 One Constitution Center - 3rd Floor

Boston, Massachusetts 02129

14 617.241.3000 ~ Fax 617.241.5115

cmone@campbell-trial-lawyers.com

15
16 Present via telephone for Berryman Products, Inc.:

17 Brian P. Voke, Esq.

Campbell Campbell Edwards & Conroy, P.C.

18 One Constitution Center - 3rd Floor

Boston, Massachusetts 02129

19 617.241.3000 ~ Fax 617.241.5115

bvoke@campbell-trial-lawyers.com

20
21 Present for The Sherwin-Williams Company; Henkel
Corporation:

22 Kevin R. Powers, Esq.

Cetrulo & Capone LLP

23 World Trade Center East

Two Seaport Lane - 10th Floor

24 Boston, Massachusetts 02210

617.217.5500 ~ Fax 617.217.5200

25 kpowers@cetcap.com

1 Present for WD-40 Corporation:

Christopher G. Betke, Esq.

2 Coughlin Betke, LLP

175 Federal Street - 9th Floor

3 Boston, Massachusetts 02110

617.988.8050 ~ Fax 617.988.8005

4 cbetke@coughlinbetke.com

5
6 Present for The Sherwin-Williams Company:

James R. Miller, Esq.

7 Dickie McCamey & Chilcote, P.C.

Two PPG Place - Suite 400

8 Pittsburgh, Pennsylvania 15222-5402

412.281.7272 ~ Fax 412.392.5367

9 jmillers@dmclaw.com

10
11 Present for United States Steel Corporation;
Aristech Chemical Corporation; USX Corporation:

12 Tim Gray, Esq.

Forman Perry Watkins Krutz & Tardy, LLP

13 City Centre Building - Suite 100

200 South Lamar Street

14 Jackson, Mississippi 39201

601.960.8600 ~ Fax 601.960.8613

15 tgray@fpwk.com

16
17 Present via telephone for Sunnyside Corporation:

Leslie A. Sheehan, Esq.

18 Gordon & Rees LLP

633 West Fifth Street - Suite 4900

19 Los Angeles, California 90071

213.576.5000 ~ Fax 213.680.4470

20 lsheehan@gordonrees.com

21
22 Present for Rust-Oleum Corporation:

Matthew P. Zayotti, Esq.

23 Keegan Werlin LLP

265 Franklin Street - 6th Floor

24 Boston, Massachusetts 02110

617.951.1400 ~ Fax 617.951.1354

25 mzayotti@keeganwerlin.com

1 Present for Nu-Calgon Wholesaler, Inc.
Eileen P. Kavanagh, Esq.
2 Litchfield Cavo, LLP
6 Kimball Lane - Suite 200
3 Lynnfield, Massachusetts 01940-2682
781.309.1500 ~ Fax 781.246.0167
4 kavanagh@litchfieldcavo.com
5

6 Present for Boyle-Midway, Inc.
William R. Covino, Esq.
7 McDonough Hacking & Lavoie, LLC
One Washington Mall - 9th Floor
8 Boston, Massachusetts 02108
617.367.0808 ~ Fax 617.367.8307
9 wcovino@mhlattys.com
10

11 Present for Acuity Specialty Products Group, Inc.;
The Steco Corporation:
12 Curtis L.S. Carpenter, Esq.
Morrison Mahoney LLP
13 250 Summer Street
Boston, Massachusetts 02210
14 617.439.7500 ~ Fax 617.439.7590
ccarpenter@morrisonmahoney.com
15
16

Present for CRC Industries, Inc.:
17 Joseph J. Leghorn, Esq.
Nixon Peabody LLP
18 100 Summer Street - 25th Floor
Boston, Massachusetts 02110
19 617.345.1000 ~ Fax 617.345.1300
jleghorn@nixonpeabody.com
20
21

Present for The Steco Corporation:
22 Christopher Sullivan, Esq.
Sullivan & Collins, LLC
23 500 W. Cummings Park - Suite 4000
Woburn, Massachusetts 01801
24 781.939.5840
25

1 Present via telephone for American Grease Stick
Company:

2 Syd A. Saloman, Esq.
Tucker Heifetz & Saltzman, LLP
3 100 Franklin Street
Boston, Massachusetts 02110
4 617.557.9696 ~ Fax 617.227.9191
saloman@ths-law.com

5
6 Present for NCH Corporation (Chemsearch Division):

7 Wesley A. Weathers, Esq.
Weathers Riley & Sheppard, LLP
8 4848 S.W. 21st Street - Suite 202
Topeka, Kansas 66604
9 785.273.2020 ~ Fax 785.273.4662
wes@wrslaw.net

10
11 Also Present:

12 Vic Houston Henry, Esq.
Henry Oddo Austin & Fletcher, P.C.
13 1700 Pacific Avenue - Suite 2700
Dallas, Texas 75201
14 214.658.1900 ~ Fax 214.658.1919
vhhenry@hoaf.com

15
16
17
18
19
20
21
22
23
24
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GARABRANT EXHIBITS	FOR IDENTIFICATION

1 Document headed Legal Testimony by David H. Garabrant, MD, MPH since November 2004 (2 pages)	7
2 Dr. Garabrant's report, dated November 19, 2008 (7 pages)	19
3 Dr. Martyn Smith's supplemental declaration (SMITH000694 - 715)	21
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(ORIGINAL EXHIBITS DELIVERED TO ATTORNEY JENSEN)

1 -----
2 MORNING SESSION

3 9:17 a.m.
4 -----

5 DAVID H. GARABRANT, M.D., M.P.H.,
6 having been first duly sworn on oath,
7 was examined and testified as follows:

8 EXAMINATION

9 BY MR. JENSEN:

10 Q. Good morning, Dr. Garabrant.

11 A. Good morning.

12 Q. We were introduced earlier this morning,
13 but I'm Steve Jensen and I'm here on behalf of the
14 plaintiffs today to ask you some questions.

15 And I apologize to everyone. I have not
16 brought extra copies of my materials this morning,
17 but I don't believe I'm going to mark anything that
18 isn't part of reliance materials or reports, so
19 everyone should have copies available to them.

20 Marking as Exhibit 1 two pages that I
21 believe is a list of your legal testimony since
22 November 2004. Take a look at that and see if
23 that's correct, Doctor.

24 A. I believe it is.

25 Q. And do you have your own copy of that or

1 do you need to look at it?

2 A. No, I do not.

3 Q. Okay. Taking a look at that list on
4 Exhibit 1, Dr. Garabrant, are there any of those
5 cases that are listed on Exhibit 1 in which you were
6 engaged to testify by plaintiff's counsel in a
7 personal-injury case?

8 A. I don't believe so.

9 Q. And approximately how many cases -- Strike
10 that.

11 Were there any of the cases that are
12 listed on Exhibit 1 which involved litigation other
13 than personal injury litigation, to your knowledge?

14 A. I'm not sure I know the definition of
15 personal injury litigation. Some of these are
16 workers' compensation cases, so I'm not sure how to
17 answer that.

18 Q. Fair enough.

19 In each of those cases, were you asked to
20 provide opinions as to the extent to which an
21 exposure to a particular toxic substance is capable
22 of causing a particular disease?

23 MR. LEGHORN: Object to the form.

24 A. I am not sure I understand that question.
25 As I hear it, the answer would have to be no.

1 Q. And why is it no?

2 A. Well, I'm not sure what you mean by an
3 exposure to a toxic substance is capable of causing
4 particular disease. That's the part that I'm not
5 sure what you mean.

6 Q. Okay. And how would you in your own words
7 describe in general terms the areas of testimony in
8 which you have provided opinions in the cases
9 described in Exhibit 1 or listed in Exhibit 1?

10 A. (Pause) Well, many of those cases have to
11 do with chemical exposures and whether the
12 plaintiffs' alleged exposures caused them to have
13 various illnesses.

14 Q. And were you -- Let me back up for a
15 second. If I use the term general causation, does
16 that have any significance to you? Do you
17 understand what that means?

18 A. I believe so.

19 Q. And if I use the term specific causation,
20 does that have some meaning for you?

21 A. I believe I understand that.

22 Q. Okay. In those cases listed in Exhibit 1,
23 were you always providing opinions going to general
24 causation?

25 A. I would say in most of these cases, yes.

1 I'm not sure that it was always true, but certainly
2 in most of them it was true.

3 Q. Were there also cases in which you
4 provided both a general causation opinion and a
5 specific causation opinion?

6 A. Yes.

7 Q. Can you recall looking at the list which
8 of those cases that was true for?

9 MR. LEGHORN: Object to form.

10 A. That I provided both general and specific
11 causation opinions?

12 Q. That's my question, yes.

13 A. Not from memory, no.

14 Q. From memory, is it possible for you to
15 look at this list and identify what toxin or toxins
16 or alleged toxin or toxins were at issue in each of
17 those cases?

18 A. I could do most of them from memory; not
19 all.

20 Q. In approximately how many of those cases
21 was the toxin at issue asbestos?

22 A. When you say the toxin at issue was
23 asbestos, are you referring to cases involving
24 friction products?

25 Q. I am referring to any case listed on

1 Exhibit 1 in which you provided opinions and the
2 allegation by the plaintiff was that they had become
3 ill with some disease as a result of asbestos
4 exposure.

5 A. Well, there are a substantial number of
6 cases here where the allegation was that exposure to
7 friction products has caused them to become ill, and
8 I would estimate that that's perhaps half of the
9 cases listed here.

10 Q. And am I correct that the allegation by
11 the plaintiff in those cases was that the friction
12 product contained some amount of asbestos to which
13 the plaintiff was exposed and which contributed to
14 their injury. Is that correct?

15 A. Those cases involved allegations of
16 chrysotile asbestos in friction products.

17 Q. Let me take a quick look at that list,
18 please. Thank you.

19 Is it true that some of the cases listed
20 on Exhibit 1 involved allegations of injury from
21 benzene exposure?

22 A. Yes.

23 Q. Can you identify those cases?

24 A. (Pause) I think the one listed as Garcia
25 v. Allied Diagnostic; Henricksen v. Chevron. And

1 the third one I'm not sure, Baker v. Chevron. I
2 can't recall whether the allegation included benzene
3 or not. I know it had to do with petroleum-based
4 fuels such as diesel fuel and gasoline, but I don't
5 remember whether there was an allegation of benzene
6 exposure.

7 Q. In the Garcia v. Allied Diagnostic case,
8 do you recall what the injury was of the plaintiff,
9 or injuries, that was at issue?

10 A. I believe Mr. Garcia had a non-Hodgkin's
11 lymphoma.

12 Q. And do you recall what the substance of
13 your opinions in the case were in Garcia?

14 A. Mr. Garcia was a printer and it was my
15 opinion that his work as a printer did not cause his
16 non-Hodgkin's lymphoma.

17 Q. And is it your opinion generally that
18 exposure to organic solvents is not capable of
19 causing non-Hodgkin's lymphoma?

20 MR. LEGHORN: Object to the form.

21 A. It is my opinion that there is not
22 sufficient scientific evidence to reach a conclusion
23 that exposure to solvents causes non-Hodgkin's
24 lymphoma.

25 PHONE VOICE: I'm having a little trouble

1 hearing Dr. Garabrant.

2 MR. LEGHORN: Try to keep your voice up.

3 MR. SULLIVAN: We're having difficulty

4 down here at the end of the table too,

5 Dr. Garabrant.

6 THE WITNESS: I will speak more loudly.

7 MR. SULLIVAN: Thank you.

8 BY MR. JENSEN:

9 Q. Is it also your opinion, Doctor, that the
10 scientific evidence is insufficient to conclude that
11 benzene specifically is capable of causing non-
12 Hodgkin's lymphoma?

13 MR. LEGHORN: Object to the form.

14 A. It's my opinion that the scientific
15 evidence is not adequate to support a conclusion
16 that benzene causes non-Hodgkin's lymphoma.

17 Q. And were both of those opinions part of
18 the opinions you delivered in the Garcia case?

19 A. I'm not sure I recall exactly what my
20 opinions were in Garcia, but I think that's roughly
21 correct.

22 Q. Turning to the Henricksen versus Chevron
23 case, do you recall what the injury alleged in
24 Henricksen was?

25 A. I am not sure, but I believe it was acute

1 myelogenous leukemia. I could be wrong, though.

2 From memory I'm not sure.

3 Q. All right. Do you recall what the
4 substance of your opinions in that case were?

5 A. Mr. Henricksen worked as a fuel truck
6 driver, to the best of my recollection, and it was
7 my opinion that his exposure to motor fuels,
8 including gasoline and diesel fuel, did not cause
9 his AML.

10 Q. And was your opinion in that regard based
11 on the lack of evidence of sufficient exposure?

12 A. It was based on the scientific evidence
13 that fails to show that either gasoline or diesel
14 fuel is causally associated with increased risk of
15 AML.

16 Q. So am I correct in understanding that you
17 were providing a general causation opinion in that
18 case? Is that correct?

19 A. Yes. And I believe I also provided a
20 specific causation opinion.

21 Q. And with respect to your general causation
22 opinion, your general causation opinion was tied to
23 the specific kinds of products and materials that
24 Mr. Henricksen alleged exposure to. Correct?

25 A. Yes, my opinion was tied to the alleged

1 exposures of the plaintiff.

2 Q. Did the plaintiff allege that the benzene
3 in the gasoline was the cause of his injury?

4 A. I don't recall offhand.

5 Q. Do you recall what type of AML
6 Mr. Henricksen had?

7 A. No.

8 Q. Do you recall what injuries were at issue
9 in Baker versus Chevron?

10 A. There were four plaintiffs in the Baker
11 versus Chevron case. They suffered from monoclonal
12 gammopathy of unknown significance, or MGUS; a
13 chronic lymphadenopathy that persisted for years but
14 in which no cancer diagnosis was reached; AML that
15 was successfully treated, I believe. And I take
16 that back. I don't recall if there was an AML.
17 There was a case of Hodgkin's disease that was
18 successfully treated with radiation therapy and the
19 plaintiff subsequently developed breast cancer in
20 the radiation field; and one of the plaintiffs also
21 had a monoclonal gammopathy.

22 Q. So there were two monoclonal gammopathies
23 in the group of four?

24 A. That's correct. So one of the plaintiffs
25 had two diagnoses. Actually, and another one of

1 them had a diagnosis of Hodgkin's disease and then
2 subsequent breast cancer.

3 Q. Do you recall what substances the
4 plaintiffs alleged were causing these various
5 diseases?

6 A. The allegations revolved around a refinery
7 in which alleged contamination of groundwater under
8 the neighborhood where these plaintiffs lived had
9 caused their disease.

10 Q. Where was that case?

11 A. In southern Ohio. I'm blocking out the
12 name of the town. Hooven, Ohio.

13 Q. And Exhibit 1 indicates that you testified
14 both in a deposition and in a Daubert hearing in
15 that case. Is that correct?

16 A. Yes.

17 Q. Do you know what the outcome of the
18 Daubert hearing was?

19 A. I do not.

20 Q. As far as you can recall, do any of the
21 cases listed in Exhibit 1 involve allegations of APL
22 from exposure to benzene or benzene-containing
23 materials?

24 A. Not to my recollection.

25 Q. You have also provided testimony in legal

1 cases before November of 2004. Is that correct?

2 A. Yes.

3 Q. As best you can recall, have any cases in
4 which you have ever testified involved allegations
5 of acute promyelocytic leukemia from exposure to
6 benzene or benzene-containing materials?

7 A. I don't believe so.

8 Q. Do you recall when was the first time you
9 ever provided legal testimony in litigation?

10 A. I believe it was in the mid 1980s.

11 Q. Have you ever provided testimony at the
12 request of a plaintiff's counsel in a personal-
13 injury case?

14 A. Yes.

15 Q. Do you know how many times?

16 A. Offhand I do not know.

17 Q. And what kinds of cases, if you can
18 recall?

19 A. Well, the first time I gave testimony in
20 the mid 1980s had to do with a plaintiff who had
21 colon cancer in which he alleged it was caused by
22 asbestos exposure and I testified on behalf of the
23 plaintiff.

24 Q. Any other examples of testifying on behalf
25 of the plaintiff in a personal-injury case that you

1 can recall?

2 A. There have been numerous cases where
3 people have alleged injury from various chemicals
4 causing asthma, chronic dermatitis and other
5 conditions where I've testified on their behalf.

6 Q. Aside from the example of a plaintiff
7 alleging that his asbestos exposure was related to
8 his colon cancer, can you recall other examples of
9 cases in which you've testified on behalf of a
10 plaintiff who alleged that a chemical caused him to
11 develop cancer? Or her.

12 A. (Pause) There may well have been. I
13 can't recall any from memory, though.

14 Q. All right. Is it fair to say that of the
15 cases listed on Exhibit 1, that the vast majority if
16 not all of them involve allegations by a plaintiff
17 that they have developed cancer as a result of one
18 or more chemical exposures?

19 A. No.

20 Q. That's not fair to say?

21 A. No.

22 Q. Please identify for me the cases listed on
23 Exhibit 1 which involved injuries other than cancer
24 and, to the best of your recollection, what those
25 allegations of injury were.

1 A. I'm not sure I can do that for each and
2 every case. I will do my best, however.

3 Essenmacher versus Orkin Exterminating
4 Company, I believe the plaintiff alleged multiple
5 chemical sensitivity from pesticides.

6 Jerry Ward versus Albion Personnel
7 Services was not a cancer case. I don't remember
8 what Mr. Ward had.

9 MDL Docket 1535, Welding Rod Products, had
10 to do with allegations that welding caused
11 Parkinson's disease.

12 Presler versus Lincoln Electric involved
13 allegations that welding caused Parkinson's disease.

14 Karen Brown versus Christus Spohn Health
15 System had to do with a woman who received a barium
16 enema and they perforated her colon and she got
17 barium in her peritoneum and alleged that she had
18 barium poisoning.

19 I could go on, but that's out of the first
20 maybe eight or nine, about half of them had nothing
21 to do with cancer, and I think that that pattern is
22 true probably throughout the entire exhibit.

23 Q. I have put an exhibit sticker marking as
24 Exhibit 2 what I believe is a copy of your report in
25 this case, Dr. Garabrant, if you would verify that

1 that's true.

2 A. Yes.

3 Q. Have you done additional work in this case
4 since you prepared the report marked as Exhibit 2?

5 A. Yes.

6 Q. What additional work have you done?

7 A. I have reviewed Dr. Martyn Smith's
8 supplemental declaration. I have reviewed
9 additional scientific literature. I have reviewed
10 the declarations prepared by Dr. Pyatt and
11 Dr. Bennett, and I have reviewed Dr. Martyn Smith's
12 deposition testimony.

13 Q. Have you formed additional opinions that
14 are not reflected in Exhibit 2?

15 MR. SULLIVAN: Excuse me, counsel. Could
16 you just identify Exhibit 2 by the date of the
17 report?

18 MR. JENSEN: It's dated November 19, 2008.

19 MR. SULLIVAN: Thank you.

20 A. I have formed opinions regarding
21 statements made by Dr. Smith in his supplemental
22 declaration and in his deposition testimony.

23 Q. What opinions are those?

24 A. We would have to go through his
25 declaration and his deposition in order to identify

1 all of them.

2 Q. Dr. Garabrant, have you brought with you
3 today copies of Dr. Smith's original declaration and
4 supplemental declaration?

5 A. Yes.

6 Q. At the time you prepared Exhibit 2, you
7 had already reviewed Dr. Smith's original
8 declaration. Is that correct?

9 A. I believe I had.

10 Q. Do you have any opinion today relating to
11 Dr. Smith's original declaration that you did not
12 express in your report marked as Exhibit 2?

13 A. I believe Exhibit 2 contains all of my
14 opinions regarding Dr. Smith's original declaration.

15 Q. All right. If you could put in front of
16 you Dr. Smith's supplemental declaration, please.
17 And for the record, I will mark a copy of that as
18 Exhibit 3. And let me verify that Exhibit 3 is
19 indeed the same thing as what we're talking about,
20 Dr. Smith's supplemental declaration.

21 A. Yes, I believe that to be the supplemental
22 declaration.

23 Q. Okay, thank you.

24 Can you tell me what opinions you have
25 developed relating to Dr. Smith's supplemental

1 declaration that are not reflected in your report
2 which is Exhibit 2?

3 A. Well, okay, I'll do my best. The first is
4 at Smith's paragraph 2 where he claims defense
5 experts make the error that because of this unique
6 clinical standing, it is also a unique etiological
7 or causal entity, and there he's referring to APL.
8 I don't think that I made any such error. There is
9 simply good scientific evidence that the etiology of
10 APL has nothing to do with benzene, which makes it
11 different than some of the other forms of AML.

12 At paragraph 6 Dr. Smith says "Thus,
13 common environmental causes are likely for these
14 leukemias and may include low-level benzene
15 exposure, quinone compounds in our diet, ionizing
16 radiation and chemicals producing oxidative stress."
17 I believe he is simply wrong on that with respect to
18 APL. There is not evidence that benzene is a risk
19 factor for APL.

20 Dr. Smith reiterates a similar view at the
21 top of the next page, and I would again respond with
22 the point I just made.

23 At paragraph 7 Dr. Smith comments "If APL
24 were really so different from AML, why is it not
25 listed as a separate cause of death on death

1 certificates and why are there no epidemiological
2 studies which focus only on APL and benzene
3 exposure?" He then goes on to say "The problem is
4 that death certificates and case referrals in
5 epidemiology studies consider AML as one disease or
6 in our analogy as a car, independent of the color."

7 Dr. Smith seems to have forgotten that he
8 has identified a substantial body of epidemiological
9 literature which looks specifically at APL and
10 benzene and which fails to show any association, so
11 it's odd that he would question the existence of
12 that literature when he has reviewed it.

13 Secondly, death certificates in
14 epidemiology do not always consider AML as one
15 disease. There are many studies which consider
16 different forms of AML and Dr. Smith has done some
17 work to identify those studies.

18 At paragraph 8 he comments that "Because
19 APL is seen in studies of workers exposed to benzene
20 where the subtypes of AML have been separately
21 analyzed," I would comment that he has just
22 contradicted his previous point that there are no
23 studies that have done that, and he goes on to say,
24 quote, "and has been found at higher levels than
25 expected with an odds ratio that exceeds 2." There

1 are no studies that show an odds ratio that exceeds
2 2 for APL and benzene, and Dr. Smith has failed to
3 show any.

4 At paragraph 12 Dr. Smith comments on the
5 study by Golomb, G-o-l-o-m-b, and having reviewed
6 that myself, Dr. Smith has not counted up the number
7 of cases properly and has not calculated the odds
8 ratios properly, and there is no incidence of APL
9 that is approximately two times higher in those
10 patients with probable benzene exposure than in
11 unexposed patients, as Dr. Smith claims.

12 At paragraph 14 Dr. Smith says he has used
13 this information to calculate an odds ratio for APL
14 in the Chinese cohort study that is in excess of 2.
15 And I believe he has made errors in the assumptions
16 behind his calculations as well as in his methods of
17 calculating.

18 At paragraph 16 Dr. Smith comments
19 regarding my critique, quote, "Overall, his critique
20 could have some validity if APL were a separate
21 disease, which it is not." I believe Dr. Smith in
22 paragraph 2 indicated that, quote, "I do not dispute
23 that APL is a unique therapeutic entity among the
24 myeloid leukemias," so I am perplexed by his
25 contradiction of his earlier paragraph.

1 He then goes on to say at paragraph 16,
2 quote, "My point is that we know benzene causes AML
3 and that the Travis study shows that a significant
4 proportion of these AMLs, four of nine cases
5 analyzed, see below, are APL cases." I disagree
6 that we know that four of the nine are APLs because
7 of deficiencies in the diagnostic information in the
8 Travis report.

9 At paragraph 19 Dr. Smith attributes a
10 statement to me at lines 3 through 7 that I don't
11 believe I ever made. He then goes on to make an
12 error in claiming that the proportion of acute
13 leukemias that were APL is 28.6 percent. That's not
14 correct and the literature that Dr. Smith claims to
15 have read shows it's not correct, so I don't know
16 how he arrived at that conclusion.

17 He then calculates an odds ratio -- I'm
18 still at paragraph 19, at the top of page Smith
19 000704 -- he claims he calculated an odds ratio. In
20 fact he did not calculate an odds ratio; he is
21 wrong. His calculation is not based on any reliable
22 methodology. Moreover, he did not calculate any
23 test of statistical significance or any confidence
24 interval regarding his measure of association, which
25 he incorrectly calls an odds ratio, and therefore he

1 has no basis for saying at the bottom of paragraph
2 19, quote, "Thus, one can say to a reasonable degree
3 of scientific probability that the odds ratio for
4 APL associated with benzene exposure in the Chinese
5 cohort is more likely than not greater than 2."

6 At paragraph 20 Dr. Smith makes a comment
7 that my calculations have no validity because the
8 proportion of APL among AML cases in China appears
9 to be much higher than among AML cases in the U.S.;
10 and two, the Chinese cohort is a group of healthy
11 workers that is not representative of the general
12 population, making direct comparisons to other
13 population incidence untenable. He is simply wrong.
14 There's no basis for those claims.

15 At paragraph 21 Dr. Smith claims that both
16 Dr. Bennett and I ignore the difficulties of
17 performing research in China in the time period
18 before 1987. He's wrong about that; I don't ignore
19 that. I simply pointed out that the diagnoses of
20 APL were not reliable and were not based on the
21 diagnostic materials that are essential in order to
22 reach those diagnoses.

23 Dr. Smith then goes on to address a number
24 of responses to the declaration by Dr. David Pyatt.
25 Although I do not agree with a number of things he

1 has said there, I will not address those since those
2 are really directed at Dr. Pyatt's opinions.

3 Q. Starting with your opinion in regard to
4 paragraph 2 of Exhibit 3, Dr. Smith's supplemental
5 declaration at paragraph 2, as I recall, you told me
6 that your opinion is that there is good evidence
7 that the etiology of APL has nothing to do with
8 benzene exposure. Is that correct?

9 A. I don't think that's what I've said.

10 Q. Okay. The record will reflect what you've
11 said. Here's my question about what you've said.
12 Are you basing your opinion in regard to paragraph 2
13 on any evidence other than epidemiological studies?

14 A. Yes.

15 Q. What evidence besides epidemiological
16 studies?

17 A. Well, first let me comment on the role of
18 the epidemiological studies. The epidemiological
19 studies provide a direct test of the hypothesis that
20 APL is causally associated with benzene and they
21 fail to show that association, and so they provide
22 the strongest evidence in response to Dr. Smith's
23 allegation that APL is not a unique etiological or
24 causal entity with respect to benzene.

25 Secondly, I rely on more general concepts

1 derived from my education, training and experience
2 in medicine that goes to the issue of how do we
3 actually differentiate one disease from another. In
4 other words, why is rheumatoid arthritis different
5 from systemic lupus erythematosus? Why is
6 rheumatoid arthritis different from osteoarthritis?
7 Why is tuberculosis different from other mycoplasmal
8 diseases?

9 The history of medicine provides a firm
10 foundation for the overall process of
11 differentiating diseases based on seeing that they
12 have different clinical presentations, different
13 clinical courses, and different treatments, as well
14 as different etiologies. And so over the history of
15 medicine we have split broad diagnoses into more
16 clearly defined and differentiated diseases. At
17 times we have taken diseases that appeared to be
18 different and then realized that they were different
19 manifestations of the same underlying disorder, and
20 so on.

21 What I believe is clear for APL, and
22 particularly since cytogenetics and
23 immunophenotyping has been brought into widespread
24 use in the diagnosis of leukemias, is that not only
25 is it a distinct clinical entity, but to the extent

1 that we have been able to examine the association of
2 that distinct entity with various putative causes,
3 it has different causes than some of the other forms
4 of AML.

5 Q. Will you be providing any opinions in this
6 case dealing with the cellular origins for APL or
7 any other form of leukemia?

8 MR. LEGHORN: Object to the form.

9 A. No.

10 Q. Will you be providing any opinions in this
11 case relating to the natural history of how APL
12 develops?

13 MR. LEGHORN: Object to the form.

14 A. I'm not sure I know exactly what you mean
15 by that question. If that question includes how it
16 develops with respect to etiology, yes.

17 Q. Will you be providing any testimony in
18 this case relating to what is going on at the
19 cellular, subcellular, genetic level that is causing
20 APL to develop?

21 MR. LEGHORN: Object to form.

22 A. I have not been asked to cover that area
23 and at the moment I have no plans to testify in that
24 area.

25 Q. Moving on to paragraph 6 of the

1 supplemental declaration of Dr. Smith, which is
2 Exhibit 3, the first sentence of paragraph 6 says
3 "Common genetic variants in a gene called NQO1 for
4 NAD(P)H:quinone oxidoreductase, one, confer
5 susceptibility to AML and two studies in England and
6 China show that this is strongest for de novo AML
7 cases harboring translocations and inversions,
8 including t(15;17) in APL." Do you have any
9 opinions as to whether that sentence is correct?

10 A. I do not have any opinions regarding
11 whether that sentence is correct.

12 Q. I won't read the second sentence but I'll
13 ask you the same question about the second sentence.
14 Do you have any opinions as to whether the second
15 sentence of paragraph 6 is correct?

16 A. I do not.

17 Q. Am I correct, Dr. Garabrant, that your
18 opinions regarding paragraph 6 of Dr. Smith's
19 opinion are really relating to the third sentence of
20 the paragraph, which says "Thus, common
21 environmental causes are likely for these leukemias
22 and may include low-level benzene exposure" and he
23 goes on from there. Is that right, your opinions
24 are addressed at that sentence?

25 A. Well, my opinions are addressed at the

1 hypothesis that Dr. Smith has that since he has a
2 possible mechanism by which environmental causes
3 might cause APL, he then makes a conclusion that
4 common environmental causes are likely. And I think
5 that his conclusion is not borne out by the
6 available evidence. He's got a hypothesis that does
7 not have proof.

8 Q. The fourth sentence of paragraph 6,
9 Dr. Garabrant, says "Further, in a recent
10 epidemiological study of childhood AML, we separated
11 the, quote, good prognosis, close quote, AMLs with
12 translocations and inversions from other forms of
13 AML and found a strong association between parental
14 exposure to solvents and increased risk of these
15 specific forms." Then he gives a citation. Up to
16 that point in the sentence, do you have any opinions
17 as to whether what I've read so far is correct?

18 A. I don't have an opinion regarding that
19 sentence. This litigation is not about childhood
20 AML and so I don't know the relevance of Dr. Smith's
21 reference to that.

22 Q. Well, the rest of the sentence after the
23 citation says "Again suggesting a common cause for
24 APL and other AMLs harboring translocations and
25 inversions." I assume you disagree with that

1 portion of the sentence. Am I correct?

2 A. Well, again Dr. Smith is talking about
3 childhood AML. This is not a case about childhood
4 AML. The childhood leukemias are different than the
5 adult leukemias in many ways, and I do not know the
6 literature on childhood AML and parental exposure to
7 solvents in depth and I didn't review it with
8 respect to this case, and so I can't comment on the
9 validity of his claim without having actually
10 explored that area in detail. All I can say is I
11 think he is invoking something of marginal or no
12 relevance to the case at hand.

13 Q. And your view that the evidence he is
14 invoking is of marginal or no relevance is based on
15 the fact that it in this particular instance comes
16 from a study relating to childhood AML which you
17 believe is of marginal or no relevance to an adult
18 AML. Is that correct?

19 A. Well, there is a large literature on
20 childhood leukemia looking at parental exposures to
21 solvents, benzene, pesticides, incense, parental
22 occupations, and Dr. Smith is referring to a single
23 study pulled out of that large body of literature
24 and reaching a conclusion based on that single
25 study. And my comment is if we're going to talk

1 about childhood AML, that is a very large area that
2 needs to be examined rigorously and systematically.
3 You cannot reach a valid conclusion by citing to a
4 single study that has a finding that you like,
5 without considering all of the rest of the body of
6 literature.

7 This case is not about childhood AML and
8 neither Dr. Smith nor I have done a rigorous
9 examination of that large body of literature, and so
10 I think it is irrelevant to simply pick a single
11 study and to say "See, this supports me."

12 Q. Do you have any knowledge as you sit here
13 today, Dr. Garabrant, as to how many other studies,
14 if any, have looked at comparing translocations and
15 inversions of AMLs that have the so-called good
16 prognosis with other forms of AML in childhood AMLs?

17 A. Again, as I have already stated, I have
18 not reviewed the literature on childhood AML in
19 preparation for this case. I think it is of
20 marginal or no relevance to the case, but if
21 Dr. Smith wants to bring that up, I'd be happy to
22 review it and provide opinions regarding childhood
23 AML and what we know about its etiology. Dr. Smith
24 has not done that. He has cited to a single study.

25 Q. Turning to paragraph 12 of Dr. Smith's

1 supplemental declaration, you indicated that it was
2 your opinion that Dr. Smith had not correctly
3 counted the cases from the Golomb study. Is that
4 right?

5 A. I believe so.

6 Q. How do you believe the Golomb cases should
7 be counted?

8 A. (Pause) In the 58 unexposed patients in
9 the Golomb study, there are five patients who have
10 APL. Dr. Smith counts four and he then uses that
11 erroneous count to calculate an odds ratio of 2.1.
12 So he's counted wrong.

13 Q. Other than the four should have been a
14 five, are there any other errors in the calculation
15 that Dr. Smith made that is reflected in paragraph
16 12, in your opinion?

17 A. From memory, I don't know exactly how he
18 made his calculation, so I would have to repeat that
19 calculation in order to verify whether he has done
20 his arithmetic properly.

21 Q. Well, without pulling out a calculator to
22 do the arithmetic, what I'm asking you is whether
23 you have any methodological criticism of the way
24 that that calculation was done as it is described in
25 paragraph 12?

1 A. Again, without working back through what
2 he has described, I can't comment. I'd be happy to
3 do that, but I can't do that without sitting down
4 and working back through his logic and doing the
5 arithmetic.

6 Q. So it is fair to say as you sit here today
7 you have not developed any opinions beyond what
8 you've already told me regarding any errors that
9 Dr. Smith may have made in paragraph 12. Is that
10 correct?

11 MR. LEGHORN: Object to the form.

12 A. Well, no. I've already told you that he's
13 counted wrong. There are five cases; he only
14 counted four of them, and therefore his calculation
15 is in error.

16 Q. My question intended to encompass that
17 criticism, Doctor. But besides the counting four
18 cases when he should have counted five, as you sit
19 here today you have identified no other
20 methodological errors that Dr. Smith made in his
21 calculations at paragraph 12. Is that correct?

22 MR. LEGHORN: Object to the form.

23 A. No. In fact, to the extent I understand
24 what he's done, he chose to exclude one of those
25 five cases because there were not confirmed or I

1 should say because cytogenetic testing was not done
2 or was not reported in the Golomb paper. He is
3 inconsistent in his methods when he comes to the
4 Travis paper where he specifically includes cases
5 that have no confirmed cytogenetic abnormalities.
6 So in Golomb he excludes the one where there's no
7 confirmed cytogenetic abnormality; in Travis he
8 specifically includes them. That is a methodologic
9 inconsistency that is inconsistent with rigorous and
10 carefully done science.

11 Q. Again, that criticism goes to how many APL
12 cases are reflected in Golomb that should have been
13 counted in his calculation. Is that right?

14 MR. LEGHORN: Object to the form.

15 A. No. It goes to his methods and it goes to
16 the reliability of his thinking.

17 Q. Besides the number of APLs that are used
18 in the calculation in paragraph 12, as you sit here
19 today you have no other criticisms related to that
20 calculation. Is that correct?

21 MR. LEGHORN: Object to the form. It has
22 been asked and answered.

23 A. As I have already answered, in order to
24 determine whether I have other criticisms I would
25 need to check his arithmetic.

1 Q. Which you have not done as we sit here.

2 Correct?

3 A. I have not done it as I sit here. I did
4 work through his arithmetic previously, but I don't
5 have it and I don't recall whether I found any other
6 errors.

7 I should also mention that Dr. Smith fails
8 to provide any test of statistical significance or
9 any confidence interval with his calculated odds
10 ratio, and as a consequence of that, it is unclear
11 whether that odds ratio is reliable or not. Let me
12 rephrase that: It is unclear whether that odds
13 ratio is reliably different than 1.0 or not.

14 Q. Is it your view, Dr. Garabrant, that an
15 epidemiological study that shows an odds ratio or a
16 relative risk or a standardized mortality ratio or
17 whatever the equivalent measure of association is
18 for that type of study which is not statistically
19 significant but is above the odds ratio of relative
20 risk, whatever is above 1, that such a study
21 provides no evidence of an association?

22 MR. LEGHORN: Object to the form.

23 A. Well, the evidence of association from an
24 epidemiological study is whatever is calculated by
25 the SMR or odds ratio or whatever other measure of

1 association one calculates. The issue is whether
2 that measure of association is meaningfully
3 different from the value 1.0, which means there is
4 no association.

5 Q. Right. And my question for you is: If
6 the association calculated is not statistically
7 significant, is it your opinion that such a study,
8 regardless of whatever else its findings or data
9 show, provides no meaningful evidence of an
10 association?

11 A. It is my opinion that when you have
12 measures of association that are not statistically
13 significant, the data are reasonably compatible with
14 there being no association whatsoever.

15 Q. And if the data are reasonably compatible
16 with there being no association whatsoever, is it
17 your opinion that any such data should not be
18 considered as evidence of an association?

19 A. It's my opinion that good scientists
20 consider all reliable and relevant data, which is
21 what I think Dr. Smith systematically does not do in
22 his supplemental declaration.

23 Q. Let me pose a hypothetical to you, Doctor.
24 If there were ten epidemiological studies all
25 looking at the issue of whether benzene exposure may

1 cause APL and each of those studies found an
2 elevated relative risk above 2.0 but the confidence
3 interval for each of those studies encompassed 1.0,
4 so all ten of them come up with similar results,
5 none of them are statistically significant but all
6 of them have a relative risk that is calculated at
7 above 2.0. Would it be your opinion that the data
8 is insufficient to conclude that more likely than
9 not benzene exposure causes APL in that
10 circumstance?

11 MR. LEGHORN: Object to the form.

12 A. Your hypothetical isn't adequate to reach
13 any such conclusion one way or the other.

14 Q. I would add to the hypothetical that's the
15 only epidemiological evidence that was available
16 that was specific to both APL and benzene exposure.

17 MR. LEGHORN: Object to the form.

18 BY MR. JENSEN:

19 Q. Does that make any difference to you?

20 MR. LEGHORN: Object to the form.

21 A. No. The hypothetical still is not
22 sufficient to reach any meaningful conclusion one
23 way or the other.

24 Q. In your opinion, is it ever appropriate to
25 reach an opinion that a particular chemical causes a

1 particular disease in the absence of statistically
2 significant epidemiological evidence that supports
3 that proposition?

4 MR. LEGHORN: Object to the form.

5 A. It is my opinion that when epidemiological
6 evidence exists, you must consider it.

7 Q. That wasn't my question. Do you need me
8 to repeat the question?

9 A. No. If your question is whether you can
10 reach a conclusion of causation in the absence of
11 epidemiological evidence, my answer is that's not
12 the instance we're in. We have a case here where
13 there is a substantial body of epidemiological
14 evidence which must be considered. There are many
15 settings where we can conclude causation in the
16 absence of epidemiology, such as gunshot wounds,
17 such as falling out of airplanes when your parachute
18 doesn't open. You don't need epidemiology. But
19 when you have situations such as low-level chemical
20 exposures and the development of diseases years or
21 decades later and there is epidemiologic evidence,
22 you have to consider it and you have to consider it
23 fully.

24 Q. Is it your view, Dr. Garabrant, that the
25 available epidemiological evidence that exists with

1 respect to acute promyelocytic leukemia and benzene
2 exposure establishes that benzene exposure cannot
3 cause APL?

4 MR. LEGHORN: Object to the form.

5 A. As Dr. Smith himself pointed out, you
6 cannot prove the null hypothesis. I agree with that
7 statement. He got that one right. The
8 epidemiologic evidence that we have regarding
9 benzene and APL fails to prove the alternative
10 hypothesis.

11 Q. Am I correct, Dr. Garabrant, that because
12 in your view the epidemiological evidence with
13 respect to APL and benzene fails to prove the
14 alternative hypothesis, it is your view that it is
15 unnecessary to look to other bodies of evidence such
16 as biological mechanism evidence to determine the
17 question of whether benzene is capable of causing
18 APL?

19 MR. LEGHORN: Object to the form.

20 A. I am a strong adherent of Austin Bradford-
21 Hill's comments on causation. And consistent with
22 Hill's comments, I believe that one has to look for
23 evidence of an association when the purpose is to
24 determine whether an environmental exposure is
25 causally associated with a disease such as APL. One

1 has to look for evidence of an association and that
2 if one does not find evidence of an association,
3 then most of the other considerations that Hill
4 discusses are rendered moot. In other words, when
5 there's no association, you can't look for dose
6 response because there's no dose response when
7 there's no association. You really can't look at
8 specificity when there's no association. You can't
9 really judge coherence of the literature -- Well, I
10 guess you can when there's no association and when
11 you see a coherent picture of lack of association,
12 you can say "You know? There's really no
13 association."

14 So your comments about biological
15 plausibility I think go to the issue of, well,
16 should you look for biological plausibility when you
17 can't find evidence of an association? And my
18 comment to that would be I've spent my career as a
19 scientist, and smart scientists can come up with a
20 biologically plausible explanation for almost any
21 hypothesis. That's not hard. The issue is whether
22 you have empirical evidence that supports that the
23 hypothesis is correct in the first place.

24 Q. And in your view the only type of evidence
25 that counts as empirical evidence to support the

1 hypothesis with respect to environmental exposures
2 and latent disease are human epidemiological
3 studies. Is that correct?

4 A. No. My opinion is that when human
5 epidemiological studies exist, you can't ignore what
6 they may say, and that's the instance we're in here.
7 We have a substantial body of literature which
8 Dr. Smith has largely identified which is directly
9 relevant to whether there is an association between
10 benzene exposure and APL. You can't ignore it.

11 MR. JENSEN: Let's take a quick break.

12 MR. LEGHORN: I was going to say, before
13 we go to another paragraph, it would be a good time
14 for a break.

15 (In recess 10:38 a.m. to 10:51 a.m.)

16 BY MR. JENSEN:

17 Q. Turning to paragraph 19 of Dr. Smith's
18 supplemental declaration, I believe you testified
19 that it was your opinion that Dr. Smith's statements
20 about the proportion or assumptions about the
21 proportion of APLs in the Chinese population is
22 wrong. Am I correct that that was your opinion or
23 that is your opinion?

24 A. Let's read back exactly what I said about
25 that, because I don't think that's what I said.

1 Q. Well, I don't want to quibble with you
2 about what your opinion is, so I'll ask you: What
3 is your opinion with regard to the proportion of
4 APLs and what Dr. Smith has to say about that in
5 paragraph 19?

6 A. Well, my first opinion is that I can't see
7 any reason in the world why you would calculate
8 something based on proportions when the data is
9 available to actually compare incidence rates. So
10 Dr. Smith chose to do a calculation that has the
11 potential to have many sources of bias instead of
12 doing a calculation that would be much more
13 reliable. So, again, his fundamental approach is
14 just wrong.

15 Q. Do you have an opinion about whether his
16 statement in paragraph 19 -- it's the third
17 sentence, I believe -- that begins "Thus, four cases
18 of APL in fourteen cases of acute leukemia would be
19 equal to the general representation of APL in the
20 Chinese population, i.e., 4/14 equals 0.286 or 28.6
21 percent." Do you have an opinion about whether the
22 statement in that sentence is correct?

23 A. Well, it's wrong. We know from the Yang
24 paper that the correct proportion is not 28.6
25 percent. Now, that's a paper that Dr. Smith read.

1 So why would he choose to make an assumption that's
2 wrong when the actual proportion is in the paper?
3 That's the sort of incorrect methodology that we see
4 recurring in Dr. Smith's opinions.

5 Q. What is the proportion reflected in the
6 Yang paper of APLs to the total ANLL incidence in
7 China?

8 A. Well, the more important question is why
9 would you bother to use proportions when you can
10 actually use the actual incidence rates? Here's the
11 problem with proportions. When one disease is
12 overrepresented, the proportions of all others are
13 underrepresented; and conversely when one disease is
14 underrepresented, the proportions of all other
15 diseases are overrepresented. So proportions by
16 their very nature are a less reliable measure than
17 are actual incidence rates.

18 So I cannot think of a reason why any
19 reliable scientist would use proportions when he has
20 access to the underlying rates. It's just the wrong
21 thing to do.

22 Q. What is the proportion of APL in ANLL as
23 reported by the Yang paper?

24 A. 18.7 percent.

25 Q. Besides what you've already testified to,

1 do you have any other specific criticisms of
2 paragraph 19 and the approach that Dr. Smith took in
3 his paragraph 19?

4 A. Yes. One of the principles of science is
5 that a good scientist considers all relevant and
6 reliable data. Dr. Smith chose not to do that in
7 paragraph 19. So by choosing to make an erroneous
8 calculation based on proportions and ignoring the
9 far more reliable and relevant incidence rates, he
10 has not followed good scientific procedure. He has
11 ignored good data and chosen to do something that is
12 highly likely to give an unreliable answer, and in
13 fact the proportion he chose was based on a poorly
14 documented statement at the very end of the Travis
15 paper where Dr. Travis says the proportion of APL in
16 de novo ANLL was similar to that in the general
17 population.

18 So he took that and said, well, it's
19 really the same, it's 28.6, when it wasn't the same;
20 it was actually, in my opinion, not even very
21 similar. And my underlying opinion is I wouldn't
22 even use that approach because it's not a good
23 approach. The Yang paper gives tell you incidence
24 rates. You can calculate an incidence ratio.

25 Q. What is the healthy worker effect?

1 A. The healthy worker effect is a term that
2 really is an umbrella term for the commonly observed
3 phenomenon in the occupational epidemiology
4 literature coming from the United States and Western
5 Europe that in many cohort studies of working
6 populations, the workers are in general healthier
7 than the general population.

8 Q. Does the healthy worker effect, in your
9 opinion, play any role with respect to analysis of
10 the Travis et al. papers and series of studies by
11 Travis, Yin, Hayes et al.?

12 A. I think it deserves careful consideration.

13 Q. How did you consider the healthy worker
14 effect and what role did it play in your opinions in
15 this case?

16 MR. LEGHORN: Object to the form.

17 A. The Travis paper reports on hematopoietic
18 malignancies among benzene-exposed workers in China.
19 If we go back to some of the earlier reports from
20 that population, we find that the Yin paper
21 published in 1989 gives us the ability to evaluate
22 whether there is a healthy worker effect among
23 benzene-exposed workers in China. And what we find
24 is that the all-cause-of-death mortality rate in the
25 benzene-exposed workers is roughly double that in

1 the nonexposed workers.

2 So not only is there not a healthy worker
3 effect in the population under study in the Travis
4 paper, it goes the other way. It's a twofold
5 increase in risk. So if Dr. Smith were really
6 interested in adjusting for the healthy worker
7 effect, he's got it backwards. In fact, that
8 working population has double the all-causes
9 mortality of the general population. And if he's
10 going to adjust for it, then he should have adjusted
11 the other way. That's point number one.

12 So he has invoked something in a manner
13 where he's actually done it backwards.

14 Number two, to my recollection Dr. Smith
15 objected to using the incidence rates in the general
16 population of China derived from the Yang paper as
17 we've discussed and he chose to use the proportion
18 of APLs among AMLs instead of using the incidence
19 rates. And in part he did that because he said
20 there was a healthy worker effect in using the
21 population reported in the Yang paper.

22 His comment suggests that although he's
23 read the Yang paper, he didn't read it very
24 carefully, because many of the populations studied
25 in the Yang paper were working populations. They

1 came from the Daqing Oil Field, the Changchun
2 Automobile Company, the Yanju Forestry Farm, the
3 Baotou Steel Company, the Pingdingshan Coal Mine,
4 the Kailuan Coal Mine, the Fengfeng Coal Mine,
5 et cetera.

6 So in fact those rates to which he
7 objected using were largely derived from working
8 populations and so it would have been perfectly
9 appropriate to have compared the rates derived from
10 the Yang study, the cancer incidence rates, to the
11 cancer incidence in the Chinese benzene cohort.
12 Perfectly appropriate to do.

13 Q. Does the Yang paper indicate what
14 proportion of the people from which the incidence
15 rate for APL is calculated are workers as opposed to
16 nonworkers?

17 A. They didn't tabulate it or they didn't
18 give the summaries, but that could easily be derived
19 from Table 1.

20 Q. With respect to your statements regarding
21 the Yin 1989 paper, did I hear you correctly that
22 Yin was calculating all cancer mortality of the
23 exposed worker population by comparing it with an
24 unexposed worker population?

25 MR. LEGHORN: Object to the form.

1 A. That's not what I said. I was talking
2 about all causes of death. You were talking about
3 cancer.

4 Q. I'm sorry. But Yin did calculate all
5 causes of death in the exposed population by
6 comparing it with an unexposed population of
7 workers. Is that right?

8 A. Yes.

9 Q. So both populations were workers with
10 respect to the calculations made by Yin. Right?

11 A. That's correct.

12 Q. Isn't comparing an exposed worker
13 population to an unexposed worker population one of
14 the methods that epidemiologists commonly use to
15 avoid any impact of the healthy worker effect?

16 A. Yes. And that's why Dr. Smith should have
17 carefully looked at the Yin paper. He would have
18 realized that there's no healthy worker effect in
19 the benzene-exposed cohort. In fact, their all-
20 cause-of-death mortality is double that in the
21 unexposed population.

22 Q. Well, in order to measure the healthy
23 worker effect, if any, don't you have to compare a
24 worker population to a nonworker population?

25 A. If you want to measure the healthy worker

1 effect, yes, you have to.

2 Q. So there could be a number of different
3 reasons besides any healthy worker effect or lack
4 thereof for the mortality in the exposed workers
5 described in Yin to be higher than the unexposed
6 workers. Right?

7 A. Yes. And the most likely reason is far
8 better disease surveillance in the benzene-exposed
9 than in the unexposed. In other words, what this
10 really suggests is that there is systematic
11 difference in the way they're doing their
12 surveillance in the benzene-exposed population
13 compared to the unexposed. I mean, a doubling of
14 mortality is extraordinary. We never see that.
15 Never. It's absolutely extraordinary.

16 Q. Another contributor could be that the
17 exposures themselves are causing mortality.
18 Correct?

19 A. I think that's extremely unlikely here.
20 What exposures could you hypothesize that would
21 double mortality across all causes of death? I
22 can't think of any. There are no chemicals that
23 cause increases in cardiovascular disease,
24 cerebrovascular disease, renal disease, pulmonary
25 disease, all causes of cancer, diabetes, infectious

1 diseases, accidents, suicides, psychiatric disease,
2 neurologic disease. There aren't any.

3 Q. Exhibit 2, which is your report of
4 November 19, at page 3 -- Are you with me, Doctor?

5 A. Yes.

6 Q. Okay. The first full paragraph there on
7 page 3 discusses the Travis study and your analysis
8 of Dr. Smith's opinion of the Travis study. Is that
9 right?

10 A. Well, the first paragraph is what it is.

11 Q. And in that paragraph it says "If we use
12 Dr. Smith's estimate of three to eight cases of APL
13 per one million person-years," that estimate that
14 you're describing there is an estimate based on
15 incidence in the United States. Is that correct?

16 A. I don't recall where Dr. Smith got that
17 estimate, but that's his estimate.

18 Q. And you were comparing in that paragraph
19 an incidence rate that derives from a general
20 population with the incidence in the benzene-exposed
21 group discussed in Travis. Is that correct?

22 A. Well, I'm using Dr. Smith's own estimate
23 of the incidence rate to make an estimate of the
24 expected number of cases you'd see in Travis.

25 Q. And I'm asking you whether you even know

1 if that incidence rate that is estimated, you say,
2 by Dr. Smith at about three to eight cases of APL
3 per year, is that an incidence rate for a worker
4 population or for a general population?

5 A. I don't recall where Dr. Smith got that
6 number. To the best of my recollection, he never
7 said. Or, I should say, the range.

8 Q. So I take it, then, that it doesn't make
9 any difference to you whether or not the incidence
10 rate that Dr. Smith is using of three to eight cases
11 of APL per one million person-years is an incidence
12 rate derived from a general population or a worker
13 population for purposes of your analysis?

14 A. Well, it does matter. And in fact it is
15 entirely consistent with the incidence rate of APL
16 in what I was referring to as the Yang study, it's
17 actually Yang Chongli, in the Chinese Medical
18 Sciences journal. In that study, which is largely
19 derived from working populations in China, the
20 incidence rate is 3.5 per million person-years, so
21 it is entirely consistent with Dr. Smith's
22 estimates.

23 Q. Dr. Garabrant, have you ever published any
24 article, textbook chapter or any other publication
25 in which you've expressly discussed the issue of

1 whether benzene exposure is capable of causing acute
2 promyelocytic leukemia?

3 A. I don't believe so.

4 Q. Have you ever published a study of
5 benzene-exposed workers and leukemia risk?

6 A. I'd have to look at my C.V. I don't
7 recall any. I know my colleagues and I published a
8 study of childhood leukemia looking at parental
9 exposures where we specifically looked at benzene
10 and other solvents. I would have to look back
11 through my C.V. to see if there's anything else.
12 I know I've done research that is not published that
13 has looked at that issue.

14 Q. Is the paper you describe on childhood
15 leukemia the Buckley et al. paper of 1989?

16 A. Yes.

17 Q. What unpublished work have you written
18 with respect to benzene and leukemia?

19 A. We did a large cohort mortality study of
20 auto industry workers where we looked at each and
21 every one of about ninety causes of death, including
22 leukemias, and over a hundred different exposures.
23 In that list of exposures we included many different
24 types of solvents, oils, lubricants, greases,
25 machining fluids, petroleum distillates, paint

1 thinners. So these were auto industry workers and
2 we looked at leukemia mortality.

3 Q. In that cohort of workers, were there any
4 measured air concentrations of benzene that were
5 available for purposes of exposure analysis?

6 A. We did not have any at the time we did
7 this study. I am aware that there is some
8 information regarding industrial hygiene data from
9 the plants we studied, but it was not incorporated
10 into our research.

11 Q. Was that study commissioned by one or more
12 of the auto companies?

13 A. No. It was commissioned by the United
14 Auto Workers-Ford Motor Company National Joint
15 Committee.

16 Q. What were your findings with respect to
17 benzene and -- Excuse me. What were your findings
18 with respect to solvent petroleum exposures and
19 leukemia in that study?

20 A. To the best of my recollection, I don't
21 think we found any significant associations.

22 Q. Are you familiar generally with
23 Dr. Smith's work in the field of benzene toxicology?

24 A. I have seen some of it. I wouldn't say
25 I'm generally familiar with it.

1 Q. Do you have an opinion as to whether
2 Dr. Smith is considered a highly respected benzene
3 toxicology researcher?

4 MR. LEGHORN: Object to the form.

5 A. I don't know whether he is a highly
6 respected benzene toxicology researcher. I do
7 believe he's a highly respected scientist.

8 Q. Did you read either or both of
9 Dr. Cranor's reports in this case?

10 A. I read Dr. Cranor's report dated October
11 9, 2008.

12 Q. Dr. Cranor issued a supplemental report on
13 December 9, 2008. Did you have the opportunity to
14 review that?

15 A. I don't believe so.

16 Q. In paragraph 6 of his supplemental report
17 Dr. Craner states, quote, "As a part of a review of
18 the science, whether for assessing claims in physics
19 or human health, scientific judgment is critically
20 involved not only for drawing the ultimate
21 conclusions but also for a number of steps along the
22 way," close quote. Do you agree with that
23 statement?

24 MR. LEGHORN: Object to the form.

25 A. I've never read the report. Could I see

1 it and read that section of it? (Pause) Okay,
2 what's your question?

3 Q. If I may have it back, I'll repeat the
4 question.

5 A. Well, I need it. Do you want me to read
6 that sentence back?

7 Q. Yes. The sentence that I read to you, and
8 my question was simply whether you agreed with that
9 sentence.

10 A. I would say yes, I agree with that
11 sentence, and I strongly agree with the preceding
12 paragraph in which Dr. Cranor says "A scientist in
13 reviewing and assessing all the scientific evidence
14 for a conclusion such as whether benzene exposure
15 can cause APL must consider and integrate all the
16 available relevant evidence." And that is of course
17 the critical issue that Dr. Smith has not done. He
18 has not properly looked at the incidence rates of
19 APL in China. He has relied on calculations that
20 are not reliable, choosing them over calculations
21 based on much more reliable evidence. He has
22 invoked a healthy worker effect where none exists as
23 a potential objection to comparing incidence rates
24 in the benzene-exposed cohort in the Travis paper
25 compared to incidence rates in other populations in

1 China.

2 If he were to integrate and evaluate all
3 the relevant evidence, I think he would find that
4 his conclusions are not well-supported by the data.

5 Q. And in your opinion, biological mechanism
6 evidence is not relevant in this instance. Correct?

7 A. That is not my opinion.

8 Q. It is your opinion that because the
9 epidemiology in your view fails to show an
10 association between benzene and APL, that
11 consideration of biological mechanism evidence is
12 unnecessary. Isn't that true?

13 MR. LEGHORN: Object to the form.

14 A. That is not my opinion. It is my opinion
15 that the epidemiology provides a critical test of
16 whether hypotheses derived from mechanistic evidence
17 are borne out by the experience of the human
18 population, and in this setting they appear not to
19 be borne out.

20 Q. Is it your opinion that biological
21 mechanism evidence with respect to environmental
22 exposures and latent disease is never sufficient of
23 itself to establish that exposure causes the
24 disease?

25 MR. LEGHORN: Object to the form.

1 A. That is not my opinion. It is my opinion
2 that when you have epidemiologic evidence as we have
3 in this case, you must consider it, and it provides
4 a test of whether your mechanistic hypothesis is or
5 is not correct.

6 Q. And it is your view that regardless of the
7 limitations on that epidemiology, to the extent that
8 epidemiology fails to itself show an association,
9 that it renders the mechanistic evidence incapable
10 of reaching the conclusion of causation. Isn't that
11 right?

12 MR. LEGHORN: Object to form.

13 A. That is not my opinion. My opinion is
14 that you have or I should say Dr. Smith has a
15 mechanistic hypothesis. The epidemiology provides a
16 real test of whether populations exposed to benzene
17 are at increased risk of APL; it does not support a
18 conclusion that they are.

19 Q. And the flip side is, Doctor, that you've
20 admitted that the epidemiological evidence also
21 cannot prove the null hypothesis. Correct?

22 A. That is correct, and that is a widely
23 accepted principle of scientific inference across
24 all branches of science.

25 Q. So the epidemiological evidence with

1 respect to benzene and APL in your opinion neither
2 proves that benzene does cause APL nor does it prove
3 that benzene does not cause APL. Correct?

4 MR. LEGHORN: Object to the form.

5 A. The issue is this. In science if you have
6 a hypothesis that some agent causes a disease, let
7 it be the hypothesis that benzene causes APL, the
8 burden of proof is on the person who forms the
9 hypothesis to show that their hypothesis is correct.
10 We largely endorse the approach written by Karl
11 Popper, the idea of refutation of hypothesis, that
12 what most scientists do is try to refute hypotheses
13 that have been proposed.

14 So we are in a setting where Dr. Smith has
15 hypothesized that low-level benzene exposure causes
16 APL, and it is appropriate that the epidemiology
17 evidence and other forms of scientific evidence be
18 examined carefully and systematically to determine
19 whether they provide proof of Dr. Smith's hypothesis
20 or whether they fail to provide it. In other words,
21 do they refute it or do they provide data that are
22 inconsistent with his hypothesis? The answer is
23 they provide data that fails to support Dr. Smith's
24 hypothesis.

25 What that means then is he has a

1 hypothesis that lacks proof.

2 Q. I am going to repeat my question which you
3 didn't answer. Isn't it your opinion that the
4 epidemiological evidence with respect to benzene
5 exposure and APL is neither sufficient to show that
6 benzene exposure can cause APL nor is it sufficient
7 to show that benzene exposure cannot cause APL?

8 MR. LEGHORN: Object to the form, asked
9 and answered.

10 A. As Dr. Smith testified, you cannot prove
11 the null hypothesis. That is widely agreed across
12 all areas of science. So your question has a
13 presumption in it that somehow I should be forced to
14 do something that we all agree can't be done. In
15 science we seek to prove alternative hypotheses such
16 as that benzene causes APL and, failing to do so,
17 which is the situation we're in, we are left with
18 the conclusion that there is not adequate evidence
19 to conclude that benzene causes APL, period.

20 Q. Based on the human epidemiology studies.
21 Right?

22 A. Based on a consideration of all the
23 evidence.

24 Q. Well, Doctor, if the human epidemiology
25 studies themselves can't prove the null hypothesis,

1 then how can they be affirmative evidence that
2 benzene exposure does not cause APL?

3 MR. LEGHORN: Object to form,
4 argumentative.

5 A. I've already answered that question.

6 Q. Is there any circumstance in which you
7 would find that biologic mechanism evidence itself
8 is sufficient to reach a conclusion scientifically
9 that more likely than not exposure to agent A causes
10 disease B?

11 MR. LEGHORN: Object to the form.

12 A. There are numerous instances where that
13 could be true. If you had a gunshot wound to the
14 head and you explored the anatomy of what had
15 happened as the bullet passed through the head, I
16 think you could easily invoke mechanistic evidence
17 that would say that was the cause of death; you
18 don't need epidemiology.

19 Q. I'll try it again. Is there any
20 circumstance in which you would find based on
21 mechanistic evidence in the absence of epidemiology
22 evidence that an exposure to an environmental agent
23 causes a particular disease following some latency
24 period?

25 MR. LEGHORN: Object to the form.

1 A. I would be reluctant to accept mechanistic
2 evidence as the sole basis for a conclusion that an
3 environmental agent caused a disease such as cancer
4 with an appreciable time lag between exposure and
5 disease and in the setting where there were other
6 known or unknown causes of that disease. That
7 approach, I believe, is entirely consistent with for
8 example the International Agency for Research on
9 Cancer, which uses a set of rules in which they are
10 almost invariably unwilling to reach a decision that
11 a chemical agent is known to cause human cancer in
12 the absence of sufficient epidemiologic evidence.
13 They consider mechanistic evidence as a far less
14 important form of support for reaching those
15 conclusions than do they consider the epidemiologic
16 evidence.

17 If I were to try to apply the logic and
18 rules that IARC uses to the setting of benzene and
19 APL, it would be quite clear that the epidemiologic
20 evidence is inadequate to support a conclusion that
21 benzene causes APL. Whether you have mechanistic
22 evidence or not, I do not think you could sustain a
23 conclusion that benzene is known to cause APL in
24 humans.

25 Q. In fact, IARC has a classification for

1 whether something is a known carcinogen and whether
2 something is a probable carcinogen as well.

3 Correct?

4 A. I believe they use the terms known,
5 probable, possible, and not classifiable. They also
6 have a fourth classification for known not to cause.
7 There's only one chemical that's ever been
8 classified in that last category.

9 Q. And IARC will classify agents as probably
10 carcinogenic to humans in circumstances when there
11 is limited evidence of carcinogenicity in humans and
12 sufficient evidence of carcinogenicity in
13 experimental animals. Correct?

14 A. I believe that's correct. And I am not
15 aware that we have sufficient evidence in animals
16 for benzene and APL and I believe we do not even
17 have limited evidence in humans. I doubt this would
18 be IARC class 2A. It would probably be IARC
19 category 3.

20 Q. IARC also classifies a carcinogen or
21 possible carcinogen as 2A as a probable carcinogen
22 when there is inadequate evidence of carcinogenicity
23 in humans, sufficient evidence of carcinogenicity in
24 animals, and strong evidence that carcinogenesis is
25 mediated by a mechanism that also operates in

1 humans. Is that right?

2 A. I believe that's one of the set of
3 criteria by which they will classify a chemical as a
4 2A or probable carcinogen. But I don't believe that
5 we've satisfied those conditions.

6 Q. Would you agree that the human
7 epidemiology studies that have examined benzene
8 exposure and acute nonlymphocytic leukemias as a
9 group have shown a strong association between
10 benzene and ANLL?

11 MR. LEGHORN: Object to the form.

12 A. The epidemiology studies that have
13 examined that have shown that there is a strong
14 association between benzene exposure at levels that
15 exceed 40 to 50 ppm-years in some studies, in some
16 more than 200 ppm-years and increased risk of ANLL.

17 Q. And that association in the studies
18 between benzene and ANLL has also been a consistent
19 association. Would you agree?

20 MR. LEGHORN: Object to the form.

21 A. I'm not sure what you mean by consistent
22 association.

23 Q. I mean it has been consistently found in a
24 number of different cohorts and a number of case-
25 control studies that there is an association between

1 ANLL as a group and benzene exposure.

2 MR. LEGHORN: Object to the form.

3 A. There is a body of well-done epidemiology
4 that I believe supports the conclusion that benzene
5 exposures greater than 40 to 50 ppm-years are
6 causally associated with increased risk of ANLL.

7 Q. And those studies show or many of them
8 show an increase in the risk with increased dose.
9 Correct?

10 MR. LEGHORN: Object to the form.

11 A. I would not characterize it as many. It's
12 actually a few have adequate exposure information to
13 be responsive to your question.

14 Q. And taken as a whole, the body of
15 literature, epidemiological literature looking at
16 benzene exposure and ANLL shows a coherent picture.
17 Would you agree?

18 MR. LEGHORN: Object to the form.

19 A. You would have to define what you mean by
20 coherent. The previous series of questions I think
21 were more consistent with the concept of replication
22 rather than coherence.

23 Q. How do you interpret coherence as it
24 relates to the set of considerations described by
25 Sir Austin Bradford-Hill?

1 A. Let's use Hill's own description of it.
2 He says "Coherence. On the other hand, the
3 cause-and-effect interpretation of our data should
4 not seriously conflict with the generally known
5 facts of the natural history and biology of the
6 disease." He goes on to say "Thus, in the
7 discussion of lung cancer, the committee finds its
8 association with cigarette smoking coherent with the
9 temporal rise that has taken place in the two
10 variables over the last generation and with the sex
11 difference in mortality, features that might well
12 apply in an occupational problem. The known
13 urban/rural ratio of lung cancer mortality does not
14 detract from coherence, nor the restriction of the
15 effect to the lung."

16 And so Hill is actually talking about the
17 overall pattern of the findings whereas I think your
18 previous questions were talking about replication of
19 findings. Clearly, with respect to benzene and APL,
20 there's no coherent picture. There's none
21 whatsoever. We don't have the characteristics such
22 as Hill just described for smoking and lung cancer
23 where you have time trends that lag the prevalence
24 of smoking, where you have a sex ratio that reflects
25 the difference in smoking patterns between men and

1 women, where you have an urban/rural difference. In
2 other words, that's a coherent picture. We don't
3 have that for benzene and APL.

4 Q. Do you have a coherent picture for benzene
5 and ANLL?

6 A. I think we do. I might mention on that,
7 one of the important elements in that coherent
8 picture is the evidence that only the exposures
9 within about ten to fifteen years of diagnosis
10 convey risk. So not only do we have evidence of
11 dose response where the risk is clearly increased at
12 exposure levels above 40 to 50 ppm-years, but we
13 also have evidence that distant past exposures don't
14 matter. And that is really a picture that has
15 become coherent in the past ten years.

16 MR. JENSEN: Counsel, I think that I have
17 less than thirty minutes of questioning left. I
18 don't know for sure, but that's what I think.

19 MR. LEGHORN: Do you want this on the
20 record?

21 MR. JENSEN: Either way. I don't care.
22 We're getting close to lunchtime. I think it should
23 be the doctor's decision primarily what we do in
24 terms of whether to take a lunch break or not. But
25 if we were to take a short bathroom break now, I

1 could organize my thoughts and probably finish
2 within thirty minutes. It's up to you.

3 THE WITNESS: I would prefer to finish
4 without taking a lunch break.

5 MR. JENSEN: All right. Let's take a very
6 short break now.

7 (In recess 11:43 a.m. to 11:53 a.m.)

8 BY MR. JENSEN:

9 Q. Dr. Garabrant, is it true that for
10 purposes of your opinion in this case, you have not
11 done any review of the literature relating to the
12 issue of the extent to which topoisomerase
13 inhibitors can and do cause APL?

14 A. No, actually I have looked at some of that
15 literature.

16 Q. But you are not offering any opinion on
17 that subject. Is that correct?

18 A. That's correct. It is my understanding
19 that Dr. Pyatt is addressing that issue.

20 Q. And you are not offering any opinion on
21 the subject of the extent to which benzene exposure
22 inhibits topoisomerase II. Is that correct?

23 MR. LEGHORN: Object to the form.

24 A. I have no intention of offering opinions
25 regarding the extent to which benzene inhibits

1 topoisomerase II. I have not been asked to do that.

2 Q. I have just put an exhibit sticker,
3 Exhibit 4, on a collection of binder-clipped
4 articles that were left for me at my hotel when I
5 arrived last night. And I would ask you to take a
6 quick look, Doctor, and see if you recognize those
7 articles.

8 A. (Pause) Yes.

9 Q. Did you recently identify those articles
10 as articles that you wished to rely on for purposes
11 of your opinions in this case?

12 A. I believe these are articles I assembled
13 since submitting my report on November 19, 2008,
14 although -- Let me restate that. These are articles
15 I have reviewed since submitting my report in
16 November of 2008, although some of them are already
17 cited in my report and I had reviewed them prior to
18 submitting my report in addition. I mean, I don't
19 know how to answer these things I recently
20 accumulated. Some of them I've had for years; some
21 of them I've cited in my report; some of them I
22 re-reviewed; some of them were new since submitting
23 my report.

24 Q. My understanding from counsel is that you
25 have recently been out of the country. Is that

1 correct?

2 A. Yes.

3 Q. And after you got back from your trip, you
4 identified the set of articles that have been marked
5 as Exhibit 4 as materials that you wished to rely on
6 to defense counsel in this case. Is that correct?

7 A. I'm not sure that's exactly correct.
8 These are articles for which I provided defense
9 counsel with copies just yesterday. Some of them
10 are cited in my report of November 19 and I assume
11 that everybody has had copies of them since November
12 or had copies prior to that. Some of them are
13 articles that I looked up and read in response to
14 Dr. Smith's supplemental declaration. So it's a
15 combination of factors. The common theme is that I
16 gave copies of these electronically to Mr. Leghorn
17 yesterday. So some of them are old, some are new,
18 some are in response to Dr. Smith's work, some are
19 ones that I had reread looking now at some new
20 aspect of them.

21 Q. Okay, thank you.

22 MR. JENSEN: And just for the record, to
23 the extent that the articles in Exhibit 4 include
24 materials that were previously referenced in
25 Dr. Garabrant's original report, we certainly don't

1 have any objection to his reliance on them. To the
2 extent that they're new, we do have an objection.
3 That can be resolved however it will be resolved.
4 Thank you.

5 BY MR. JENSEN:

6 Q. Dr. Garabrant, who compiles for you your
7 list of legal testimony as reflected by Exhibit 1?

8 A. I do.

9 Q. You do. How do you do it?

10 A. I try to keep track of all the times I
11 have been deposed or testified in court.

12 Q. And the reason I ask is because I'm aware
13 of at least one area or one instance of testimony
14 that doesn't seem to be reflected, which is trial
15 testimony you gave in the case of Turner versus
16 Chevron. You've got your deposition testimony
17 reflected but no trial testimony.

18 A. If I've omitted it, it isn't by intent.
19 That list, I believe, reflects that that was
20 prepared on November 18. I don't remember when I
21 testified in Turner, whether it was just before that
22 or just after that. It was sometime last fall, so
23 I may have missed it or it may have been brand-new;
24 I don't know.

25 Q. Does anyone besides yourself check the

1 accuracy of the list?

2 A. No.

3 Q. If you will turn with me to Dr. Cranor's
4 report of October 9 at page 10, there is a
5 subparagraph iii in the middle of page 10 and the
6 third sentence of that paragraph says "As both
7 medical and legal commentators put the point, 'In
8 the final analysis, assessment of evidence and
9 causal inferences depend on accumulating all
10 potentially relevant evidence and making a
11 subjective judgment about the strength of the
12 evidence'." Do you agree with that statement?

13 MR. LEGHORN: Object to the form.

14 A. I would have to go back and read reference
15 39, which is footnoted at the bottom Kassirer &
16 Cecil, Annals of Internal Medicine, before I would
17 comment on what Dr. Cranor is concluding there.

18 Q. Do you have an opinion as to whether the
19 following sentence after that sentence is true,
20 which says "When professional judgment is so central
21 to the drawing of inferences, professionals may
22 disagree at various stages of reasoning in an
23 inference to the best explanation, and thus may have
24 disagreements about their overall view of the
25 scientific conclusions"?

1 A. Well, I mean, I think what Dr. Cranor is
2 saying there is nothing more than people can
3 disagree on their view of what a body of evidence
4 says. So I don't think there's any content to that
5 statement. We know that people can disagree, and of
6 course what we try to do is to explore why they
7 disagree and try to uncover whether they have looked
8 at all of the evidence, whether they have considered
9 it properly, whether they have made their
10 calculations properly, whether they have followed
11 the rules of science that are widely accepted and so
12 on.

13 So I don't think there's much content to
14 Dr. Cranor's sentence. People can disagree.

15 Q. Outside the context of litigation,
16 scientists disagree with one another about
17 scientific propositions all the time. Is that true?

18 MR. LEGHORN: Object to the form.

19 A. The essence of the scientific method is
20 based on the idea that every scientist has a
21 responsibility to be skeptical of everyone else's
22 work and to try to refute it. That's what we do as
23 scientists, and that's not limited to medicine or
24 epidemiology or physics or biology. That's part of
25 the method. And when we can refute other people's

1 conclusions with evidence, then their conclusions
2 should not be trusted as reliable.

3 Q. Well, oftentimes scientists have competing
4 inferences and competing conclusions based on the
5 same evidence. Isn't that true?

6 MR. LEGHORN: Object to the form.

7 A. I would have to say no because of your use
8 of the word "often." Largely scientists live in a
9 world where we agree to the vast body of evidence
10 and disagree only around the fringes. Once in a
11 while that's not true. As Cranor points out,
12 Newton's laws of motion were widely accepted until
13 Einstein came along and showed that at very high
14 velocities, newtonian physics don't really -- aren't
15 supported by real evidence, by data or by theory.
16 But largely we live in a world where bridges don't
17 fall down; where when you turn your furnace on it
18 heats your house; where when you hit the brakes in
19 your car, it stops; where when you have a blood test
20 done, the results mean what we all agree they mean.
21 The methods of science have turned out to provide an
22 extraordinarily broad and reliable body of evidence
23 that has allowed mankind to move forward and to make
24 great progress in almost every realm of life.

25 And so when you say we often disagree, my

1 answer is no. Overwhelmingly we agree. It's only
2 around the fringes of discovery where the pattern of
3 evidence is unclear that we disagree. And that is
4 of course what is going on in this case. Is there
5 evidence that benzene causes APL? And the answer
6 is: not nearly enough to conclude that there is.
7 And as we've discussed, Dr. Smith has simply looked
8 at the epidemiology improperly and failed to
9 consider the epidemiology that is directly relevant
10 to his opinions and that is a direct test of his
11 hypothesis and doesn't support it.

12 MR. JENSEN: I will let you close with
13 your closing argument. That's all the questions
14 I have.

15 MR. LEGHORN: Thank you.

16 (Deposition concluded at 12:10 p.m.)
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1 COURT REPORTER'S CERTIFICATE

2 I, J. Edward Varallo, RMR, CRR, Registered
3 Professional Reporter and Notary Public in the
4 Commonwealth of Massachusetts (my commission expires
5 12/24/2015), hereby certify that the deposition of
6 David H. Garabrant, M.D., M.P.H. taken on February
7 18, 2009, in the matter of Brian K. Milward and
8 Linda J. Milward v. Acuity Specialty Products Group,
9 Inc., et al. was recorded by me stenographically and
10 transcribed; that before being sworn by me, the
11 deponent provided satisfactory evidence of
12 identification as required by Executive Order 455
13 (03-13) of the Governor.

14 I certify that the deposition transcript
15 produced by me is true and accurate to the best of
16 my ability.

17 I certify further that I am not counsel,
18 attorney, or relative of any party litigant, and
19 have no interest, financial or otherwise, in the
20 outcome of this suit.
21
22
23
24

25 DATED: 2/25/2009

J. Edward Varallo

1 WITNESS: David H. Garabrant, M.D., M.P.H.

2 DATE: February 18, 2009

3 CASE: Brian K. Milward and Linda J. Milward v.
4 Acuity Specialty Products Group, Inc.,
5 et al.

6
7
8 DISTRIBUTION TO COUNSEL The original signature
9 page/errata sheet was sent to Joseph J. Leghorn,
10 Esq., to obtain signature from the deponent. When
11 signed, please send original to Steve Baughman
12 Jensen, Esq., who will supply a copy of the signed
13 errata sheet to other counsel present at the
14 deposition.

15
16
17 WITNESS INSTRUCTIONS After reading the transcript
18 of your deposition, please note any change or
19 correction and the reason for it on the errata
20 sheet. DO NOT make any notations on the transcript
21 itself. Use additional sheets if necessary.

22
23
24 SIGN AND DATE THE ERRATA SHEET and return it, along
25 with the transcript, to your counsel.