

TRANSCRIPT OF PROCEEDINGS

IN THE CIRCUIT COURT
OF MARSHALL COUNTY, WEST VIRGINIA

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KATHLEEN LAVENDER, Individually, as
Executrix of the Estate of JOHN D.
LAVENDER, JR., Deceased, and as the
Next Friend of MARY BETH LAVENDER,
a Minor,

Plaintiff,

v.

Civil Action Number

93-C-226 K

MILES INC., an Indiana Corporation,
Individually and as Successor-in-
Interest of MOBAY CORPORATION; SHELL
OIL COMPANY, a Delaware Corporation;
BP EXPLORATION AND OIL INC. d/b/a BP
OIL COMPANY, an Ohio Corporation;
BP CHEMICALS, INC., an Ohio
Corporation; HERMAN R. RING, and
COLUMBIAN CHEMICALS COMPANY, a
Delaware Corporation,

Defendants.

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DEPOSITION OF DAVID H. GARABRANT

Washington, D. C.

Tuesday, March 14, 1995

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LAVENDER, JR., Deceased, and as the
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v. : Number
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Individually and as Successor-in- :
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OIL COMPANY, a Delaware Corporation: ■
BP EXPLORATION AND OIL, INC. d/b/a :
BP OIL COMPANY, an Ohio Corporation; :
BP CHEMICALS, INC., an Ohio Corpora- :
tion; HERMAN R. RING; and COLUMBIAN :
CHEMICALS COMPANY, a Delaware :
Corporation, -
Defendants. ■
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DEPOSITION OF DAVID H. GARABRANT

Washington, D. C.

Tuesday, March 14, 1995

Deposition of DAVID H. GARABRANT, called for
examination pursuant to notice of deposition, on
Tuesday, March 14, 1995, in Washington, D. C., at the
law offices of Spriggs and Hollingsworth, 1350 Eye
Street, N.W., Suite 900, at 9:08 a.m. before JULIE
BAKER, a Notary Public within and for the District of
Columbia, when were present on behalf of the
respective parties:

R. DEAN HARTLEY, ESQ.

Hartley & O'Brien

827 Main Street

Wheeling, West Virginia 26009

On behalf of Plaintiffs.

-- continued --

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1 APPEARANCES (CONTINUED):

2

3

JOE G. HOLLINGSWORTH, ESQ.

4

BARBARA A. MILNAMOW, ESQ.

5

Spriggs & Hollingsworth

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1350 Eye Street, N.W.

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Washington, D. C. 20005-2305

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On behalf of Defendant Miles Inc.

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1 P R O C E E D I N G S

2 Whereupon,

3 DAVID H. GARABRANT

4 was called as a witness and, having first been duly
5 sworn, was examined and testified as follows:

6 EXAMINATION

7 BY MR. HARTLEY:

8 Q Doctor, would you tell us your full name.

9 A David H. Garabrant.

10 Q And we met a little bit ago. My name is
11 Dean Hartley. We're here to discuss the John
12 Lavender case.

13 Can you tell me what you've reviewed for
14 this case?

15 A Yes. Actually, I prepared a list of what I
16 reviewed, which needs to have a couple of things
17 added to it.

18 Q What does it need to have added?

19 A It needs to have added this tabulation of
20 John Lavender's work assignments and this deposition
21 by Herman Ring and this deposition by James Taylor.

22 Q Anything else?

1 A No.

2 Q Is there a legend on here somewhere to
3 describe what MR II is? Do you know what MR II is?

4 A I believe it is one of the plants in the
5 Mobay facility in Atrium, West Virginia.

6 Q I figured that too, but do you know
7 specifically what plant it is?

8 A I do not know -- I can point to it on a
9 map. I do not know what is made in this plant.

10 Q How about in the polyol, p-o-l-y-o-l? Have
11 you considered this at all in your determination for
12 today?

13 A Yes, I have.

14 Q Can you tell me what the polyol,
15 p-o-l-y-o-l area of the plant is?

16 A I believe that's a facility that handles
17 polyols, which is a common term for polymers of
18 alcohols, also called glycol ethers or polyethers.

19 Q Do you have a map someplace of the plant?

20 A I do not have a copy of the map.

21 Q Do you know what building polyols are in?

22 A I could point to it on the map.

1 Q But sitting here, you have no independent
2 recollection of what building it's in; correct, other
3 than if you see a map it would refresh your
4 recollection?

5 A I could draw it.

6 Q Okay. Give me a drawing.

7 MR. HOLLINGSWORTH: Dean, while he's
8 drawing this diagram of the plant, he should
9 mention -- I think also he's probably neglected to
10 mention that he looked at the industrial hygiene data
11 as we referred to it so far in these depositions,
12 which, of course, would include the monitoring data
13 that we discussed most recently, I think, in
14 Dr. Rose's deposition.

15 MR. HARTLEY: Did he review the infamous
16 Carlos study?

17 MR. HOLLINGSWORTH: I believe that's on his
18 list.

19 THE WITNESS: Yes, I did, and I forgot to
20 add that. I apologize.

21 BY MR. HARTLEY:

22 Q Is the map on here? What is your

1 understanding of which way is north and which way is
2 south on your map? So you have nitrobenzene above
3 the polyols?

4 A North of the polyols.

5 Q Where's iron oxide, which is north of ..
6 okay.

7 And how did you rely on this work history
8 in any way to come to your opinions in this case,
9 Doctor?

10 A I relied on that to give me information
11 regarding the job assignments that Mr. Lavender had
12 while he was on the Mobay site.

13 Q Did you have corresponding industrial
14 hygiene data for each of these job sites?

15 A I reviewed industrial hygiene data for the
16 benzene samples that were taken at a number of
17 locations throughout the plant that relate to the
18 benzene exposures and many of those areas.

19 Q Did you find any industrial hygiene studies
20 specifically dealing with electricians, contract
21 electricians?

22 A I reviewed industrial hygiene data that

1 consisted of area and personal samples in many of the
2 plants -- or I should say in a number of the plants
3 at the Mobay site. Those samples would be relevant
4 to anyone working in those plant areas.

5 Q Let me see if I can rephrase the question
6 for you. Did you find any specific monitoring,
7 specifically of the electricians, contract
8 electricians?

9 A I do not recall if there were any personal
10 samples made on contract electricians. There were
11 personal samples made on Mobay employees and area
12 samples in a number of areas throughout the plant
13 which are relevant to the determination of exposure
14 of whoever is in the area.

15 Q Did you determine whether there were
16 adequate studies of the facility?

17 A I do not understand.

18 Q Did you determine whether there were
19 adequate studies done of benzene monitoring in the
20 MNB area to assess the average benzene exposure in
21 that area?

22 A I'm not sure what you mean by "adequate."

1 Q Quantitywise?

2 A I'm sorry?

3 Q Quantity, not quality. Were there enough
4 studies done to give you a basis to determine what
5 the average benzene exposure was for that area?

6 A I would say yes, there were many, many
7 samples taken. My impression, in thinking back over
8 the data, is yes.

9 Q What did you find to be the high exposure
10 in your review of the data?

11 A I don't -- you mean the highest exposure in
12 all the data?

13 Q Yes.

14 A I didn't make a mental note of the highest
15 exposure.

16 Q Did you make any notes at all of industrial
17 hygiene monitoring results?

18 A Do you mean written notes?

19 Q Yes, sir.

20 A Written notes, no, I did not.

21 Q Do you recall the lowest?

22 A I do not recall the lowest either. I

1 believe th t th **lowest** was below the limit o
2 detection and on some of those, I think that that was
3 in the range of .01 parts per million.

4 Q What was the limit of detection for
5 benzene?

6 A Using what method?

7 Q What methods were used at the Mobay plant?

8 A There were charcoal tubes, and there was an
9 HNU detector.

10 Q Are you familiar with charcoal tubes?

11 A I am somewhat familiar with charcoal tubes.

12 Q Are you familiar with what the lowest
13 amount of benzene a charcoal tube can detect?

14 A I do not know the literature on that. It
15 certainly has some dependence on the volume of air
16 that's drawn through the tube, and so I don't have a
17 single number. To some extent, it depends on how the
18 sample is taken as well as on the tube.

19 Q Do you have any recollection of whether a
20 charcoal tube can detect benzene out to 3 decimals,
21 decimal places, .001, .0012?

22 A What units?

1 Q I'm sorry?

2 A What units are you referring to?

3 Q Units of parts per million.

4 A I do not know -- I do not know. The lowest
5 numbers I recall seeing were .01 PPM. I didn't see
6 anything lower than that, so I don't know if they can
7 detect .003 or .009.

8 Q What did you find significant about the air
9 monitoring at Miles in light of Mr. Lavender's
10 disease process?

11 A I don't want to misinterpret your
12 question. What do you mean by "significant"?

13 Q You reviewed all the monitoring; correct?
14 Did you find anything significant that would apply to
15 this particular case?

16 A I found things that were relevant, if
17 that's the term.

18 Q Are you having a hard time with my word
19 "significant"?

20 A Yes.

21 Q What did you find relevant?

22 A Okay, thank you. There were many, many

1 samples that indicated that the exposure levels were
2 routinely in the range of .01 to a few tenths of a
3 part per million of benzene.

4 Q Did you find -- there were likewise samples
5 above that, were there not?

6 A There were occasional samples that were in
7 the few parts per million range.

8 Q Did you notice the particular study that
9 was done by the trench on the day when an upset
10 occurred in the MNB unit? Were you aware of that
11 study?

12 A I do not recall that specifically. I'd be
13 happy to look at that, if you want to direct me to
14 it.

15 Q Do you have the monitoring data with you?

16 A I do not.

17 Q Did you rely on the monitoring data?

18 A Yes.

19 MR. HOLLINGSWORTH: Do you want us to get
20 that?

21 MR. HARTLEY: See if I can do it this way
22 because there's no sense in wasting time.

1 MR. HOLLINGSWORTH: We can get it for you.

2 BY MR. HARTLEY:

3 Q Do you have a recollection that there were
4 studies done indicating when an upset occurred at the
5 MNB unit, the monitoring above the trench was between
6 5 and 50 parts per million of benzene?

7 A I do recall seeing some monitoring data
8 that was in that range. I don't recall the incident
9 around which the monitoring was done, but I do recall
10 that in that range.

11 Q Would you agree that is a high level of
12 benzene in the air?

13 A I don't know what you mean by "high."

14 Q Would you agree it's above the OSHA
15 standard?

16 A Yes.

17 Q Do you agree that level of benzene could
18 cause leukemia?

19 A No.

20 Q What level do you think, Doctor, is
21 necessary to cause leukemia?

22 A I think that the epidemiologic literature

1 supports the conclusion that cumulative exposures in
2 excess of 50 parts per million years is associated
3 with leukemia, although there's some range of
4 estimates. Some studies show no risk below 200 parts
5 per million years, so I'd say somewhere between the
6 range of 50 and 200 parts per million years.

7 Q Do you agree acute monocytic leukemia is
8 associated with benzene?

9 A Acute monocytic leukemia specifically?

10 Q Yes.

11 A The evidence showing that there's an
12 association for that specific type of leukemia is
13 relatively sparse.

14 Q Do you have an opinion -- have you
15 testified before -- that's related in the Bradley
16 case specifically -- that there's an association
17 between the two?

18 A For acute monocytic leukemia?

19 Q Yes.

20 A To my recollection, I don't believe I've
21 said that about that specific type of leukemia.

22 Q Well, let's look at it first so we can get

1 that out of the way. Do you remember Mr. Nace taking
2 your deposition?

3 A Yes.

4 Q I know you said it in here, Doctor, because
5 I remember you spelling it out for them. I don't
6 remember where, but bear with me for a second.

7 Have you seen Kenny Crump's reevaluation of
8 the Rinsky and Infante cohort that was published in
9 1994?

10 A I don't recall that.

11 Q Have you searched the literature before the
12 testimony today?

13 A I did.

14 Q You didn't come across that new update?

15 A I seem to have missed it. Could you give
16 me a citation on it?

17 Q Could I? Sure could. I think it's in Risk
18 Analysis -- sorry. Journal of Toxicology and
19 Environmental Health, line 42, page 219. You haven't
20 seen that before today?

21 A I do not recall that article.

22 Q Do you think you'll have time to read

1 Rinsky and Infante's cohort before trial?

2 A I would certainly like to.

3 Q When did you last search the literature?

4 A Last week.

5 Q Did you come across that?

6 A Missed it.

7 Q Let me show you, Doctor, pages 118 and 119
a of your deposition in the -- titled Mary Montgomery
9 versus Tricontinental Industries, et al.,
10 specifically to the pages that I have marked down at
11 the bottom.

12 A Yes.

13 Q In that deposition, did you say there was
14 an association between benzene and acute myelogenous
15 leukemia?

16 A Yes, I did.

17 Q Did you include within the acute
18 myelogenous leukemia acute monocytic leukemia?

19 A It is one of the subtypes that is included
20 in the broader heading acute myelogenous leukemia.

21 Q During this deposition in May of 1994, did
22 you say that acute monocytic leukemia was related to

1 benzene or associated with benzene?

2 A I don't think that is what I said.

3 Q What do you think you said in that
4 particular instance?

5 A I think I said that the broader heading,
6 acute monocytic leukemia, is associated with
7 benzene. I did not say that there was adequate
8 evidence for each of those specific subtypes to
9 establish that they're associated with benzene.

10 Q The question was asked "is it your opinion
11 that there is sufficient evidence to show that
12 benzene does cause some leukemias?

13 "Answer: I've answered that and the answer
14 is yes."

15 That was your answer.

16 "Question: Which leukemia?

17 "Answer: Acute myelogenous.

18 "Question: Anything else?

19 "Answer: That families of leukemia, which
20 means the leukemias that are derived from the
21 monocytic series of cells.

22 "Question: Name them."

1 You go on to name them and include
2 monocytic. Are you telling me that doesn't mean that
3 monocytic leukemia is related to benzene or
4 associated with benzene?

5 A What I'm telling you is that the scientific
6 evidence is adequate to establish the association for
7 the family. It is not adequate to establish the
8 association for each specific member of the family.

9 Q Did you say that here?

10 A Did I say what there?

11 Q Just what you told me. The question was
12 asked "which means what?" And you said "the
13 leukemias that derive from the myelocytic series of
14 cells."

15 You didn't say anywhere in this -- and you
16 can look at it again if you'd like -- that there is
17 not adequate association between each of these
18 individual types under the AML category.

19 Are you saying that's what your testimony
20 is today?

21 A What I'm saying today is I consider the
22 scientific evidence adequate to establish a causal

1 association between benzene and acute myelogenous
2 leukemia under circumstances of exposure that exceed
3 20 -- I should say 50 to 200 parts per million years
4 assuming adequate latency is met. I am not saying
5 that the scientific data are adequate to establish a
6 causal association for each of the specific subtypes
7 of leukemia that are categorized under the heading
8 acute myelogenous leukemia.

9 Q That's what you're saying today?

10 A That is what I'm saying today, and I
11 believe that's consistent with what I told Mr. Nace.

12 Q And you haven't reviewed Kenny Crump's
13 article in '94 which discusses specifically acute
14 monocytic leukemia?

15 A To my recollection, I have not seen that
16 article. If you might let me look at it, I might
17 recall having seen it.

18 Q Do you have a list of all the articles you
19 have reviewed?

20 A I do not have a list. I have brought them
21 with me.

22 Q Do you want to take a look through your

1 binders to see if it's in there?

2 A I have.

3 Q It's not in there?

4 A No.

5 Q Would it be fair to say you've included
6 every article you've read for today's deposition in
7 these binders?

8 A Yes.

9 Q Does individual susceptibility have any
10 part in the development of leukemia as it relates to
11 benzene?

12 A I'm not sure what you mean by "individual
13 susceptibility."

14 Q What does that term generally mean to you?
15 Some people are more susceptible than others? Some
16 people may have a development of a disease process,
17 regardless of what the toxin is, at lower levels than
18 the person next to him who may have a disease process
19 or no disease process at a higher level? Do you
20 understand individual susceptibility?

21 A Let me try to rephrase what I think you're
22 saying, that people's susceptibility to the

1 leukemogenic effect of benzene varies such that some
2 people are more susceptible to that than others. Is
3 that what you mean?

4 Q I'm asking you if .. what is your .. let me
5 do it this way.

6 What is your understanding of "individual
7 susceptibility," if you understand the term, and if
8 you don't understand the term, we'll move on.

9 A Well, it's a vague term.

10 Q Is it in the literature?

11 A I don't know if it's in the literature or
12 not.

13 Q Is it in the benzene literature?

14 A I'm not aware of it in the benzene
15 literature.

16 Q Do you think you'll be looking at the
17 benzene literature between now and trial to see if
18 that term is in there, because I'm representing to
19 you that it is.

20 A Put it this way. In the epidemiologic
21 literature, I'm not aware that there is any support
22 for the notion that individual susceptibility to

1 leukemia explains risk.

2 Q I understand that. I understand that
3 completely. But what is your understanding of the
4 term "individual susceptibility" since you've now
5 used it in your own answer.

6 A Well, again, I'm not sure I understand what
7 you mean by it. The question I asked you was whether
8 you meant that individuals varied in their
9 susceptibility to leukemia in terms of the dose of
10 benzene that would cause that. That's what -- I
11 don't know what you mean.

12 Q Is that your definition of individual
13 susceptibility, what you just said?

14 A I don't have a definition of it in this
15 setting because I'm not sure what you're asking me.
16 There are myriad definitions of individual
17 susceptibility, and I'm not sure what it is you're
18 trying to ask me.

19 Q Have you ever used the term "individual
20 susceptibility" before?

21 A I don't recall whether I've used that term
22 or not.

1 Q Have you used that term in the TDI cases?

2 A Have I used that term in the TDI --

3 Q You've done TDI cases in the past, haven't
4 you?

5 A What do you mean "done TDI cases"?

6 Q You've testified as an expert in the TDI
7 cases; correct?

8 A I don't recall testifying in a TDI case.
9 Perhaps you could refresh my memory.

10 Q We'll do it at a later time. How about in
11 an MDI case? Have you testified in an MDI case?

12 A I don't recall testifying in an MDI case.

13 Q Have you testified in any isocyanide case?

14 A Not to my recollection.

15 Q Have you reviewed TDI cases for litigation
16 purposes?

17 A I believe I have.

18 Q And you didn't testify in those cases?

19 A Not to my recollection.

20 Q What about the MDI cases?

21 A What about them?

22 Q You did not testify -- you reviewed those

1 cases for litigation purposes?

2 A I don't recall whether I've ever reviewed
3 an MDI case for litigation purposes or not. I've
4 seen such cases in clinic. And I've reviewed their
5 records. I don't remember if I've done that in
6 support of litigation or not.

7 Q Isocyanide cases, have you reviewed any
8 isocyanide cases generally for litigation purposes?

9 A I think I just said I've reviewed TDI
10 cases, and TDI is one of the isocyanides, so the
11 answer is yes, I think I've reviewed records on TDI
12 for litigation purposes.

13 Q Did you prepare reports in those cases?

14 A I do not recall whether I did or didn't. I
15 know I've seen lots of them in clinic, and have
16 prepared clinical summaries. I don't remember if
17 I've prepared documents in addition to that. I've
18 certainly done clinical summaries.

19 Q How does benzene damage the bone marrow, do
20 you know? Let me ask you this first. Is benzene
21 known to be a myelotoxic agent?

22 A Benzene is known to damage the bone marrow

under circumstances of very high exposure.

2 Q And how does that occur?

3 A Do you mean what are the cellular
4 mechanisms and biochemical mechanisms?

5 Q Yes.

6 A I don't know.

7 Q What is your understanding, other than the
8 fact that at high levels it causes bone marrow
9 problems? Do you know anything else about how
10 benzene affects the bone marrow?

11 A I know at high exposure levels, it can
13 it can cause aplastic anemia.

1 Q Yes.

2 A Actually, I've never studied that. I don't
3 know.

4 Q Will that be something you'll look at
5 before trial, too?

6 A Well, you've intrigued me. I'll probably
7 take a look at that.

8 Q Is benzene a mutagenic agent?

9 A I'm sorry. I didn't quite understand the
10 question. What agent?

11 Q Mutagenic. I can never pronounce the
12 word.

13 A My understanding of that area, of the
14 scientific literature is it's been very difficult for
15 laboratory scientists to demonstrate that benzene is
16 mutagenic. And I remember it took a long time to
17 develop an appropriate animal model or model in
18 animal cell lines to demonstrate that.

19 I do not actually know the details of the
20 circumstances under which mutagenicity of benzene has
21 and has not been demonstrated.

22 Q Are you making a note to yourself, Doctor?

1 A I am.

2 Q Do you intend to review that subject before
3 trial?

4 A Briefly.

5 Q How about chromosome damage? Does benzene
6 cause chromosome damage?

7 A I'd have to say again, that's an area where
8 I don't know the literature in great detail. My
9 recollection is that there have been reports of
10 chromosome changes related to benzene, but again, I
11 did not review the molecular and genetic literature
12 on the effects of DNA in preparation for this
13 deposition.

14 Q That doesn't come into your equation as to
15 whether there's a causal relationship between
16 Mr. Lavender's exposure to benzene at the Miles
17 facility and his subsequent development of acute
18 monocytic leukemia?

19 A The answer is it plays a fairly modest
20 role. I'm certainly interested in whether there is
21 biologic plausibility for the association between
22 benzene and leukemia, and consideration of biological

1 plausibility does play a role in my assessment of
2 whether that association is causal.

3 The actual mechanisms by which benzene
4 might cause leukemia in humans and in other animal
5 species are fairly detailed, and I did not review
6 them in depth -- I did not review them in preparation
7 for this deposition.

8 Q Have you ever reviewed them?

9 A I've read some of that literature, yes,
10 over the years.

11 Q How long ago -- when was the last time you
12 read that literature?

13 A I don't recall the last time I read any of
14 that literature. I know I've seen that literature on
15 any number of occasions over the past 15 years.

16 Q Did you review that literature for the
17 Bradley deposition, the Mary Montgomery deposition by
18 Mr. Nace?

19 A The literature on genetic damage and
20 mechanisms of carcinogenicity of benzene?

21 Q That's correct.

22 A No.

1 Q Have you ever testified in a specific pure
2 benzene case prior to Bradley?

3 MR. HOLLINGSWORTH: Objection. Bradley
4 wasn't a pure benzene case.

5 MR. HARTLEY: I understand that. I'm not
6 implying that it was. I'm looking to see if he
7 testified in a pure benzene case --

8 MR. HOLLINGSWORTH: Even though your
9 question implied that it was.

10 MR. HARTLEY: I apologize that it was.

11 THE WITNESS: I don't believe I have.

12 BY MR. HARTLEY:

13 Q You have never testified in a pure benzene
14 case before this one; correct?

15 MR. HOLLINGSWORTH: Same objection.

16 THE WITNESS: To my knowledge, I've never
17 testified in a pure benzene case.

18 BY MR. HARTLEY:

19 Q Is this a pure benzene case? Benzene is
20 not part of a solvent. It's not part of a gasoline.
21 It is basically released into the atmosphere by the
22 process of making MNB. Would you agree this is a

1 pure benzene case?

2 MR. HOLLINGSWORTH: Objection.

3 THE WITNESS: I'm not sure whether this is
4 a pure benzene case or not. I've been asked to
5 review the epidemiologic literature on benzene and
6 leukemia. I'm not sure I know the breadth of the
7 issues in the case.

8 BY MR. HARTLEY:

9 Q Other than the Bradley gasoline case, can
10 you tell me what other cases you've testified in that
11 concern benzene in any way?

12 A To my recollection, none.

13 Q Have you testified in any cases concerning
14 benzene in solvents?

15 A To my recollection, no.

16 Q Was Bradley the very first case that you've
17 testified in concerning benzene exposure and the
18 development of leukemia?

19 A Yes.

20 Q Was Bradley the first case that you
21 testified in concerning benzene exposure and any
22 hemopoietic cancer?

1 A I believe so.

2 Q Was Bradley the first case that you
3 testified in concerning benzene and any type of blood
4 disorder?

5 A I believe so.

6 Q Have you written anything on benzene?

7 A I'd have to look at my CV.

8 Q Do you have it with you?

9 A I do.

10 Q Take a look at it and see.

11 A Yes.

12 Q What was that?

13 A I coauthored a report on electric and
14 magnetic field exposures, chemical exposures and
15 chemical risks in electrical occupations.

16 Q Which one is that?

17 A Number 49, to the Electric Power Research
18 Institute. That study included some discussion of
19 benzene, I believe.

20 Q Excuse me, Doctor. Just a second. When
21 was that published?

22 A That was published in 1992.

1 Q What page are you on?

2 A I'm on page 12.

3 Q I don't have that on this. What's the date
4 of your CV?

5 A November '94.

6 Q I've got July of '94. That's what I was
7 provided.

8 A Can I assist you?

9 Q Sure.

10 A 43.

11 Q 43 is now 49?

12 A Yes. I also authored an article looking at
13 mortality in shoe and leather workers, cancer
14 mortality in shoe and leather workers in
15 Massachusetts.

16 Q What number was that?

17 A It's 5 on my version. I think it's
18 probably 5 on yours. Those numbers don't change.

19 Q Yes. 1984?

20 A Yes. That's certainly relevant to the
21 topic of benzene. I can't recall specifically what
22 it says about benzene in that it's been 11 years.

1 Q And the article or the study that you did
2 for the Electric Power Research Institute in '92,
3 that was not peer reviewed, was it?

4 A That version of it was published by EPRI
5 with an internal review. We actually did publish a
6 peer reviewed paper which came out in 1994, which is
7 number 37 on your copy of my CV, and that derived
8 from that same report.

9 Q When I compare the 37 on my CV and the 43,
10 "chemical exposures" are missing from the peer
11 review study. Can you explain that from the title
12 anyway?

13 A Well, I don't know why we chose to edit the
14 title, other than to make it shorter. The fact is
15 that the study was designed to look at chemical
16 exposures among people who held jobs that had
17 electromagnetic field exposure to determine if those
18 chemical exposures confounded the relationship
19 between EMF and leukemia.

20 Q Did you come to any conclusions?

21 A Yes.

22 Q Which were?

1 A That the chemical exposures did not
2 confound that relationship.

3 Q Based upon what?

4 A Based upon an assessment of which
5 occupations had chemical exposures and analyses that
6 showed that they were not -- that they didn't
7 appreciably confound the relationship.

8 Q Was it because the individuals who
9 allegedly developed leukemia from electromagnetic
10 fields were not exposed to chemicals that could cause
11 or could be associated with leukemia?

12 A My recollection is that the prevalence of
13 exposure was low and that it didn't act as a
14 confounder, and so there was not founding.

15 Q The chemical exposure?

16 A The chemical exposures did not act as
17 confounders, that's correct.

18 Q Do you have a copy of that study, Doctor,
19 that I can get from you, since it's not published in
20 the open literature?

21 A The EPRI version of it?

22 Q Yes.

1 A Yes, I do.

2 Q Do you happen to have it with you?

3 A No, I do not.

4 Q Can I get a copy of that from you?

5 A Yes, I'll have to Xerox it. It's a big
6 thing.

7 Q That's okay. You can bill me for it.

8 A That can also be purchased directly from
9 EPRI. You can phone him up --

10 Q In Palo Alto?

11 A Sure. It's one of their publications. You
12 can call them up.

13 Q I'll do that. Don't worry about it. What
14 do you have on your list so far --

15 A Of things to do?

16 Q Yes.

17 A I need to get the Crump article from the
18 Journal of Toxicology and Environmental Health,
19 1994. I need to look and see whether benzene causes
20 leukopenia in the absence of pancytopenia, and I need
21 to review if benzene is mutagenic.

22 Q What specific chromosome, if any, does

1 benzene affect?

2 A I don't know.

3 Q Does benzene cause -- have you looked at
4 the literature at all with regard to benzene in
5 chromosomes?

6 A Not in a systematic matter, no.

7 Q Have you done any of that review for
8 today's deposition, systematic or nonsystematic?

9 A No, I haven't.

10 Q You're not aware of the new studies out
11 discussing chromosome damage from benzene that were
12 published in '94 and '95?

13 A I'd have to say I don't believe I have.

14 Q Are you going to rely on any of the
15 chromosome damage in this particular case to express
16 an opinion that benzene did or did not cause or is
17 associated with Mr. Lavender's AML?

18 A No.

19 Q Do you have any understanding at all of the
20 significance of chromosome damage from benzene in
21 exposed workers? I'll change "significant" to
22 "relevant" for you.

1 A I'd have to say that's not an area that
2 falls within my -- that part of the literature
3 doesn't fall within my area of expertise. I know I
4 have seen some articles on that that discuss
5 chromosomal changes in the various types of leukemia,
6 but I would have to say I have very little knowledge
7 of that area.

8 Q So you don't have an appreciation of the
9 true significance or relevance of the literature
10 concerning chromosome damage as it relates to benzene
11 exposure, do you? You know it's out there, but you
12 don't really know what the significance of it is in a
13 cause-and-effect relationship? Go ahead. I'm
14 listening.

15 A My knowledge of that area is limited, and I
16 did not review that in preparation for this
17 deposition.

18 Q Do you think an understanding of that area
19 would help you determine whether there is a causal
20 relationship between Mr. Lavender's benzene exposure
21 and his development of acute monocytic leukemia?

22 A No.

1 Q And why is that?

2 A Because Mr. Lavender doesn't satisfy the
3 criteria that are necessary to think that benzene was
4 a risk factor in his leukemia at all.

5 Q But then again, you don't know what the
6 literature says on chromosomes, so you can't totally
7 exclude that, can you, from your equation?

8 A I think I can based on the strength of the
9 epidemiologic literature.

10 Q So because of the strength of the
11 epidemiological literature, you don't need to know
12 about the chromosome damage; correct?

13 MR. HOLLINGSWORTH: I object to the
14 question on the ground that it proceeds from an
15 inference that there is chromosome damage, evidence
16 unavailable in this case at all.

17 MR. HARTLEY: Sure. Your own doctor said
18 there was evidence of chromosome damage. He didn't
19 think it was related to benzene, but he didn't know
20 the literature on benzene and chromosomes either.

21 MR. HOLLINGSWORTH: He knew it better than
22 anybody on this planet, but he didn't say there was

1 damage. He said there was a translocation.

2 BY MR. HARTLEY:

3 Q With all that said, Doctor.

4 A Would you repeat the question.

5 Q Basically your opinion is based on the
6 epidemiological study and the lack of association
7 between Mr. Lavender's exposure and his development
8 of acute monocytic leukemia; correct?

9 A My opinion is based on knowledge of the
10 scientific literature that strongly supports the
11 conclusion that he was not adequately exposed to
12 place him at risk of leukemia.

13 Q And it is not that specific basis that
14 permits you to exclude a review of the literature
15 which discusses chromosome damage in your analysis?

16 A That is one of the reasons, and it is the
17 major reason, yes.

18 Q What are the other reasons? One, that you
19 don't understand it, the chromosome area in the
20 literature?

21 A No. I don't think that would be one of my
22 reasons.

1 Q You do or don't think it would be?

2 A I don't think that would be. The
3 exploration of a mechanism by which benzene might
4 have caused Mr. Lavender's leukemia is conditional on
5 his having had adequate exposure to think that
6 benzene caused it.

7 If he wasn't adequately exposed, there's no
8 reason to review literature on the mechanisms by
9 which benzene causes leukemia at very high exposure
10 levels or prolonged exposure.

11 Q But you don't know whether the literature
12 discusses high exposure levels or low exposure levels
13 when they're discussing chromosomes, do you, because
14 you haven't reviewed that literature?

15 A I think I know enough about that literature
16 to say that there is not literature that would relate
17 to exposures in the less than 1 part per million
18 range in demonstrating mechanisms by which leukemia
19 would cause benzene.

20 Q Benzene would cause leukemia?

21 A Excuse me, benzene would cause leukemia.

22 Q You don't understand how benzene causes

1 leukemia; correct? All you know is that it causes
2 bone marrow damage and from that springs leukemia
3 with the appropriate exposure and appropriate latency
4 and period of time; correct?

5 A I do not know all of the mechanistic steps
6 by which benzene causes leukemia, and it's my
7 impression that those steps are largely unknown.

8 Q Have you reviewed the literature on the
9 mechanistic steps to leukemia from benzene exposure?

10 A I have not reviewed that systematically. I
11 have read that over the years, keeping up with
12 progress in that area.

13 Q Do you think you'll review that issue
14 before we get to trial on the 15th of May?

15 A I will probably look at review articles to
16 see what the current state of knowledge is. I will
17 probably not go back to the original literature on
18 that topic.

19 Q You're writing that one down, too?

20 A Yes.

21 Q Okay. How old are you, Doctor?

22 A How old am I?

1 Q Yes.

2 A 44.

3 Q You look younger than I do. I'm only 39.

4 A Thank you.

5 Q It was a compliment.

6 A And I took it in that spirit.

7 Q Will you be looking at the studies that
8 discuss low level exposure to benzene and chromosome
9 damage before trial?

10 A Probably not.

11 Q If I would tell you that there are articles
12 out there which discuss exposure to less than 3 parts
13 per million causing chromosome damage, would you find
14 that in any way relevant to the equation of
15 Mr. Lavender's acute monocytic leukemia?

16 MR. HOLLINGSWORTH: I object to that.

17 MR. HARTLEY: Assume for purposes that
18 there are.

19 MR. HOLLINGSWORTH: Are you talking about
20 animal studies?

21 MR. HARTLEY: Yes.

22 THE WITNESS: I would find that of --

1 BY MR. HARTLEY:

2 Q Let me do it this way. You've reviewed
3 other animal studies in other situations, haven't
4 you?

5 A I'm not sure what you mean by "other
6 situations."

7 Q In attempting to determine a causal
8 association, you have reviewed animal studies and
9 have testified that animal studies do provide some
10 evidence of a causal association, have you not?

11 A I don't recall to what I've testified.
12 However, it is my opinion that animal studies are
13 relevant to the determination of carcinogenicity to
14 humans, so they certainly can be relevant.

15 Q Do you think that there would be any
16 relevance in animal studies which demonstrate
17 chromosomal changes at below 3 parts per million?

18 MR. HOLLINGSWORTH: What kind of chromosome
19 damage?

20 MR. HARTLEY: He doesn't understand the
21 chromosome changes anyway, so it doesn't really
22 matter. Generally speaking, chromosome damage.

1 MR. HOLLINGSWORTH: obje t.

2 MR. HARTLEY: That's okay.

3 BY MR. HARTLEY:

4 Q That means you can answer.

5 A I think they are peripheral to the issue of
6 whether benzene causes leukemia in that the human
7 epidemiologic literature quite clearly does not show
8 evidence of risk at levels of exposure that low.

9 Q That's because the studies don't have the
10 power to do that, do they?

11 A No, I would not agree with that answer.

12 Q The studies are smaller studies, aren't
13 they, that do show a relationship?

14 A I don't understand that question.

15 Q Number of people in the cohort.

16 A I still don't understand the question.

17 Q What studies have you reviewed for this
18 particular case? Would you itemize them for me, the
19 epidemiological studies.

20 A Well, I'd have to go through my books. Do
21 you want everything, or do you want the important
22 studies?

1 Q I'd like to have the pertinent ones, the
2 ones that you're basing your opinion on specifically
3 that there needs to be 50 parts per million years
4 with an adequate latency period.

5 A Okay.

6 Q Can you do that for me.

7 A Yes. I would include the study by Wong in
8 1967 of chemical workers, a study by Ott of chemical
9 workers published in 1978. I would include the two
10 studies by Paxton in 1994.

11 Q What did she study?

12 A Pliofilm workers.

13 Q She reevaluated the Rinsky and Infante
14 study. Any other ones?

15 A Yes. I would also include the studies by
16 Devine published in '85 and '87.

17 Q Of what cohort?

18 A Gasoline workers. I would include --

19 Q Gasoline workers?

20 A Yes, or I should say refinery workers
21 exposed to gasoline. I would include the studies by
22 Rushton, also of refinery workers and petroleum

1 distribution workers.

2 Q What year was that?

3 A That was 1993.

4 Q Was that Rushton by himself or Rushton and
5 Alderson?

6 A It's a she, I think, and I think it's with
7 Alderson. I'd have to look, but I believe it's both.

8 Q Is that the study of the eight refineries
9 in Great Britain?

10 A Yes, it's in Great Britain. I would
11 include Wong in 1995, another reanalysis of the
12 pliofilm workers. And those are the major ones.

13 I'm sure if we start to talk about some of
14 the issues in detail, I may use other literature in
15 addition but those are the principal studies upon
16 which my answer rests.

17 Q You did not include the Bond update of the
18 Ott study. Do you intend to utilize that?

19 A Yes, I should use that as well. Thank
20 you.

21 Actually, let me add one other. There's a
22 new update by Delzel of the Shell cohort.

1 Q That was Thorpe's study earlier?

2 A I'm sorry?

3 Q Not Shell. That was Exxon. Go ahead.

4 A That's 1995.

5 Q Where is that published?

6 A JOM.

7 Q M, you did say M?

8 A JOM, yes -- excuse me, JOEM, Journal of
9 Occupational Environmental Medicine.

10 Q Doctor, how is benzene removed from the
11 body?

12 A I believe it's metabolized to phenol which
13 is excreted by the kidneys.

14 Q Do you know what the half-life is?

15 A It's fairly short. I don't have a number,
16 but it's in the matter of days, at least.

17 Q Is that the only way for it to be removed
18 from the body?

19 A Oh, I think you could exhale a small
20 proportion of it unchanged. There are probably other
21 metabolic pathways that are less important than
22 phenol. I don't recall them offhand.

1 Q Can the worker exposed to benzene eliminate
2 all of the benzene that he is exposed to over a
3 workweek?

4 A I would have to look that up. It certainly
5 would eliminate a very large proportion of it.

6 Q Would some still remain in the bone marrow
7 by the end of the workweek?

8 A I do not know the literature on that
9 point. My understanding of the kinetics of excretion
10 of benzene are that a very large proportion of it
11 would be eliminated within a few days, which would
12 imply that a very small proportion would be
13 retained. Since it's a lipophilic solvent, I would
14 expect it would be retained in fatty tissues of the
15 body.

16 Q Do you have an opinion as to whether all
17 the benzene is ever removed if the individual worker
18 continues to be exposed? Do you understand the
19 question?

20 A No.

21 Q In other words, is there a cumulative -- is
22 there an accumulation of the benzene in the marrow

1 over the course of the workweek or work-month or
2 work-year?

3 A The answer is no, there's not an
4 accumulation. It would be true that under a steady
5 state exposure, you would reach equilibrium such that
6 the absorbed amount and the amount eliminated by
7 excretion, both of the parent compound and the
8 metabolites, would be in balance, but I don't think
9 that it actually accumulates in any sense.

10 Q At what level does the bone marrow become
11 damaged?

12 A I'm not sure what you're referring when you
13 say "level." Are you talking about air level, blood
14 level, cell level, fat level?

15 Q At what particular air level of benzene
16 does the bone marrow become damaged?

17 A I don't think we know a specific number.
18 The literature that addresses that issue comes
19 largely from circumstances of very high uncontrolled
20 exposure, such as in the shoe industry in Turkey, the
21 reports by Aksoy and some from Italy. It's clear
22 from the descriptions in those reports and the small

1 amount of measurement of exposure, that the levels
2 are well up in the many hundreds of parts per
3 million.

4 Q But you have not reviewed the literature
5 concerning lower level exposure and damage to bone
6 marrow, have you? Not the epidemiological
7 literature, but the other scientific literature.

8 MR. HOLLINGSWORTH: I object to that
9 question. You're referring to animal data again or
10 epidemiologic data?

11 MR. HARTLEY: Any data. I'm not referring
12 specifically to epidemiologic data on that.

13 THE WITNESS: The human literature, I do
14 know some of that. So the answer is yes, I do know
15 some of that literature.

16 BY MR. HARTLEY:

17 Q Specifically what literature would you rely
18 on concerning that specific point?

19 A I couldn't cite specific articles offhand.
20 I know there have been a number of studies of
21 hematologic changes among workers exposed to low
22 levels of benzene in industry that do not show any

1 affects on cell counts or bone marrow function, and
2 so I can't cite the articles offhand. I could
3 certainly pull those together for you.

4 Q Did you review them before today?

5 A I have reviewed that topic in the past. I
6 did not specifically prepare that topic for today,
7 but I'm generally familiar with the area.

8 Q I notice you didn't indicate the Wong '83
9 study. Did you review the Wong '83 study?

10 A I do not know offhand which study that is.
11 What's the citation on that?

12 Q It was a study done for CMA.

13 A I don't believe I know that study.

14 Q Have you ever seen that study cited in the
15 literature when you were doing the --

16 A Without having a citation to know exactly
17 which study we're talking about, it's hard for me to
18 know if I've seen that cited.

19 Q I'll see if I can get you a cite. It's not
20 in the open literature. I think it's published by
21 his group. Here it is. "Comments on the NIOSH study
22 of leukemia in benzene workers technical report"

1 submitted to Gulf Canada Limited by Environmental
2 Health Associates, August '83.

3 A I do not know that particular document.

4 Q So you don't know what Wong said in that
5 particular study, do you?

6 A No, I do not.

7 Q Have you reviewed Dr. Wong's testimony
a before OSHA?

9 A On what occasion?

10 Q Have you reviewed any of Dr. Wong's
11 testimony before OSHA?

12 A I do not recall offhand. I know I've read
13 a lot of things he's said. I don't recall offhand if
14 I've read testimony before OSHA.

15 Q March 4, 1986 was the date of the
16 submission.

17 A Was it published in the Federal Register?

18 Q You know, I don't know whether it was or
19 not. I know it's on the OSHA docket.

20 A I probably did not read it if it did not
21 come out in the Federal Register.

22 Q Doctor, do you agree that cytotoxicity is

1 associated with the development of cancer?

2 A Sometimes. Sometimes not.

3 Q When would it not be?

4 A There are many instances. We could kill
5 cells by making them hypoxic and that's cytotoxicity,
6 and that's not associated with the development of
7 cancer.

8 Q Anything else?

9 A We could kill them with cyanide. That's
10 not associated with cancer. We could physically burn
11 them. We could disrupt them osmotically. We could
12 come up with a long list of agents and circumstances
13 that were cytotoxic that had nothing to do with
14 cancer.

15 Q Could we damage them through benzene
16 cytotoxically?

17 A I suppose you could manage that in the
18 laboratory. I don't know that you can do that in
19 living people, if we're talking about people. Which
20 species are we talking about?

21 Q We'll talk about people right now. Does
22 benzene cause cytotoxic damage, or is benzene a

1 cytotoxic agent?

2 A In living, intact human beings? We're not
3 talking human cell lines in a lab. I would have to
4 say it can be cytotoxic to some of the cellular
5 elements in the bone marrow under circumstances of
6 extremely high exposure.

7 Q What do you base that on?

8 A What do I base that on?

9 Q Yes. What's the basis for your statement?

10 A The literature that shows that people who
11 have extremely high levels of exposure can develop
12 pancytopenia and anemia in response to benzene
13 exposure.

14 Q And what literature would that be, the one
15 that we've talked about here earlier?

16 A It would include the earlier literature by
17 Aksoy and Vigliani. There's more. I can't recall
18 names of articles offhand.

19 Q Since you've mentioned Aksoy's studies, do
20 you recall Aksoy's study of leukemia in a family of
21 shoemakers?

22 A Not offhand.

1 Q Let me try to help you recall at a study
2 that talked about a mother and either a son or a
3 daughter -- I think it was a son -- who developed
4 leukemia very quickly working with their husband and
5 father in the same setting and the father did not
6 develop the leukemia for 12 or 15 years later. Do
7 you recall that article?

8 A No.

9 Q If that were the case, if I have accurately
10 related to you the study that the mother and the son,
11 I think it was, developed the leukemia much quicker
12 than the father working under the same conditions,
13 would that support the theory of individual
14 susceptibility to the myelotoxic nature of benzene?

15 A I would have to look at that literature
16 before I would try to answer that question.

17 Q Do you need to look at that before trial?
18 Will you be looking at that before trial?

19 A I suppose I could. I regard case reports
20 as being of relatively modest value in determining
21 whether benzene causes leukemia and frankly, in
22 answering any questions, any scientific questions in

1 a reliable manner.

2 Q Part of the overall picture but it's not
3 controlling the case studies, case reports? They're
4 some piece of the puzzle?

5 A Case reports play an important role in the
6 development of scientific information because they
7 often stimulate people to investigate issues with
8 scientific tools. Case reports in and of themselves
9 I regard as largely nonscientific and of questionable
10 reliability.

11 Q Does that mean that all of Aksoy's work
12 should be thrown out the window?

13 A No.

14 Q Why not? It's questionable reliability.

15 MR. HOLLINGSWORTH: Are you talking about
16 the case report on the mother and father?

17 MR. HARTLEY: No, all these case reports
18 that he has done. And basically, all his studies are
19 case reports, aren't they, and all his articles are
20 case reports based upon what he has seen over the
21 course of his practice.

22 THE WITNESS: No.

1 BY MR. HARTLEY:

2 Q They're not?

3 A No.

4 Q Which one is not?

5 A 1976 was a PMR study.

6 Q Other than that study, was everything else
7 published because of the question of reliability?

8 A I'd have to look at everything he's
9 published to see which were scientific studies and
10 which were case reports. I would be reluctant to
11 throw out valid scientific inquiries.

12 Q Does that mean that case studies can't have
13 valid scientific -- a valid scientific place in the
14 determination of whether benzene can cause acute
15 monocytic leukemia?

16 MR. HOLLINGSWORTH: Objection; asked and
17 answered.

18 MR. HARTLEY: He's changed his answer. His
19 first answer was that they have questionable
20 relevance. Now he's saying that they have a valid
21 place in the determination. I just want to know
22 which one it is.

1 MR. HOLLINGSWORTH: I object to your
2 characterization of the testimony. My understanding
3 of the testimony is that case reports are
4 nonscientific and are of questionable reliability.

5 BY MR. HARTLEY:

6 Q Is that your answer, Doctor? If that's
7 your answer, I'll accept that answer.

8 A That was half of my answer.

9 Q What's the other half?

10 A The other half is that they play an
11 important role in generating systematic scientific
12 inquiry.

13 Q When was the last time you looked at the
14 Ott/Bond study?

15 A Yesterday.

16 Q Did you look at the exposure levels that
17 were reported for the Ott/Bond study?

18 A I believe I did.

19 Q What did you find?

20 MR. HOLLINGSWORTH: Can we take a
21 five-minute break?

22 MR. HARTLEY: Can we take long enough for

1 me to go down and smoke a cigarette?

2 THE WITNESS: Sure. That would be fine.

3 (Recess.)

4 BY MR. HARTLEY:

5 Q Did you pull out the Ott portion of the
6 study?

7 A Yes.

8 Q Have you pulled out Bond's update of the
9 study?

10 A Yes.

11 Q Would you look at Bond's table 5.

12 A Yes.

13 Q Is it your opinion that the Ott/Bond study
14 is a reliable study?

15 A I'm not sure what you mean by "reliable,
16 but it appears to be a well-conducted study.

17 Q Did you look at the exposure data on table
18 5, the summary of myelogenous leukemia?

19 A Yes.

20 Q Have you attempted to calculate what the
21 parts per million months were in parts per million
22 years?

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21 Q I am. I'm confirming what you are saying.

22 I'm in agreement with you.

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1 MS. MILNAMOW: You used a calculator?

2 MR. HARTLEY: I used a calculator. In
3 fact, it's 21.89.

4 BY MR. HARTLEY:

5 Q Case number 2, age of death was 51. How
6 old was Mr. Lavender when he died?

7 A 46 -- is that right? Yes, 46.

8 Q Young man?

9 A Middle-aged, I believe, unfortunately.

10 Q I guess it's all relative, isn't it?

11 A The standard definition says 40s are
12 middle-aged.

13 Q Case number 2 had developed the leukemia
14 with only 1.5 parts per million years; correct?

15 A Yes.

16 Q Can you explain that to me in light of your
17 theory that you need 50 parts per million years and
18 you need a latency period of five years and so forth?

19 A Yes, I can.

20 Q Would you.

21 A We expect to see leukemia in every
22 population, even without benzene exposure, and so the

1 fact that one case occurred with short duration of
2 exposure and relatively low cumulative exposure
3 probably has nothing at all to do with exposure.

4 If we look at table 4, we see that -- in
5 fact, we expected to see two cases of leukemia based
6 on general population rates without benzene exposure,
7 so two of those cases would have been expected to
8 have occurred on their own even if this cohort of
9 chemical workers had never handled benzene at all.

10 Q So this one, in your opinion, is not a
11 benzene-related leukemia?

12 A Well, there's no reason to think that -- I
13 should say there is very strong reason to think that
14 we should not conclude all cases of leukemia in a
15 cohort are due to exposure. If that was true, it
16 would support the conclusion that leukemia doesn't
17 occur in the population in the absence of benzene
18 exposure, which is clearly false.

19 Q If we would assume that this is a
20 benzene-induced leukemia, the fact that he was only
21 exposed for 1.5 parts per million years would
22 undermine your theory of 50 parts per million year

1 history; correct, if we assume this to be a
2 benzene-induced leukemia?

3 A If we assume things that are clearly not
4 true, the conclusions we reach are not valid.

5 Q Let's look at case number 3 and case number
6 5. Those are both less than 50 parts per million
7 years that you're suggesting. Do we exclude those
8 cases as well? Are they not, therefore,
9 benzene-induced leukemia either?

10 A The answer is that when you review a
11 scientific study, you have to look at the pattern of
12 disease to see if there is evidence that associates
13 exposure with disease.

14 You can't -- it has no meaning to go
15 through the cases and pick and choose the ones you
16 like and discard the ones you don't like. That's not
17 a scientific method, and it will not answer any
18 questions about causation. So to pass judgment on
19 one case as being included and one case being
20 excluded is not a meaningful exercise.

21 What this paper shows is a modest increase
22 in leukemia risk in a cohort where there was

1 substantial benzene exposure.

2 Q It also shows, does it not, leukemia risk
3 with low level exposure? As reported, 1.5 parts per
4 million years, the man developed leukemia. 25 parts
5 per million years, the man developed leukemia. 28
6 parts per million years, the man developed leukemia.
7 It doesn't necessarily demonstrate a relationship
8 between high level exposure, does it, Doctor, and
9 leukemia?

10 A It shows no evidence of risk associated
11 with any of those individuals.

12 Q We'll take your hypothesis that you need 50
13 parts per million years to develop leukemia;
14 correct? That is your hypothesis. Is it 50 or 60
15 now?

16 A I believe what I said was that the
17 scientific literature indicates that there is a
18 causal association between acute myelogenous leukemia
19 and cumulative benzene exposure in the range of 50
20 parts per million years to 200 parts per million
21 years.

22 Q There's also other literature out there

1 that indicates that it's less than 50 parts per
2 million years; correct?

3 A There is not adequate literature to
4 conclude with reasonable -- with a high degree of
5 certainty that that association is not due to chance.

6 Q Didn't the NIOSH study find that?

7 A Which NIOSH study are we referring to?

8 Q The Rinsky study, that it was only 40 parts
9 per million, one part per million for 40 years.

10 A I'd have to look at the Rinsky study. You
11 and I are well aware that many people have reexamined
12 that cohort and the exposure estimates upon which
13 Rinsky's conclusions were based, and their findings
14 are considerably different than Dr. Rinsky's,
15 indicating that the risk estimates he derived were
16 based on underestimation of the exposures.

17 Q Of course, the people who reevaluated the
18 literature were sponsored by API, were they not?

19 A Some were.

20 Q Do you think that there would be some bias
21 in the work that was done when it was sponsored by
22 API?

1 A I would make no such assumption.

2 Q Would API want the exposure limit to be
3 higher than an independent NIOSH examination, based
4 on your understanding?

5 A My understanding is that API would want to
6 find the truth, not a biased representation of the
7 truth.

8 Q Under your view of the literature, and that
9 is 50 parts per million years, would it be safe to
10 assume, then, that case number 2, case number 3 and
11 case number 5 are not benzene-induced leukemias and
12 that the study is therefore invalid as relating to
13 any showing of a causal relationship because you
14 would only have two cases left and you're only
15 expecting 2.1?

16 A I think I've already answered that
17 question. It is not a valid method of scientific
18 inquiry to go through the cases and discard the ones
19 you don't like and keep the ones you like. That
20 doesn't lead to a reliable answer.

21 The answer in this paper is that when all
22 of the data are considered, it shows a modest

1 association between leukemia and benzene exposure in
2 a cohort that had substantial exposure.

3 Q You keep saying "substantial exposure."
4 But the truth of the matter is three of the five
5 cases that had substantial exposure, as you define
6 it, is less than what you're suggesting the
7 literature says you need, so what do you base your
8 substantial exposure on?

9 A And two of the five cases would have
10 occurred by chance alone in the absence of benzene
11 exposure, based on general population mortality
12 rates.

13 The second half of my answer is derived
14 from table 8, where the mortality is summarized by
15 estimated cumulative dose of benzene exposure, and
16 what it shows is that below 500 parts per million
17 months, which works out to about 41 parts per million
18 years, there is a modest association between exposure
19 and leukemia and that that association is not
20 statistically significant.

21 Q You testified in the past that statistical
22 significance is not a necessary requirement to

1 determine a causal association haven't you?

2 Didn't you look at other issues as well --
3 you looked at what the article was saying in addition
4 to other causative factors in determining a causal
5 relationship; is that true?

6 A I think there were two questions there.
7 Could you give me one at a time.

8 Q Sure. Have you testified that statistical
9 significance is not all-encompassing and not
10 absolutely positively necessary to draw a causal
11 association, yes or no? And then you can explain
12 it.

13 A Statistical significance is a concept we
14 use to evaluate the role of chance --

15 Q Let me interrupt you. Yes or no. Then you
16 can explain it. Have you testified in the past that
17 statistical significance is not absolutely necessary
18 to determine a causal relationship, yes or no, and
19 then you can explain it, but I want a yes or no
20 answer. I'm entitled to that.

21 A I do not recall the exact wording of
22 everything to which I've testified in the past. You

1 would have to show me my testimony that says exactly
2 that.

3 Q I do happen to have this one marked. Page
4 186 of your deposition.

5 A And what is the question?

6 Q Have you testified that statistical
7 significance is not an absolute requirement to
8 determine a causal association?

9 A I don't believe that characterizes my
10 testimony accurately.

11 Q What does -- how would you characterize
12 your testimony, Doctor?

13 A Why don't I read it into the record.

14 Q Sure.

15 A I said "when epidemiologists talk about
16 studies, they commonly refer to positive studies as
17 ones that show evidence of an association, whether
18 it's significant or not, and again the context of how
19 they're talking about it is probably more important
20 than the term 'positive study.' "

21 Then the next question was "you mean you
22 have a positive study that's not statistically

1 significant?"

2 My answer was "there are any number of
3 references that I've reviewed and listed for you that
4 show positive associations between various types of
5 leukemia and various exposure" -- it should have said
6 "exposures" -- "under study which are not
7 statistically significant. So a positive association
8 or positive study in my experience just means that
9 there is some evidence of an association of whether
10 it's statistically significant or not."

11 That was the end of my testimony. I don't
12 think that that can be characterized in any way as
13 what you claimed I said. I'd be happy to try and
14 give you my view of the role of statistical
15 significance in establishing causality.

16 Q Okay. Do that.

17 A "Statistical significance" is a term that
18 relates to the assessment of whether we can exclude
19 chance as an explanation for an observed
20 association. When a finding has a very low
21 probability of having occurred by chance, in other
22 words, as statistically significant, we conclude that

1 chance is a very unlikely explanation for the
2 finding. That is one of the things that we consider
3 in assessing whether an association is causal or is
4 noncausal.

5 Q But it is not the only one; correct?

6 A It is not the only one.

7 Q So if a study is not statistically
8 significant but it does show an elevated risk of a
9 particular disease entity, do you rely on that
10 particular study in assessing the causal association?

11 A I would certainly consider such a study.
12 The difficulty with such a study is that it is
13 difficult to be confident that the findings are not
14 due to chance.

15 Q But if you have other things that you can
16 look at which would support the association, then it
17 would be acceptable to utilize a nonstatistically
18 significant finding, correct, which was elevated?

19 A The determination of causation is based on
20 many factors, of which the ability to exclude the
21 role of chance is one. Rarely, if ever, in
22 epidemiology do we assess causation based on a single

1 study, whether it's significant or not.

2 Studies that do not show -- I should say
3 studies that cannot adequately exclude the role of
4 chance in their findings contribute very little to
5 the determination of causality. They're far less
6 important because we're not sure that we're seeing a
7 real association versus a chance association.

8 Q You said you could not take the five cases
9 in the Ott/Bond study, dissect them and exclude them
10 based on your review of the literature that in your
11 opinion requires 50 parts per million years;
12 correct? That wouldn't be fair?

13 A I don't think that's what I said.

14 Q What did you say specifically, then?

15 A I believe I said you can't pick and choose
16 and just keep the cases that meet some criterion and
17 exclude the others. That is not a scientific
18 exercise. The scientific approach to determining
19 whether benzene exposure was associated with leukemia
20 is based on considering all the data and analyzing it
21 in an appropriate fashion.

22 Bond and his colleagues present some of

1 those analyses in table 8, in which they show a weak
2 and nonsignificant association between benzene
3 exposure below 500 parts per million months, in other
4 words, 41 parts per million years, and leukemia
5 risk. That is not clear evidence of risk below 40
6 parts per million years. It's inconclusive evidence.

7 Q I understand that. If you look at the
8 entire study of what they conclude and what they said
9 here, all five of them, based on table 4 or table
10 8 -- the cumulative exposure table?

11 A I was referring to table 8.

12 Q Table 8. He had two cases below 499 parts
13 per million on table 8.

14 A Yes, 499 parts per million months.

15 Q Right, which is how many parts per million
16 years?

17 A About 41.

18 Q Had two cases there. We had zero cases
19 between 500 and 999; correct?

20 A Yes.

21 Q And we had one case over a thousand?

22 A That is correct.

1 Q And it's your testimony that based on table
2 8, you need in excess of 500 parts per million months
3 of benzene to develop a leukemia?

4 A It is my testimony that this paper does not
5 support the view that there is increased risk below
6 50 parts per million years.

7 Q And the basis for that is?

8 A That in table 8, there is no .. there is
9 only a weak and nonsignificant association between
10 exposure below 500 parts per million months and
11 leukemia.

12 Q Where is the association above 500 parts
13 per million months? There isn't any, is there?

14 A This paper only has three leukemia deaths.
15 It's a small study. It doesn't provide conclusive
16 evidence that benzene is associated with leukemia.
17 It's a small study.

18 Q How do we take the 50 parts per million
19 years that you're suggesting and apply it to
20 Mr. Lavender? It's an individual case, which .. you
21 can't pick and choose which cases you want to apply
22 it to and which ones you want to throw it out in.

1 So how do we apply your 50 parts per
2 million years to Mr. Lavender's case? You couldn't
3 apply it to any one of these cases here and throw
4 these cases out, so how do we apply it to
5 Mr. Lavender, solely on the exposure issue?

6 A The first step is to determine how much
7 exposure Mr. Lavender had in terms of his cumulative
8 exposure. Then the second step is to examine the
9 epidemiologic literature to determine whether there
10 is sufficient evidence to say that exposures of that
11 magnitude cause leukemia.

12 The answer to that second issue is there is
13 not adequate literature to conclude that those
14 exposures cause leukemia, and so there is simply no
15 basis for saying that leukemia -- that benzene
16 exposure caused Mr. Lavender's leukemia.

17 Q Now then, why would it be improper to go
18 back to the Ott/Bond study and do the same analysis?
19 In your opinion, you need 50 parts per million years
20 to 200 parts per million years. Why would it then be
21 improper to go back here and say case number 2, case
22 number 3 and case number 5 don't meet that criteria,

1 so they shouldn't be included in this study?

2 MR. HOLLINGSWORTH: Objection; asked and
3 answered.

4 THE WITNESS: The answer is that in the Ott
5 and Bond study we are doing a scientific inquiry to
6 examine a pattern of exposure and leukemia to see if
7 there is an association. That is a different goal
8 than looking at Mr. Lavender and trying to determine
9 whether benzene caused his leukemia.

10 I'm not basing my opinion that it takes 50
11 parts per million years of benzene exposure or more
12 to cause leukemia on Bond and Ott alone. That
13 opinion is based on a synthesis of a large amount of
14 epidemiologic literature that strongly supports that
15 conclusion.

16 Once I've made that conclusion, I can
17 justify setting exposure criteria by which it is
18 defensible -- or I should say by which it is reliable
19 to assess whether Mr. Lavender was at increased risk
20 from his exposure. That's a very different goal than
21 trying to do science.

22 Let me add to my answer. If we were to do

1 an epidemiologic study of Mr. Lavender's peers at the
2 Mobay facility to see if there was evidence of
3 leukemia risk associated with benzene exposure in
4 that facility, we would most certainly include
5 Mr. Lavender and all other members of that work group
6 or that cohort in the study. It would be inexcusable
7 to pick through the people and say this guy's in,
8 this guy's out. That's not science.

9 So we would most certainly include him in a
10 study to see if there was an association. If we saw
11 no evidence of an association, then we would conclude
12 that Mr. Lavender was not at increased risk because
13 of benzene exposure in his plant.

14 BY MR. HARTLEY:

15 Q When you do an epidemiological study of the
16 Mobay facility, as you suggested could be done, do
17 you include contract workers as well as Mobay
18 employees in that study?

19 A You certainly could. There'd be no reason
20 to exclude them. They could be included. They
21 could -- the study could be done with or without
22 them, depending on feasibility issues.

1 Q Would the fact that they were not included
2 in a study give you any enlightenment as to disease
3 processes in the nonincluded group? Do you
4 understand my question?

5 A I believe so. If the contract workers were
6 similar to the workers who were included in terms of
7 their other risk factors for leukemia and were
8 similar in terms of their distribution of exposure,
9 the findings from the workers who were included would
10 be applicable to the contract workers.

11 Q But if the contract workers were not
12 provided with respirators, were not provided with
13 safety equipment when they were working around
14 benzene and the hourly Mobay people or salary people
15 additionally were, then there would be a difference
16 in exposure, would there not? That way you could not
17 utilize the epidemiological study of the Mobay people
18 to support a lack of causation for the contract
19 employees?

20 A Could I ask you to split that into two
21 questions, and I'll try to answer them separately.

22 Q Sure. First question is, if the contract

1 employees are not given safety equipment to protect
2 against exposures to benzene, their rate of exposure,
3 potential exposure would be higher than the hourly
4 people who are given the adequate protection and use
5 it; correct?

6 A Not necessarily, or I should say not
7 necessarily appreciably higher. It's my impression
8 from the industrial hygiene data that the exposures
9 in the Mobay plant were well under 1 part per million
10 virtually all the time.

11 Q What do you rely on for that?

12 A I'm sorry?

13 Q What do you rely on for that?

14 A The industrial hygiene data, and that
15 that's an exposure level which would not cause people
16 to wear their respiratory protective equipment.

17 Q How about when there were spills ..

18 A I was still answering.

19 Q I didn't know that you were. I apologize.

20 A Insofar as the Mobay employees are not
21 wearing respiratory protective equipment routinely
22 nor are the contractors, their exposure levels in the

1 same locations are expected to be identical or
2 comparable.

3 When there are excursions of exposure to
4 very high levels of benzene, it is possible that the
5 full-time people would put their respiratory
6 protective gear on, although from my reading of the
7 depositions, it was not clear to me that that
8 occurred on any regular or even infrequent basis.

9 And so I'd have to say, based on my
10 understanding of the routine exposures and the spills
11 and unusual occurrences, that the full-time people
12 did not wear respiratory protective equipment but
13 rarely, and therefore, their cumulative exposure is
14 probably very similar, if not identical to that of
15 the contract workers.

16 Q What is your opinion of the parts per
17 million of benzene in the air when you can smell it?

18 A Are you asking me what the odor threshold
19 is?

20 Q Correct.

21 A I believe it's in the few parts per million
22 range.

1 Q Do you disagree with the American
2 Industrial Hygiene Association's geometric mean of 61
3 parts per million?

4 A Are you referring to the odor threshold
5 document they published in 1987?

6 Q That's correct.

7 A I think that estimate is high.

8 Q And what do you support that on?

9 A An estimate made by ASTDR and my own
10 personal experience.

11 Q Have you seen Paustenbach's explanation of
12 what the odor threshold is in 1992 or '3 in his
13 rereview of the pliofilm?

14 A I do not recall his discussion of that.

15 Q The study was responsive by API in that
16 particular study. Do you have it with you?

17 A Is that the one from Environmental Health
18 Perspectives in '93?

19 Q The long paper.

20 A Looks to be.

21 Q It's toward the end. Did you find it yet,
22 Doctor?

1 MR. HOLLINGSWORTH: What page is it on

2 MR. HARTLEY: It's toward the end. I know
3 it's at the top. I didn't bring my own copy of the
4 article, but it's at the top, I know, of one of the
5 pages and I believe that he says that the odor
6 threshold is 35 parts per million.

7 BY MR. HARTLEY:

8 Q You don't find it in there. Let's assume
9 that he says it's 35 parts per million. Do you
10 believe that would be too high as well?

11 A Yes.

12 Q Why would someone from API want the odor
13 threshold to be higher than what it should be, then?

14 A I really don't know the motives of the
15 people who wrote that.

16 Q It's in the section right after
17 respirators, I think. That's okay. We'll move on.
18 I don't think this is the right study.

19 A Oh.

20 Q It's a longer study. What year is this
21 one?

22 A '93.

1 Q That's not the right study. That's why you
2 couldn't find it.

3 Why did you utilize the Devine study on
4 refinery for gasoline workers?

5 A I'm sorry, why did I what?

6 Q Why did you utilize Devine when it's not a
7 pure benzene situation, or is it?

8 A It's a compound question. It is not a pure
9 benzene study. It is a study of refinery
10 petrochemical and research workers and a separate
11 study of producing pipeline workers.

12 The reason I included some of the studies
13 of petroleum workers is that they have low level
14 exposure to benzene in the course of handling
15 petroleum products and so it is relevant to the issue
16 of whether those low level exposures are associated
17 with leukemia.

18 Q What do you -- how would you calculate a
19 designation of benzene exposure, that is in
20 milligrams per cubic meter, 18 milligrams per cubic
21 millimeter?

22 A How would I do what with it?

1 Q Change it to parts per million. 18.1 would
2 be what?

3 A Roughly 6.

4 Q 6 parts per million?

5 A Yes.

6 Q Have you seen the Yin studies?

7 A I have seen studies by Yin, yes.

8 Q Did you utilize it for this particular
9 case? And I'm talking about Yin 1987.

10 A Do I have Yin '87? Yes, I do. Industrial
11 page 124?

12 Q No, 192.

13 A Yes.

14 Q Did you notice the geometric mean
15 concentration of benzene in 50,255 workplaces was 6
16 parts per million, correct, summary of the abstract,
17 Doctor?

18 A What number did you give?

19 Q The geometric mean concentration of benzene
20 in 50,255 workplaces was 18.1 milligrams per cubic
21 meter which, in your translation, was approximately 6
22 percent -- 6 parts per million?

1 A That is what he said, yes. However, if you
2 look at the distribution of exposures, they go -- he
3 only shows them from 10 to 2000 milligrams per cubic
4 meter, so he shows them from roughly 3 to 600 parts
5 per million, and there were appreciable numbers of
6 them, well above 100 milligrams per cubic meter or 33
7 parts per million.

8 Q What would 40 milligrams per cubic meter
9 be?

10 A Approximately 13.

11 Q 64 percent of the workplaces were less than
12 13 parts per million, according to the abstract.

13 A Yes.

14 Q So appreciable number -- there might be an
15 appreciable number above it, but the geometric mean,
16 what is it, Doctor, what is the definition of
17 geometric mean?

18 A For end measurements, it's the product of
19 all measurements to the one over *n*th power.

20 Q Which means what, in layman's terms?

21 A You take all the measurements, you multiply
22 them together, and **you** take the *n*th root of that

1 product and that's the geometric mean. Geometric
2 means are very far below arithmetic means for data
3 that have a long tail to the right. So what you're
4 actually looking at is a number they've chosen to
5 represent the mean in a manner that grossly
6 underrepresents the arithmetic mean.

7 Arithmetic mean is the one we're familiar
8 with where we sum the measurements and divide by the
9 number of measurements. So you could .. they could
10 have chosen a different measure of general tendency
11 like the arithmetic mean, and that mean would have
12 been much, much higher than the one they presented.

13 Q Why would you use a geometric mean?

14 A Because it is less susceptible to the
15 effect of a value or a few values that are very much
16 higher than the main body of information. So it's
17 not swayed by an extreme value the way an arithmetic
18 mean is.

19 So for example, if you and I had exposure
20 data where we had 100 values that were between 1 and
21 5 and two values of 2000, an arithmetic mean would be
22 moved up by those values. A geometric mean is not

1 skewed by those as much. It's an appropriate thing
2 to do when there are a few outlying values. It
3 becomes less appropriate as the outlying values make
4 up a larger and larger percent of the data set.

5 Q What percent would that be, in your
6 opinion?

7 A Well, looking at his probability
8 distribution function of benzene concentrations, I
9 would say there are a lot of values up above 100
10 milligrams per cubic meter. I would like to have
11 seen the arithmetic mean as well as the geometric
12 mean.

13 Q Does it say how many cases he found up
14 above the 100 milligrams per cubic meter?

15 A I'm sorry. What was the question again?
16 What percent of cases had exposure above 100
17 milligrams per cubic meter?

18 Q Yes.

19 A You'll have to direct me to that. I don't
20 see that.

21 Q What is the significance of the fact that
22 the geometric mean concentration of benzene was 18.1

1 milligrams per cubic meter and the 95 percent range
2 was .06 to 844.74 milligrams per cubic milligram on
3 page 193?

4 A What's the relevance of that?

5 Q Yes.

6 A It says that the vast majority of the
7 measurements fell within that range bounded by .06
8 and 844.74 milligrams per cubic meter.

9 Q Does that mean that 95 percent of the cases
10 fell between those two numbers?

11 A No, 95 percent of the benzene exposure
12 measurements fell in that range.

13 Q That's what I meant.

14 Why didn't you utilize the Yin study in
15 your analysis of Mr. Lavender?

16 A There are a lot of issues that remained
17 inadequately described in this paper, and I cannot
18 tell if the results are reliable as a result.

19 Q It was a peer reviewed paper, wasn't it?

20 A It's in a very fine journal that is a peer
21 review journal.

22 Q Yin had follow-up, didn't he? Did you see

1 the follow-up studies that were done on his cohort?

2 A To what are you referring?

3 Q For one, there was a 1994 update on
4 leukemia and lymphoma. Do you have that one?

5 A Are you talking by Lee?

6 Q No, by Travis. Do you have that?

7 A I do not believe I have that one.

8 Q When you were doing your search, since you
9 knew that the Yin study in your own opinion was
10 somewhat questionable, did you do your Medline search
11 to see if there was any further update on the Yin
12 study?

13 A I did my Medline search to look for new
14 literature on benzene and leukemia. If the Travis
15 study had been assigned the appropriate key words, I
16 probably would have found it.

17 Q "Benzene," "leukemia," "dysplasia," would
18 those be key words you'd be searching for on Medline?

19 A I didn't use the word "dysplasia." I did
20 use "benzene" and "leukemia."

21 Q Would "benzene" and "leukemia" have been
22 picked up in this particular article and would you

1 have found it on your Medline?

2 A I couldn't tell you without getting the
3 article and seeing what the key words were under
4 which it was indexed.

5 Q It was under -- "benzene," "leukemia" and
6 "dysplasia" were the key words. I've got it in
7 front of me.

8 A Those are the Medline key words? Those
9 aren't the Medline key words. That's not necessarily
10 what the National Library of Medicine entered for
11 it. Those are -- I used a number of key words. I
12 remember specifically using "leukemia" and
13 "benzene." And I do not remember seeing a citation
14 by Travis.

15 Q You didn't find that article either;
16 correct? That didn't come up in your search?

17 A That's correct.

18 Q Do you believe that Mr. Lavender's smoking
19 history caused or contributed to his development of
20 acute monocytic leukemia?

21 A Yes, I do.

22 Q And what is it in tobacco smoke that causes

1 leukemia?

2 A I'm not sure we know that with certainty.
3 The epidemiology points consistently toward an
4 association between smoking and leukemia. We do know
5 that benzene is present in cigarette smoke. I do not
6 believe we know that that is the reason why smokers
7 have elevated risk of leukemia, however.

8 Q Has anyone published an article saying what
9 the cause may be in cigarette smoke for leukemia that
10 you're aware of?

11 A I'm not aware that anyone has meaningful
12 data on that. I wouldn't be surprised if lots of
13 people have speculated as to the cause, but I don't
14 know that anyone has meaningful data.

15 Q Do you know what the speculations are?

16 A We could look at some of the articles and
17 see, if you want to do that.

18 Q Sure. Let's do that. Which articles do
19 you want to look at on smoking? You're writing down
20 something else. What are you looking for now?

21 A Travis.

22 Q Will you find the Travis article before

1 trial?

2 A I'd like to see that.

3 Q Did you come across the Robert Snyder
4 article in your Medline search?

5 A To which one are you referring?

6 Q 1994.

7 A I have Critical Reviews in Toxicology.

8 Q Yes.

9 A Yes. Well, now, one article by Michael
10 Segal postulates "at least three chemical leukemogens
11 (benzene, urethane and nitrosamines) are present in
12 tobacco smoke." In addition, the concentration of
13 lead 210, a radioactive leukemogen, has been
14 demonstrated to be significantly increased in the
15 bones and soft tissues of smokers, so there are a
16 number of plausible hypothesis for that association.

17 Q Benzene is plausible?

18 A I believe that each of those has to be
19 examined to determine how well the hypothesis fits
20 the existing data.

21 Q Do you have statistics, Doctor, on the
22 amount of benzene in the cigarette smoke?

1 A I know that that has been published. I've
2 seen it. I do not recall the numbers.

3 Q For the benzene and the cigarette smoke to
4 have caused Mr. Lavender's leukemia, would he need to
5 be exposed to the 50 parts per million years?

6 A It is my view that the best evidence that
7 benzene causes leukemia in humans is derived from the
8 studies we've been discussing and they strongly
9 support the interpretation that exposure levels below
10 50 parts per million years are not risk factors for
11 leukemia, so the answer would be yes.

12 Q Do you know what the number for 25
13 pack-year history for Mr. Lavender -- do you know
14 what his parts per million level of benzene would be
15 for smoking that long a period of time?

16 A I do not.

17 Q You didn't calculate that?

18 A I did not calculate that.

19 Q Will that be something you will do prior to
20 trial?

21 A I'll put it on the list.

22 Q What is the relative risk of leukemia in

1 cigarette smoke? Are you basing this on the Segal
2 article?

3 A There are a number of articles I'd like to
4 look at, but Segal is one of them.

5 Q What's the cite of Segal?

6 A American Journal of Epidemiology, volume
7 138, page 1, 1993, pages 1 to 9. The pooled odds
8 ratio for myeloid leukemia in Segal based on a
9 summary of nine studies is 1.23 with confidence
10 limits that go from 1.08 to 1.39.

11 Q 1.08 to ..

12 A 1.39. Those are case control studies.

13 Q Well, we know that those are not really
14 relevant when we're talking about epidemiological
15 studies, are they, because we threw them out when we
16 were talking about Aksoy. So the case control
17 studies that Michael Segal bases his analyses on
18 would not really have any relevance, would they, in
19 good epidemiology?

20 A I suspect that you're mistaking case
21 control studies for case reports. Case control
22 studies are scientific studies, and they are

1 different than case reports, so we would not throw
2 them out.

3 There are also six cohort studies, four of
4 which examine myeloid leukemia risk and find a pooled
5 relative risk of 1.51 with 95 percent confidence
6 interval that goes from 1.31 to 1.74. So both case
7 control studies and cohort studies demonstrate a
8 significantly elevated risk of leukemia associated
9 with smoking.

10 Q Significant?

11 A Yes, statistically significant.

12 Q But it is really only a modest relative
13 risk. You've only increased it how many, .23 above 1
14 and .5?

15 A I would call it a modest risk.

16 Q The reports are not discussing acute
17 monocytic leukemia, but are rather zeroing in on
18 acute myeloid leukemia; correct?

19 A They are zeroing in on myeloid leukemia,
20 and that is correct.

21 Q And that is not the same as acute
22 monocytic, is it?

1 A It includes acute monocytic. Now, that --
2 I'm not sure the intent of your question. If it's to
3 say that this literature is not relevant to the
4 determination of smoking risk to acute monocytic
5 leukemia, then the same argument is made of the
6 benzene literature, that very little of it examines
7 acute monocytic leukemia specifically, and there's
8 almost no data whatsoever showing that's associated
9 with benzene.

10 Q Except Kenny Crump's article that you
11 didn't find; right?

12 A I have not had time to review that article
13 in detail.

14 Q So does that mean that specifically the
15 Segal article that we've just been discussing does
16 not particularly reference acute monocytic leukemia;
17 correct?

18 A It references myeloid leukemia without
19 specifying subtypes.

20 Q Did they try to break it down into
21 subtypes?

22 A To the extent of my recollection, they did

1 not break it down any finer than myeloid leukemia.

2 Q Did they give you an indication as to
3 whether they tried and they could not do that within
4 the article itself?

5 A I do not recall whether they said that. I
6 think I know the original literature well enough to
7 know that it can't be done.

8 Q Why can't it be done?

9 A Because the original literature, to the
10 extent I know, doesn't break down the risks by
11 subtype of myeloid leukemia in any consistent
12 manner. So you're forced to summarize the study by
13 using a broader topic since you don't have adequate
14 detail to use more precise diagnostic topics.

15 Q Let's go back to the Bond/Ott study just
16 for one more second -- never mind. Forget about it.

17 Doctor, do you believe there's any safe
18 level of benzene exposure?

19 A With respect to what outcome?

20 Q Any outcome.

21 A Well, it has an explosion limit below which
22 there's safety from explosions. There are all sorts

1 of different limits that we could talk about.

2 Q Let's talk about human disease or human
3 cytogenetic studies or human changes within the **body**
4 as a result of benzene exposure, changes in the human
5 body. Is there any safe level of benzene to the
6 human as far as medical disease processes are
7 concerned?

8 A If you want to focus on leukemia risk, I
9 would say that exposure levels below 50 parts per
10 million years are not known to have any risk of
11 leukemia associated with them.

12 Q Are you familiar with McDonald studies,
13 Gene McDonald?

14 A I can't say that I am. What are those
15 about?

16 Q 20 parts per million for three months
17 causes leukemia. You're not familiar with his
18 studies?

19 A I am not familiar with this study or the
20 author to which you're referring. Can you give me a
21 citation on that?

22 Q Dr. McDonald is with NIEHS. Are you not

1 familiar with those studies?

2 A Is that published work?

3 Q Yes.

4 A Could I have a citation on some of it? I'd
5 be happy to review it.

6 Q No, I don't have a citation for it. Have
7 you studied Maltoni's literature?

8 A I have seen some of the things he's
9 written.

10 Q Does Maltoni suggest a level of exposure
11 below 50 parts per million years to develop leukemia?

12 A I don't know whether he suggests that or
13 not.

14 Q Have you read his literature?

15 A I have read his literature in the past. I
16 didn't bring it with me, didn't read it in
17 preparation for this deposition, but I know I've read
18 a number of things he's written.

19 Q Are lymphocytes affected by benzene
20 exposure?

21 A I'm sorry?

22 Q Are lymphocytes affected by benzene

1 exposure?

2 A I'm not sure what you're asking. Benzene
3 exposure is adequate to cause pancytopenia and
4 certainly affects the lymphocytic series of cells.
5 Other than that --

6 Q What is the medical term for a decrease in
7 the number of lymphocytes?

8 A Lymphocytopenia.

9 Q Is lymphocytopenia associated with benzene
10 exposure absent pancytopenia?

11 A I would have to look at that issue. I'm
12 not aware that it is.

13 Q Is that another issue you're going to look
14 at before trial?

15 A Yes.

16 Q Do you believe, Doctor, that one exposure
17 to benzene can cause leukemia to evolving?

18 A I'm not aware that there is any support for
19 that hypothesis in the scientific literature and so
20 I'd say there's no basis for making that conclusion.

21 Q Is that a proposition that is floating out
22 in the scientific community?

1 A I don't know whether it's floating out
2 there or not. It's not floating in my office.

3 Q Can one molecule cause mutation of a
4 particular stem cell?

5 MR. HOLLINGSWORTH: One molecule of
6 benzene?

7 MR. HARTLEY: Benzene, yes.

8 THE WITNESS: The answer is in theory, one
9 molecule can cause mutation. In practice, that's a
10 speculation that doesn't have any meaning. We have
11 no ability to observe the effects of one molecule of
12 exposure or to examine the risk associated with one
13 molecule of exposure, and I'd say that even in a
14 laboratory setting, we can't do that, much less out
15 in the world of free ranging human beings.

16 BY MR. HARTLEY:

17 Q Is there a biological marker for benzene
18 that you're aware of?

19 A I'm not aware of a biological marker for
20 benzene.

21 Q Have you done any research into that? Have
22 you looked at that issue?

1 A I know the epidemiology of benzene in
2 leukemia, and I'm not aware of any studies based on
3 markers of exposure to benzene. I guess I should ask
4 you what you mean by "biologic marker" to be sure I
5 understand your question.

6 Q What do you understand the term to mean?

7 A It has many definitions, depending on who's
8 using it and how they're using it, so there is no
9 single definition for "biologic marker."

10 Q Have you done any -- outside of the
11 epidemiological literature, have you done any Medline
12 search to determine whether there's a biological
13 marker for benzene, as that term is generally used?

14 A Well, my search focused on "benzene" and
15 "leukemia." That would have brought up articles
16 that looked at biological markers if they were
17 related to the issue of benzene and leukemia and none
18 came up. So I guess the answer is yes, I've done a
19 search that should have found those articles if they
20 were there.

21 Q Did you specifically ask for "biomarkers"
22 or any particular term, or what terms were you using

1 in your search?

2 A To the extent I recall, it included
3 "leukemia" and "benzene." I don't recall the other
4 terms I used. I know there were four or five other
5 terms.

6 Q Are you familiar with Dr. Goldstein's
7 studies on biological markers for benzene?

8 A Dr. Bernard Goldstein?

9 Q Yes.

10 A I have read some of his literature, yes.

11 Q Did you review any of that literature for
12 today?

13 A No, I don't think I did.

14 Q Do you have a list of what you reviewed for
15 today?

16 A No, I don't.

17 Q What do you keep referencing there in front
18 of you?

19 A I have a partial listing of the literature,
20 but it's not complete because I've added articles to
21 it and have not updated this listing.

22 Q Are all the materials that you reviewed in

1 front of you today?

2 A Yes.

3 Q What's your hourly rate, Doctor?

4 A \$350.

5 Q How many hours do you have in this case?

6 A I do not know.

7 Q Have you billed yet?

8 A I don't think so.

9 Q When did you first get this case?

10 A I'm not sure. However, I have a letter of
11 transmittal of documents. The earliest one is dated
12 October 17, 1994. My recollection is I got it before
13 that, but I don't know when.

14 Q Did you start working on it when you
15 received it?

16 A I don't remember when I started working on
17 it. I certainly didn't start the day I got the first
18 documents, but I have worked on it over the past few
19 months.

20 Q How many hours do you think you have in
21 this case? Do you have any idea, estimate?

22 A Probably between .. excluding today?

1 Q Yes, sure.

2 A 25 maybe.

3 Q 25 hours? Does that include all the
4 literature research, review of all the depositions
5 that you gave me on the list, the interrogatory
6 answers, the depositions of Dr. Shadduck,
7 Dr. Mehlman, Dr. Parmer, Dr. Przybysz,
8 P-r-z-y-b-y-s-z, Dr. Pachter, P-a-c-h-t-e-r,
9 Dr. Rose, Jack Crum's deposition, Paul Tepe, Robert
10 Eyler, Ernest Earley, which I think should be Kyle
11 Earley, Raymond LeMasters, James Johnson -- a T? --
12 Johnston, Jesse Caravaggio, William Thompson, James
13 Myers and John Lavender's deposition?

14 A Yes.

15 Q You reviewed all that material in 25 hours
16 in addition to doing the Medline search?

17 A Yes.

18 Q Have you taken the Evelyn Wood's speed
19 reading course? Because I know I've read that
20 material and I couldn't have read it in 25 hours.

21 A I have not taken the Evelyn Wood's speed
22 reading course.

1 Q When do you expect to bill in this case?

2 A I haven't set a date. Probably soon.

3 Q Did you meet with either Mr. Hollingsworth
4 or Ms. Milnamow yesterday?

5 A Yes.

6 Q How long did you meet with them then?

7 A We met for most of the day.

8 Q Eight hours?

9 A Something like that.

10 Q In the 25 hour estimate, is the eight hours
11 included in that 25 hours?

12 A No.

13 Q So we're over 33 hours. Have you met with
14 them prior to yesterday?

15 A No, I don't believe so.

16 Q Telephone conferences?

17 A Yes.

18 Q Any long telephone conferences, an hour,
19 two-hour conference?

20 A I don't think so. I don't recall, to be
21 honest with you.

22 Q How do you keep track of your time?

1 A I write it down.

2 Q Do you have a little ledger that you keep
3 on each case?

4 A I have pieces of paper.

5 MR. HARTLEY: We might as well take a lunch
6 break because I don't think I can get done in another
7 hour. Take about a half hour.

8 THE WITNESS: I'm happy to work through.

9 MR. HARTLEY: If you want to work through,
10 I'll work through.

11 THE WITNESS: It's only noon. If you can
12 do it in another hour, hour and a half, that's fine
13 with me. Let's keep going.

14 (Recess.)

15 BY MR. HARTLEY:

16 Q Let's talk about that study for a second,
17 the Yin study you and I were discussing off the
18 record in Environmental Health Perspectives. You
19 didn't refer this article in determining your 50
20 parts per million years according to what we talked
21 about earlier; correct?

22 A That is correct.

1 Q Do you have that study?

2 A I do. We're talking about Environmental
3 Health Perspectives, volume 82, page 207?

4 Q To 213.

5 A Yes, I do.

6 Q "Leukemia occurred among some workers with
7 as little as 6 to 10 parts per million average
8 exposure and 50 parts per million years, cumulative
9 lifetime exposure?

10 A What they say is "leukemia occurred among
11 some workers with as little as 6 to 10 parts per
12 million exposure and 50 parts per million years or
13 possibly less cumulative lifetime exposure."

14 Q What does that mean to you, that you have
15 to have both of them? I notice you emphasized the
16 conjunction.

17 A First off, my reading of this study leads
18 me to believe that they didn't actually analyze the
19 data by exposure level. There's nothing presented
20 about results by exposure level.

21 Q Table 5?

22 A Well, table 5 is a -- sort of a picture of

1 the exposure level of each case, but there's no
2 analysis of risk by exposure level, and so the
3 conclusions stated in the abstract doesn't seem to
4 have any foundation in the paper.

5 Q If you'd look down through the case
6 identification numbers and again, he has it by
7 milligrams per cubic meter, so if we divide by 3, are
8 we close?

9 A 3.25, yes.

10 Q Case number 9 would have had less than 30
11 parts per million cumulative exposure?

12 A Yes.

13 Q Case number 10 ..

14 A Wait a minute. I'm sorry. No. That looks
15 like it's actually case 8, but inexplicably, they've
16 dropped down a line.

17 Q I guess so. So it's case 8, which is less
18 than 30 parts per million years?

19 A Yes.

20 Q Case number 9 would be what, less than 12
21 parts per million years?

22 A Looks like about a little over 12

1 actually -- no, I'm sorry, a little less than 12.

2 Q Case 11?

3 A A little less than 10.

4 Q Did you say a little less than 10?

5 A No. 37 divided by 3-1/4, a little more
6 than 10.

7 Q Less than 11, a little more than 10?

8 A Somewhere between 10 and 11.

9 Q Case number 11, 110 milligrams per cubic
10 meter?

11 A 35 maybe.

12 Q 15, which is 52, which is what --

13 A 17 and a third -- a little less than 17.

14 Q Case number 16 at 93 which would be 33,
15 less than 31. Case number 18, 12 parts per million
16 years?

17 A Uh-huh. What's the question?

18 Q All these numbers are below 50 parts per
19 million years; correct?

20 A That's correct. However, there's no data
21 in the paper that indicates that those exposures are
22 associated with increased risk of leukemia. There's

1 no analysis in this paper that looks at risk by
2 exposure level, so that's nothing more than
3 descriptive data on the exposures of the people. And
4 as far as the analyses go, it appears not to have
5 been used.

6 Q Page 212, "findings of leukemia risk
7 subsequent to relatively low estimated average
8 accumulative lifetime of benzene exposure supports a
9 recent report by Rinsky." Do you see that?

10 A I see that.

11 Q Is that an attempt by the author to
12 correlate the low level exposures to the leukemia and
13 risk?

14 A No. It appears to be a statement not based
15 on any analyses. It appears to have no foundation.
16 We can critique this paper in more detail, if you'd
17 like to go through it. There are some weaknesses
18 that lead me to think that this is not reliable.

19 Q Is it a peer reviewed article?

20 A Yes -- or I should say it's in a journal
21 that does peer review and in fact, for which I've
22 done peer review of articles.

1 Q Would an article be published in
2 Environmental Health Perspectives without being
3 reviewed?

4 A I would think not.

5 Q What did you find deficient in this
6 particular article?

7 A The exposure information --

8 Q Specifically?

9 A .. is one of the issues that I find to be
10 inadequately described to make sense out of it.

11 Q In other words, you have problems with the
12 way the authors have indicated the exposure data
13 within the article itself or that it's lacking
14 something?

15 A It is entirely lacking from the paper what
16 exposure information existed and how it was obtained
17 and what the numbers .. the exposure numbers actually
18 looked like in a meaningful way.

19 It's **also** entirely lacking from the
20 paper .. let me rephrase that sentence. That's
21 awkward. The paper contains no results based on the
22 exposure information, and so all of the conclusions

1 that are stated in the discussion about risk at
2 exposure levels are simply without foundation in the
3 paper.

4 Q Would you say that the paper has no benefit
5 in attempting to associate benzene to low level
6 exposures?

7 A I regard it as unreliable. I draw no
8 conclusions from this because I don't think it's
9 reliable.

10 Q You haven't seen Travis's update of that;
11 correct?

12 A I have not.

13 Q What is your estimate of Mr. Lavender's
14 benzene exposure?

15 A Based on the still hygiene data that was
16 done in various areas of the plant, it appears that
17 the exposure levels while he was in the MNB area were
18 routinely in the .01 to . . I'm not sure what upper
19 limit to state somewhere in the tenths of parts per
20 million. His exposure levels for most of his work in
21 the iron oxide area were not detectable. There was
22 not benzene used in that area and so the exposures

would have been negligible.

2 Q Anywhere else?

3 A His exposure levels when he was in ECD were
4 routinely down in the .01 parts per million to a few
5 tenths of a part per million.

6 Q Anywhere else? Did you make an estimation
7 as to the amount of benzene he was exposed to when he
8 worked around the wastewater trench itself?

9 A My impression from reviewing the area
10 samples throughout the plant is that the exposures in
11 the areas in which benzene was used directly were, as
12 I've stated, in the hundredths or tenths parts per
13 million and that included working in the ECD area.

14 Based on that, I think it's unlikely that
15 the exposures near the trenches were appreciably
16 different than those levels.

17 Q And what do you base that on, the fact that
18 all the other levels in the area were low?

19 A Yeah. The fact that the area level -- many
20 of the measurements were area measurements and they
21 showed low levels almost all the time with occasional
22 excursions into the few parts per million, so it's

based primarily on the fact that the levels in every
2 area that were measured were very low.

3 Q What significance or relevance, if any, do
4 you find with the specific monitoring that was done
5 above the trench close to the DNT area, which showed
6 benzene between 5 and 50 parts per million after an
7 upset in the mononitrated benzene area?

8 A I do not recall those specific measurements
9 that you're referring to. However, I don't attach
10 great significance to a brief excursion in the
11 benzene level in air associated with a spill of
12 benzene that would -- if benzene were spilled into
13 the trench, there would be a change in exposure that
14 would accompany that spill.

15 Q Why don't you attach any significance to
16 that -- first of all, have you ever seen that data,
17 the particular memo that discusses 5 to 50 parts per
18 million over and above the trench or near the trench,
19 the different levels that were taken? Have you seen
20 that memo?

21 A I do not know which memo you're referring
22 to specifically. I went through a stack of data

1 yesterday. If I could see the memo to which you're
2 referring, I could tell you what was in there.

3 Q But you don't attach any significance to
4 that finding?

5 MR. HOLLINGSWORTH: What finding?

6 MR. HARTLEY: The 5 to 50 parts per million
7 above the trench.

8 THE WITNESS: Assuming that the sample
9 showed that, and that the sample was collected and
10 analyzed properly, it would represent the air level
11 where the sample was collected at the time of that
12 spill.

13 Spills were uncommon and my understanding
14 of Mr. Lavender's work records is such that he was
15 not exposed to spills but rarely. And so while he
16 may have had an exposure at that level in what
17 instance or a few instances, that exposure level
18 would not appreciably alter his cumulative benzene
19 exposure over the entire time he was at the Mobay
20 plant.

21 BY MR. HARTLEY:

22 Q First of all, you say that upsets or spills

1 were uncommon at the Mobay plant?

2 A That is my impression.

3 Q And what do you base that on?

4 A Record of spills prepared by Mobay that
5 tabulated them.

6 Q Do you have a copy of that with you?

7 A I do not, but I was shown that yesterday.

8 Q Fo'r what period of time did you have those
9 record of spills?

10 A For every period of time for which
11 Mr. Lavender was on the site.

12 Q What was that particular document called?

13 A I do not recall the title of it.

14 Q And it discussed all of the .. was it a
15 control log or was it a computerized printout or some
16 typewritten version? What did it look like?

17 A It was a computerized printout listing
18 every date on which there was a spill of benzene, the
19 amount spilled, the plant involved and the estimated
20 cost of the spill.

21 Q Okay. I've seen that document. That
22 document does not include all of the dates, does it?

1 Does it include every date that there was a spill, or
2 do you know?

3 A I was under the impression it included
4 every date there was a spill.

5 Q Let me represent to you that it doesn't.
6 Do you -- have you reviewed the depositions where the
7 various management employees indicated that many
8 times there were upsets that were not recorded?

9 A I do not recall that specific statement.
10 You have the list of things that I have reviewed.

11 Q Let me represent to you Ray LeMasters, who
12 was a foreman in that particular area; Kyle Earley,
13 who was a particular foreman; William Thompson, who
14 was a particular foreman in that department, all
15 indicated that there were times when upsets occurred
16 that were not recorded.

17 MR. HOLLINGSWORTH: I object to that
18 characterization of the testimony. I don't believe
19 that's what it says at all.

20 BY MR. HARTLEY:

21 Q Let me represent that to you. My question
22 would then be, the basis for your impression that

1 there were a few upsets at this facility is based
2 totally upon the particular documents that you have
3 been shown by Mr. Hollingsworth?

4 A It is based on the record of spills that I
5 was shown as well as the depositions of the coworkers
6 and the management people who were deposed which I
7 interpreted as indicating that -- and it was also
8 based on Mr. Lavender's work records showing his
9 assignment on each day of his work on site.

10 Putting all of those together, it is my
11 impression that there were very few times when
12 Mr. Lavender was present in an area in which there
13 was a spill or in which anyone could actually recall
14 that he was present or involved in any way with a
15 spill.

16 Q And you're relying on the depositions that
17 are listed on that particular exhibit you gave me?

18 A Yes.

19 MR. HARTLEY: Let's mark this as
20 Exhibit 1.

21 (Garabrant Exhibit 1 identified.)

22 BY MR. HARTLEY:

1 Q And this is the list of job functions he
2 had at the particular time he was working at Miles?

3 A Yes.

4 Q And **you** reviewed this.

5 MR. HARTLEY: Do you want to make a copy of
6 this to attach as an exhibit?

7 MR. HOLLINGSWORTH: Sure. We'll get you
a another copy.

9 MR. HARTLEY: That will be 2.

10 (Garabrant Exhibit 2 identified.)

11 BY MR. HARTLEY:

12 Q Did you talk to John Spencer about the
13 exposure in this case?

14 A I have not.

15 Q Have you talked to anyone about exposure
16 personally?

17 A I have not.

18 Q Did you talk to Dr. Irons about this case?

19 A I have not.

20 Q Have you talked to any physicians at the
21 University of Michigan .. are you still at Michigan?

22 A I am.

1 Q Did you talk to any physicians or anyone at
2 the University of Michigan about this case?

3 A No.

4 Q What level do you feel -- I know you
5 answered this before, but I want to refresh my
6 recollection. What's the odor threshold?

7 A It's in the range of a few parts per
8 million.

9 Q And a few to you is what?

10 A Less than 10.

11 Q More than five, less than 10?

12 A I'm not sure that any of us could defend a
13 single number. People's odor thresholds vary. I
14 think in the range of 3 to 10.

15 Q Is there a fatigue factor which is taken
16 into account for exposures of any length of time?

17 A It's a general principle that our ability
18 to smell things fatigues rapidly and we cease to
19 smell things as time goes on, usually within minutes.

20 Q Does that occur on a day-to-day basis? If
21 you are used to smelling something every day and
22 every day and every day, do you become fatigued or

1 just basically used to it is a poor choice of words
2 or immune to it is a poor choice of words, but you
3 recognize it and just keep on going and forget about
4 it?

5 A Well, I think there are two things that
6 happen. It's my understanding that the sensory nerve
7 endings in the nose become -- I don't know if
8 "fatigued" is the right term -- acclimated is
9 probably a better term, to a smell fairly rapidly,
10 but that leaving overnight would allow them to
11 reestablish their sensitivity to the smell.

12 Conversely, I think at the cognitive level,
13 in other words, the brain level, we tend to ignore
14 stimuli that we have become accustomed to and regard
15 as common place. And so while the smell might be
16 there and the nose would be fresh, if it's something
17 we well know, we might simply fail to recognize that
18 it's there.

19 Q Do you have an opinion based upon your
20 review of the material concerning whether there was a
21 benzene smell given off of the trench on a daily
22 basis?

1 A The records indicate that smells from the
2 trench did occur with some frequency. It wasn't
3 clear in my reading of the records that the people
4 who smelled it could always reliably identify what
5 the smell was.

6 Q If they could reliably identify the smell
7 as being benzene, would the fact that there was a
8 continuous odor around the mononitrated benzene unit
9 be significant to you in this case?

10 A Yes.

11 Q Why would it be significant?

12 A Because mononitrobenzene has a smell that's
13 similar to the smell of benzene, and it has an odor
14 threshold down in the parts per million level, so
15 this is a material you smell at very low
16 concentrations, and its smell is not clearly
17 different than benzene to most people.

18 So the fact there's MNB in the area would
19 be noticed at very low exposure levels and many
20 people can't reliably tell the difference between MNB
21 and benzene.

22 Q My question to you, though, was if someone

1 could tell the difference and was able to smell
2 benzene coming from the trench and around the MNB
3 unit on a daily basis, does that have any relevance
4 to this particular case? I want *you* to assume that
5 the person who smells it every day can tell the
6 difference. Does that have any relevance to you at
7 all?

8 A If that were true, it would indicate that
9 at the time the person was out smelling the trench,
10 that there was benzene at or above the odor
11 threshold.

12 Q Is there a latency period, Doctor in your
13 opinion for leukemia to develop from benzene
14 exposure?

15 A Yes.

16 Q What do you believe that to be?

17 A I think that the scientific literature
18 supports the conclusion that latency of 10 years or
19 greater is necessary for leukemia to develop after
20 benzene exposure.

21 Q Is it absolutely necessary for it to take
22 10 years?

1 A Well, I think that's what I just said. The
2 scientific literature does not show evidence of
3 increased risk of leukemia within the first 10 years
4 of exposure.

5 Q And what particular ones establish that you
6 need 10 years? Would you reference me to those
7 articles?

8 A Wong in his chemical workers study.

9 Q Is that Wong 1987-A, 1987-B? That's his
10 latency period?

11 A Yes.

12 Q Anyone else?

13 A Ott also supports that.

14 Q What about the Bond review of Ott? Does
15 that support it?

16 A I don't recall that Bond reexamined the
17 issue. I would have to look at it to see. The Bond
18 study would support that particular --

19 Q What portion of the Bond study are you
20 referencing?

21 A Table 5.

22 Q Table 5?

1 A Yes.

2 Q Three out of every five people have more
3 than 10 exposure; two out of the five of them do not?

4 A I thought we were discussing latency.

5 Q We are. First year of exposure. When does
6 the disease develop, 15, 15 -- yeah, you're right.
7 If you assume the first year of exposure is from then
8 to the year of death, you're going to use the year of
9 death in your determination of latency or when the
10 disease first materialized?

11 A For a mortality study, we have no choice
12 but to base it on death.

13 Q What about Yin's study? Did you look at
14 Yin's study on that particular point?

15 A I don't believe Yin addresses that point.
16 It is not relevant to that issue.

17 Q Did Aksoy address that point that you're
18 aware of?

19 A I'm not aware of any studies in which Aksoy
20 addresses that. The Aksoy studies I have include
21 case reports which are not reliable for a
22 determination on that point and the one PMR study

1 which we have to go back and look at.

2 Q Does toluene have any effect on benzene
3 exposure concomitantly?

4 A I think there's some evidence to suggest
5 that concurrent toluene in benzene exposure alters
6 the metabolism, the metabolic pathway of benzene.

7 Q Resulting in?

8 A You have to forgive me for not knowing
9 this. It's not an area I reviewed. I think it
10 shifts at a way from the pathway that forms
11 hydroquinone and eventually phenol into a different
12 pathway. I'm not confident.

13 Q You didn't consider that aspect in this
14 particular case?

15 A I did not review that topic in preparation
16 for this deposition, no.

17 Q Does the concurrent exposure to benzene and
18 toluene enhance or decrease the toxicity of benzene?

19 A Decreases.

20 Q And what do you base that on, old studies?

21 A I should say -- let me amend that answer.
22 It's probably not fair to say toxicity. It shifts

1 the metabolism down a different pathway. If the
2 leukemogenic effect of benzene occurs through
3 formation of metabolites in the pathway that goes via
4 hydroquinone, then shifting it away from that pathway
5 would be expected to reduce the risk associated with
6 benzene exposure.

7 Q But you're really not aware of the studies
8 on that particular topic?

9 A I do not recall studies on that topic
10 adequately to cite them. I know I have read
11 materials on that topic.

12 Q Do you think it's important for this
13 particular case, in light of the fact that
14 Mr. Lavender was working in the area where they were
15 nitrating toluene as well as nitrating benzene, that
16 there would be a combined effect between the two
17 particular exposures, that you should have looked at
18 that area?

19 A The answer is no. I don't think that that
20 would have changed my opinion, because the basic
21 issue with Mr. Lavender's case is that he was not
22 adequately exposed to think that benzene played any

1 role whatsoever in his leukemia, and the presence or
2 absence of toluene at the levels of benzene exposure
3 he had would not change that in any appreciable way.

4 Q Did you take into account Mr. Lavender's
5 drinking history?

6 A Yes, I did.

7 Q Did that have any factor -- or have any
8 effect on your determination of the myelotoxic effect
9 of benzene that he might have been exposed to?

10 A No.

11 Q Were there any other issues that you took
12 into account to determine whether his exposure to
13 benzene was sufficient to cause his AML?

14 A I reviewed all of his medical records and
15 his depositions and all the materials I was provided
16 about his personal habits, his medical condition, his
17 work assignments, his exposures and the circumstances
18 of his work, and I considered all of those things.

19 Q Did you consider the Carlos epidemiological
20 study to be of any significance?

21 A Yes.

22 Q And why is that?

1 A Well, the Carlos study is the most direct
2 evidence regarding the risk of leukemia in this
3 plant. It's a scientific study looking at the risk
4 of leukemia among people that worked at the Mobay
5 plant.

6 Q When was it done?

7 A It was completed in 1988.

8 Q Did you compare the deaths by either lympho
9 or hematopoietic cancers in the Carlos studies to the
10 answers to interrogatories that Miles gave in this
11 case?

12 A I'm not sure what you're getting at. I did
13 not put them side by side, no.

14 Q Did you try to determine whether, in fact
15 since 1988 to the time they answered the
16 interrogatories, there were more either lymphatic
17 cancers or hemopoietic cancers than what Carlos had
18 disclosed in '88?

19 A Well, it would surprise me if there hadn't
20 been more deaths due to those diseases in a cohort of
21 this size. Those diseases do occur in the general
22 population with a predictable frequency. So if you

1 follow a group of people through time, the longer the
2 time period, the more deaths you see due to each
3 cause of death. So that doesn't surprise me that
4 there would have been additional deaths.

5 Q Did you check the rates that were given in
6 a particular interrogatory answer and try to
7 determine if there was a statistical increase in the
8 amount of deaths from either lymphatic or hemopoietic
9 cancers, **yes** or no? Did you do that?

10 A You'd have to suggest to me in more detail
11 what it is you're asking me -- let me rephrase that.
12 It's not clear to me what it is you're asking me that
13 I did or didn't do.

14 Q Did you look at the interrogatory answers
15 which listed the amount of lymphatic cancers as well
16 as hemopoietic cancers?

17 A Yes.

18 Q Did you do anything with that data, other
19 than look at it and say here's what happened?

20 A No, I did not. If I can add, there wasn't
21 anything that could be done with the data in the
22 interrogatories.

1 Q When you look at carcinogenic risk do you
2 assume it operates multiplicatively or additively, in
3 your own opinion?

4 A I don't actually assume either. That is
5 usually one of the reasons we do epidemiology, to
6 determine whether it acts to add to risk or to
7 multiply the underlying risk.

8 Q Did you determine in your own opinion what
9 benzene does?

10 A The epidemiologic literature, I don't
11 think, is adequate to comment on whether it's a
12 multiplicative or additive risk. There are certain
13 methods in analyzing the data that assume
14 multiplicative risk, so some of the discussions we
15 discuss make that assumption, and it's probably a
16 reasonable assumption.

17 Q Specifically which ones?

18 A Paxton, Rinsky and actually, I guess -- no,
19 I guess that's it.

20 Q Why didn't you rely on the Rinsky and
21 Infante studies for your cumulative dose
22 requirements?

1 A Because I think they have systematically
2 underestimated the exposures.

3 Q Do you think there is a nonlinear or linear
4 dose response relationship between benzene and
5 leukemia?

6 A The best data at this point suggests that
7 there is increased risk above 50 parts per million
8 years and there is no evidence of increased risk
9 below 50 parts per million years. Those two
10 observations are consistent with a nonlinear dose
11 response relationship.

12 Q Have you ever treated someone with
13 leukemia?

14 A Yes.

15 Q Have you ever treated anyone with leukemia
16 who had been exposed to benzene?

17 A Not to my recollection, but I'm not sure I
18 would know at this point.

19 Q If we assume -- did you read Dr. Rose's
20 deposition?

21 A Vern Rose?

22 Q Yes.

1 A Yes.

2 Q Do you have any quarrels with any of his
3 statements, or did you disagree with any of his
4 opinions, other than the odor threshold, which I'm
5 aware of?

6 A I'd have to go back through it.

7 Q Did you make any notes on that?

8 A No, I did not.

9 MR. HOLLINGSWORTH: I object to that
10 question on the ground that it's overbroad. And
11 without reviewing the deposition in detail now or
12 beforehand, it's not a fair question. Maybe if you
13 could state what his opinions are and ask the doctor
14 whether he agrees or disagrees with them, that would
15 be a more efficient way to proceed on this issue.

16 BY MR. HARTLEY:

17 Q Did you make any highlighted pages in
18 Dr. Rose's deposition? Did you highlight any pages?

19 A I don't recall whether I did or not.
20 There's a fairly easy way to find out.

21 (Witness reviewed the document.)

22 I did.

1 Q What did you highlight?

2 A On page 81, I highlighted "to me the
3 description of these two odors are quite different
4 and therefore, I would believe that they would appear
5 to most people to be different odors." I actually
6 disagree with that.

7 Q Have you smelled mononitrated benzene?

8 A Yes, I have.

9 Q Where did you smell it?

10 A I used to smell it cleaning guns.

11 Q And have you smelled benzene?

12 A Yes, I have.

13 Q And it's your opinion they smell alike?

14 A It's my opinion they smell enough alike
15 that most people would have difficulty naming which
16 one they were smelling.

17 Q Would that be true for someone who has
18 worked in the area 20 years?

19 A I think that someone who has trained
20 himself to recognize smells could reliably
21 distinguish between them, but someone who is not
22 trained to do that probably could not.

1 Q Do you have any way to refute
2 Mr. LeMasters's statement that he could tell the
3 difference between benzene and MNB?

4 A I have no way to refute such a statement.

5 Q You haven't met with Mr. LeMasters, have
6 you?

7 A No.

8 Q Have you put benzene and mononitrated
9 benzene under his nose?

10 A No, I have not.

11 Q Other than your own conclusion that most
12 people wouldn't, there's really no way to refute that
13 Mr. LeMasters is able to differentiate between the
14 two?

15 A I have no way to refute that, no.

16 Q If we assume that Dr. Rose's assessment of
17 61 parts per million any time you could smell it
18 occurred on a daily basis for Mr. Lavender, as he
19 described in his deposition, would that be sufficient
20 in and of itself to cause his -- or to be associated
21 with his AML?

22 MR. HOLLINGSWORTH: Objection. Lack of

1 foundation. Misstates the evidence.

2 BY MR. HARTLEY:

3 Q That means you can answer, Doctor.

4 A First off, I think there are good reasons
5 to think that the exposure levels were not that high
6 and that Mr. Lavender couldn't tell what he was
7 smelling, so the characterization that the exposure
8 levels were 61 parts per million is contradicted by
9 the evidence that I read in this case.

10 Q Well, let's assume that a jury in Marshall
11 County accepts the fact that Mr. Lavender smelled
12 benzene every day, and let's further assume that this
13 jury accepts the fact that Dr. Rose will testify that
14 when you smell it at 61 parts per million.

15 Given those two set of circumstances, do
16 you have an opinion with reasonable medical
17 probability as to whether Mr. Lavender's acute
18 monocytic leukemia would be causally associated with
19 that exposure?

20 MR. HOLLINGSWORTH: Same objection.

21 BY MR. HARTLEY:

22 Q Sure. I'm just asking you to assume that.

1 I don't want you to change it for me. I want you to
2 assume that fact.

3 A If I were to accept those assumptions,
4 which I do not --

5 Q I understand that.

6 A -- it would still be my opinion that
7 Mr. Lavender's exposure to benzene at the Mobay plant
8 did not cause his leukemia.

9 Q Have you attempted to calculate his parts
10 per million years?

11 A I've made some mental calculations of that.

12 Q And in your opinion, what would that be?

13 A In my opinion, his first exposure to
14 benzene was in the 1990 to '92 employment period, and
15 that out of the 6000 hours he worked in that period,
16 the records indicate he was only in MNB for 48 hours
17 and in ECD for 208. And so even if the exposures
18 were at 60 parts per million while he was exposed,
19 with which I disagree, his parts per million years
20 would still be far below that that is known to cause
21 leukemia.

22 Q Did you see in the depositions that the

1 operators at ECD were complaining about solvent
2 smells?

3 A I do remember that there were complaints of
4 solvent smells from the trenches and from the
5 wastewater, and I believe in ECD as well.

6 Q Did you notice that Mr. Myers, who was the
7 manager for ECD, testified about the content of
8 benzene or the amount of benzene in the wastewater?
9 Do you remember that?

10 A I do not recall that specific issue.

11 Q Do you remember him testifying that they
12 had received on an average 50 pounds per day?

13 A I don't recall.

14 Q Have you seen any documents that would
15 support that?

16 A Did I see any documents that would support
17 that 50 pounds per day of benzene went into ECD?

18 Q Uh-huh.

19 A I don't recall those documents. I would be
20 happy to look.

21 Q If we assume that to be true, I just want
22 you to assume it for right now, that ECD received on

average 50 pounds per day of benzene every day from
2 1980 through 1993. Is that a significant amount of
3 benzene coming to waste?

4 MR. HOLLINGSWORTH: Are you basing this
5 question on the trench monitoring data? Is that what
6 you're referring to?

7 MR. HARTLEY: I'm asking him to assume that
8 there's 50 pounds of benzene making its way every day
9 on average to ECD.

10 THE WITNESS: I don't know how to answer
11 your question, is that significant. Whether that has
12 any relevance to human exposure isn't clear. It
13 depends on whether that wastewater stream is enclosed
14 or whether it's open. It depends on the temperature
15 of the water. It depends on how the water is handled
16 and where the people are.

17 If 50 pounds a day reached ECD and if all
18 50 pounds a day evaporated into the air, there would
19 be 50 pounds of benzene vapor in the air every day.
20 It would also matter what the wind velocity was and
21 wind direction and general ventilation. And so the
22 relevance of that number to human exposure is not

1 clear.

2 BY MR. HARTLEY:

3 Q Do you understand the ECD process at Miles?

4 A I am not familiar with the details of the
5 ECD process.

6 Q Do you have an understanding as to how the
7 contaminants that are in the water are removed prior
8 to induction into the organism pool?

9 A I do not know that specifically at the
10 Miles plant.

11 Q Will you know that by the time we go to
12 trial?

13 A I suspect I will.

14 Q That's another thing you ought to be
15 looking at, then.

16 Can you calculate for me, Doctor, if 50
17 pounds of benzene is making it to the ECD per day,
18 how much is being spilled in the MNB unit? Is that
19 calculation possible?

20 A I cannot make that calculation.

21 Q You don't have the training to do it, or
22 you don't have sufficient information to do it?

1 A I d n't ha e sufficient information to do
2 it, and I'm not sure whether I have the training to
3 do it.

4 Q Would you agree with me that if a certain
5 amount of benzene was spilled into the trench at MMB,
6 that a portion of that would evaporate by the time it
7 made its way almost a half a mile down the road to
8 ECD under normal conditions?

9 A It depends on the construction of the
10 trench and whether it's a closed stainless steel pipe
11 and what the fire traps and grates look like and how
12 much of it is caught and removed in the course of
13 going. It depends on wind velocity and temperature.
14 It depends on lots of things.

15 Some of it would certainly evaporate, but
16 the amount I cannot predict. It would take someone
17 with different credentials than mine.

18 Q What do you understand this trench to be?
19 Can you describe it for me?

20 A My recollection is there was a trench
21 system that ran throughout the plant that basically
22 had a branch that originated near or in each or most

1 of the buildings and that it flowed south to the ECD
2 area, which was at the south end of the plant.

3 The actual description of it is not
4 entirely clear to me. I had the impression in some
5 of the materials that I read that it was an open
6 trench. I had the impression that some areas were
7 covered by a grate, and I recall reading some
8 materials that talked about a stainless steel pipe,
9 and I do not know how all of that fits together, to
10 be quite honest with you.

11 Q Have you tried to determine how it fits
12 together?

13 A I have not.

14 Q Do you think that it would matter, the type
15 of trench system that was being operated through the
16 Miles facility when you attempt to determine benzene
17 exposure, assuming benzene to be in the trench?

18 A I think that the physical construction of
19 the trench system and the rate of water flow and
20 temperature and ventilation and many such factors
21 certainly will have an effect on the benzene
22 concentration.

1 I do not represent myself to be an
2 industrial hygienist or an environmental engineer,
3 and I will rely on the expert opinion of other
4 witnesses in this case for determination of what the
5 exposure levels were along the trench.

6 Q Does that mean that you'll rely on
7 Dr. Rose's evidence or testimony or only on
8 Mr. Spencer's?

9 A I will certainly read Dr. Spencer's and
10 have read Dr. Rose's and will rereview his to make my
11 own determination of what exposures I think are
12 relevant to Mr. Lavender.

13 Q Do you think there is enough air monitoring
14 at the Miles facility to calculate cumulative
15 exposure?

16 A The answer is we always want more exposure
17 samples than we have. I've been involved in studies
18 where we've had thousands of measurements and still
19 wanted and needed more.

20 In the real world, it is clear that there
21 have been hundreds of benzene measurements made at
22 Miles, and it's my impression that they are adequate

1 to come up with an estimate of Mr. Lavender's
2 exposure or a range of estimates that we can be
3 confident of. The issue of having more samples would
4 allow us to have greater precision in the estimate,
5 but nonetheless, there are many samples that I
6 believe are relevant to Mr. Lavender.

7 Q Did you determine what his cumulative parts
8 per million years were?

9 A I think we've already discussed that --

10 Q We may have. I just forgot.

11 A My answer was I made mental calculations.
12 I didn't give you a number.

13 Q I didn't think you did.

14 A My recollection is we started discussing it
15 and moved on. His exposures were in the range of .01
16 to a few tenths of parts per million with rare
17 excursions into the parts per million range, and his
18 duration of exposure based on his work records
19 appears to have been in the range of a few hundred
20 hours.

21 The product of .01 parts per million times
22 .1 working years, which would be the low end of my

1 estimate, would be .001 parts per million years. The
2 upper end of my estimate would be .3 parts per
3 million times perhaps .2 years, which would be .06
4 parts per million years.

5 Q What did you base those calculations on?

6 A I based those calculations on the still
7 hygiene measurements we've discussed, which showed
8 many samples in the range of .01 to, as I said, a few
9 tenths of a part per million, which I used as .3 in
10 these calculations, so parts per million between .01
11 and .3 and years of exposure between .1 and .2.

12 So the product of .01 times .1 is .001.
13 That's the low end of my estimate. The product of .3
14 PPM times .2 years is .06 parts per million years.
15 That's the high end of the estimate.

16 Q Did you take into account any peak
17 exposures?

18 A Yes, I did.

19 Q What peak exposures did you take into
20 account?

21 A As we've already discussed, it's my
22 understanding from Mr. Lavender's work records and

1 descriptions in the depositions and the industrial
2 hygiene monitoring that he was rarely exposed to
3 spills or unusual occurrences. So even if he had a
4 few peaks in the parts per million range, it would
5 not appreciably alter his total parts per million
6 years.

7 Maybe I can add to that. For example, if
8 he worked at 10 parts per million for a full day, a
9 full day is 1/2000 of a working year, so you can add
10 to my estimate .001 times 10, which would be .01. It
11 would only trivially change my upper limit estimate.

12 Q Did you take into account the fact that
13 Miles routinely did not record samples in the MNB
14 area?

15 MR. HOLLINGSWORTH: Objection. I don't
16 think that accurately states what the record is.

17 BY MR. HARTLEY:

18 Q Let's put it this way then. Did you take
19 into account Kyle Earley's testimony that many times
20 they did not record the monitoring of benzene in the
21 area of MNB?

22 A I am aware that there were instances in

3 which a measurement was made and the results were not
4 written down, if that's what you're referring to.

5 Q Yes. Did you take that into account in
6 your calculation of parts per million years?

7 A The answer is -- the best estimate of a
8 worker's exposure is based on full shift samples that
9 are made for surveillance purposes, not based on
10 brief grab samples or short duration samples that are
11 made for the purposes of characterizing excursions or
12 determining compliance with the law.

13 And therefore, the large number of samples
14 that represent full shift time weighted averages I
15 regard as far more reliable estimates of his real
16 exposure than do measurements made during excursions
17 and accidents where there were very brief peaks that
18 did not represent the usual working conditions.

19 Q In your opinion, how long would 2000 pounds
20 of benzene need to be exposed to the air to be
21 completely back below the OSHA level?

22 MR. HOLLINGSWORTH: Excuse me, 2000 pounds
of benzene from a 55-gallon drum or from some
container or from the trench?

1 BY MR. HARTLEY:

2 Q Let's assume that we have 2000 pounds of
3 benzene escaping from the water column. How long
4 would it take that 2000 pounds -- first of all, what
5 is your estimate of the parts per million that would
6 be in the area for the 2000 pounds?

7 A There's no meaningful way to answer that
8 question.

9 Q Do you think it would be pretty high?

10 A There's no meaningful way to answer the
11 question as it's asked.

12 Q Why not? What are you lacking that would
13 help you answer that question?

14 A I'm capable of calculating the
15 concentration of benzene in air provided I know the
16 volume of air we're talking about, and you've given
17 me no sense for the volume of air we're talking
18 about.

19 The area, as you've stated it, I don't know
20 whether you're talking about the area around the
21 entire Mobay facility, the area in Atrium, West
22 Virginia, the area around the MNB plant, the area

1 around ECD, the air inside a building.

2 If you release 2000 pounds of benzene in
3 this room, the exposure would be very, very high. In
4 fact, you probably couldn't vaporize it. So I can't
5 answer the question as it's asked.

6 Q The one area you forgot was the entire
7 state of West Virginia when we're dropping 2000
8 pounds. If we dropped 2000 pounds into the trench,
9 open air trench, what would the levels be
10 approximately when you're standing 5 feet from the
11 trench?

12 A I have no way to estimate that based on the
13 information that you've postulated.

14 Q Have you looked at those releases, the
15 releases that Mr. Hollingsworth or whomever showed
16 you yesterday, the spills?

17 A The amount spilled?

18 Q Yes.

19 A Yes, I have.

20 Q Have you attempted to calculate any level
21 of benzene that would have been released during those
22 spills?

1 A The answer is I have not, nor do I think
2 that I could do that reliably without knowing
3 temperature, wind velocity, wind direction. And
4 those calculations could be better made by someone
5 with different credentials than mine.

6 Q What is your little paper there that you
7 keep referring to?

8 A This is my notes on the chronology of
9 events in Mr. Lavender's life.

10 Q Can we mark that as an exhibit? Is that
11 your only copy? I don't want to take your only
12 copy. Yes, it is your only copy or yes, we can mark
13 it?

14 A It is my only copy, but it's on disk. I
15 can generate another one.

16 MR. HOLLINGSWORTH: We'll copy it for you.

17 MR. HARTLEY: Do you want to mark that
18 as 3.

19 (Garabrant Exhibit 3 identified.)

20 BY MR. HARTLEY:

21 Q Anything else you brought with you, Doctor?

22 A I brought everything else I was instructed

1 to bring in that folder.

2 Q This is your notes on your studies ..

3 A Yes.

4 Q -- that you reviewed in addition to
5 whatever else was in there?

6 A Those are my notes on the studies I
7 reviewed, except for some that I have recently added,
8 which are not listed on that page.

9 Q But which are in your three-ring binder?

10 A Yes.

11 Q This is an extra copy of this?

12 A That's the only one I can put my hands on
13 at the moment.

14 MR. HARTLEY: Can we make a copy of this?

15 MR. HOLLINGSWORTH: Yes.

16 BY MR. HARTLEY:

17 Q Doctor, I don't want a copy of all your
18 articles there. If you would go through the articles
19 and tell me what the titles are for the record and
20 what the cites are so I know what you're utilizing at
21 this juncture in the case.

22 A Could I suggest that I would leave these

with Mr. Hollingsworth and that they could do that or
2 copy the face pages.

3 Q That will be fine.

4 MR. HOLLINGSWORTH: There are dozens and
5 dozens of papers that you're referring to there.
6 It's probably a faster way to do it.

7 MR. HARTLEY: Whichever way you think is
8 quicker, Doctor.

9 THE WITNESS: Either way is fine with me.

10 BY MR. HARTLEY:

11 Q How many documents are in these total so --
12 I'm not suggesting that he would intentionally not
13 give me all the copies, but just so I know that there
14 wasn't a mistake made or something like that. How
15 many articles are in both these volumes?

16 A I do not know. I would estimate -- here.
17 This will list everything except the ones that have
18 yellow tags and the ones that are these. So this
19 plus approximately -- because there are probably a
20 couple that aren't tagged -- I'd say that with
21 roughly a dozen more would be about right.

22 Now, I know a couple of these are tagged

1 that are on that list. So it's th t plus about a
2 dozen.

3 Q Do you have an opinion as to what is the
4 cause of Mr. Lavender's leukemia?

5 A My opinion is that the vast majority of
6 leukemia arises for reasons that are yet to be
7 discovered and Mr. Lavender is one of those
8 unfortunate cases.

9 Q So what you're basically saying is you
10 don't know?

11 A What I'm saying is that I do not think that
12 we .. there's no evidence that his leukemia arose as
13 a result of any of the established risk factors for
14 that disease with the exception of smoking, which I
15 think increased his risk in a small way.

16 Q Outside of the increased risk, you don't
17 know what really caused his cancer? I know you've
18 said it three different ways. I just want to know
19 yes or no, if we subtract the increased risk of
20 leukemia from smoking, you do not know what caused
21 his leukemia, yes or no?

22 A It's not a question that can be answered in

1 that manner. The question presupposes there is a
2 single cause and that is not the way most, if any,
3 human disease arises. Almost every disease we've
4 studied has multiple causes and the contributions of
5 a number of causes are important in the causation of
6 each case.

7 In Mr. Lavender's case, we've identified
8 smoking as a factor that led to his leukemia,
9 although admittedly it's not a strong factor. The
10 other causes of his leukemia are not known to me or
11 to medical science.

12 Q He didn't have chemotherapy that caused it,
13 did he?

14 A Not to my knowledge.

15 Q He didn't have radiation therapy that
16 caused it?

17 A He did not have radiation therapy.

18 Q That caused his leukemia?

19 A That's correct.

20 Q He was not exposed to the atomic fallout in
21 Hiroshima?

22 A Not to my knowledge.

1 Q Or Nagasaki?

2 A Not to my knowledge.

3 Q He was not at the testing ground at
4 Los Alamos, was he?

5 A Not to my knowledge.

6 Q According to his own deposition, he was not
7 exposed to electromagnetic fields because when he was
8 working on the towers, they were deenergized. They
9 had not even been energized at all, so he would not
10 have been exposed to electromagnetic fields, would
11 he?

12 A I suspect that's an area where his
13 perceptions are simply wrong. Electricians are
14 commonly exposed to substantial electromagnetic
15 fields in the course of their work.

16 Q Do you have an opinion that his exposure to
17 electromagnetic fields caused his leukemia?

18 A No, I do not.

19 Q Do you feel there is sufficient evidence in
20 the epidemiological literature to support that
21 relationship?

22 A No.

1 Q His parents weren't known to have a
2 genetically induced leukemia, were they?

3 A No.

4 Q So we've eliminated just about everything
5 except for his smoking, correct, because we know you
6 don't think he had sufficient exposure to benzene.
7 So the only thing we can point to is the .31 to .5
8 elevation in smokers of developing leukemia, correct,
9 that you supported with the Segal study, and that's
10 the only thing we can put our finger on?

11 A Again, that has to be said in the right
12 context. We do not know the causes of leukemia in
13 the vast majority of instances and so for
14 Mr. Lavender, although we know that smoking is a
15 cause, he basically falls in the vast majority of
16 cases of leukemia where we are unable to identify
17 what caused it.

18 Q Did you review anything else, Doctor, that
19 we haven't discussed today?

20 A I do not believe so.

21 Q Would you read for me what your notes are
22 that you're going to do before trial.

1 MR. HOLLINGSWORTH: What he has written
2 down that he may do before trial is not necessarily
3 what we, on behalf of Miles or Miles's attorneys,
4 would ask him to do in connection with preparing his
5 testimony for trial.

6 These are just a few things that you have
7 asked him about, and you have asked him whether he
8 would perform certain tasks before trial or not, and
9 he said that he would. I just want to make clear
10 what he has been asked to do he has done and he has
11 given you an opinion today in accordance with what I
12 believe our 26(b)(4) statement was as to what his
13 opinion would be.

14 He's provided you with, I think, at least
15 in my opinion, what all the bases for his opinion are
16 so these other things that you have asked him,
17 whether or not he would do or look at before trial
18 are things that he has taken down on his own
19 initiative, at your suggestion, really. It's been by
20 your suggestion.

It's certainly not clear to me that any one
of those things, certainly not all of them, are

1 things that we would ask him to do in advance of his
2 testimony at trial and certainly not any one of those
3 things or all of them are necessary we believe or we
4 believe the court would find to support his opinion
5 at trial.

6 But having said that, you can ask him what
7 those things are.

8 MR. HARTLEY: Thank you.

9 MR. HOLLINGSWORTH: Or we could make a copy
10 of the list.

11 MR. HARTLEY: I'd rather him read it into
12 the record.

13 BY MR. HARTLEY:

14 Q Would you make a list and tell me what you
15 have written down there on your piece of paper?

16 A I've written down I need to get the Crump
17 article that we've already discussed. I'm interested
18 in reviewing whether benzene causes either leukopenia
19 or lymphocytopenia in the absence of pancytopenia.
20 I'm interested in reviewing the issue of whether
21 benzene is mutagenic. I'm interested in reviewing
22 the mechanisms by which benzene causes leukemia.

1 I've written down something about the Wong
2 1983 comments on NIOSH that were not in the open
3 literature, but I don't intend to do anything about
4 that since I have no access to those. I've written
5 down to get a copy of the Travis update of the Yin
6 study.

7 I've written down to calculate the benzene
8 exposure from cigarettes for Mr. Lavender. I've
9 written down the same, Gene McDonald at NIEHS with
10 the intention of trying to find out what that person
11 has published relevant to this issue.

12 I've written down the name Maltoni,
13 although I don't think I'm going to do anything about
14 it. I know a good deal of Dr. Maltoni's literature
15 and have it. I don't think it's terribly important
16 to this issue.

17 I've written down "review ECD process," and
18 I've written down in my estimate of parts per million
19 years between .001 and .06.

20 Q Have you ever acted as an occupational
21 physician for a corporation, in-house?

22 A No.

1 Q You've written on occupational medicine and
2 workers' right to know, haven't you, at least a
3 chapter in some book?

4 A Yes, I've written on communication in the
5 Handbook of Occupational Medicine by Robert
6 McCunney. I don't think the title is quite right.

7 Q '93 or '94 book?

8 A '94.

9 Q Did you read in the depositions where the
10 only access Mr. Lavender had to hazards associated
11 with the Miles job site was the fact that he was told
12 about a phosgene MSDS and there were other MSDSs
13 available if they wanted to review that? Have you
14 reviewed that?

15 MR. HOLLINGSWORTH: Objection;
16 mischaracterizes the record.

17 BY MR. HARTLEY:

18 Q That means you can answer.

19 A My recollection of that issue is the
20 testimony indicated that Miles had supplied a
21 complete set of MSDSs to Mr. Lavender's employer and
22 Mr. Lavender's employer was responsible for health

1 and safety training.

2 Q If Miles knew that the employer was not
3 following through with health and safety training,
4 did they have an obligation in your opinion to inform
5 Mr. Lavender, either through signs in the areas
6 concerning respirators being needed or through safety
7 meetings or handouts, about the hazards associated
8 with the various chemical processes?

9 MR. HOLLINGSWORTH: Objection. Calls for a
10 legal conclusion.

11 BY MR. HARTLEY:

12 Q That means you can answer.

13 A I'm not a lawyer. I do not know the OSHA
14 regulations in adequate detail to answer that.

15 Q From a purely industrial setting, as a
16 medical physician, occupational physician, would it
17 be good occupational medicine practice to inform all
18 employees, whether they be your employees or someone
19 else's employees, on the job site of the hazards
20 associated with that particular facility?

21 MR. HOLLINGSWORTH: I object to the form.
22 Same objection on the requirement for a legal

1 conclusion.

2 BY MR. HARTLEY:

3 Q Do you understand the question, from an
4 occupational medicine standpoint?

5 A I thought I did until you said that. I
6 think I understand the question. While I would
7 certainly agree that everyone at work should
8 understand all of the hazards at work fully, it is
9 extremely difficult to achieve that in the real world
10 and that there are regulations written to ensure that
11 that is -- that a system is built to do that.

12 It's my understanding that Miles did its
13 part by providing the MSDSs and that it was
14 Mr. Lavender's employer's responsibility to see that
15 he was appropriately trained in terms of the hazards
16 that were present at Miles and in terms of the
17 hazards that were uniquely present in the course of
18 being an electrician.

19 Q What is the basis for your opinion that his
20 employer was solely responsible for that training?

21 A My recollections of the OSHA regulations.

22 Q Are there any other opinions that we have

1 not talked about today that you might testify to at
2 trial?

3 A I cannot think of any.

4 MR. HOLLINGSWORTH: I object to that
5 question. How can he answer that? He doesn't know
6 what he's going to testify to at trial. The trial
7 isn't here yet.

8 You can ask him about his opinion and the
9 bases of his opinions and anything we've offered in
10 the Rule 26(b)(4) statement, but when it comes to the
11 trial, for him to testify at trial, a lot will come
12 before him, I'm sure.

13 MR. HARTLEY: That's a typical question.
14 I've been through 10,000 depositions.

15 BY MR. HARTLEY:

16 Q Is there any other opinion, Doctor, that
17 you haven't --

18 MR. HOLLINGSWORTH: But you haven't heard
19 me ask that.

20 MR. HARTLEY: You're right. I haven't. I
21 don't think I have anything else, Doctor, that I can
22 think of anyway.

1 Do you want to read and sign the
2 deposition -- we better mark this as number 4.

3 (Discussion off the record.)

4 MR. HOLLINGSWORTH: It appears,
5 Mr. Hartley, that the number of papers that he's
6 relied on which are contained in these two black
7 binders that have been present here in this
8 deposition room is about 100.

9 MR. HARTLEY: I don't want the entire
10 articles now.

11 MR. HOLLINGSWORTH: I understand that. You
12 want the names of the articles. And what we'll
13 endeavor to do is put those on a list for you, which
14 I realize it's not our duty to do. It's yours.
15 We'll do that anyway to save time, and I assume
16 you'll just return the favor to me sometime when you
17 get a chance.

18 MR. HARTLEY: I always try to.

19 MR. HOLLINGSWORTH: Okay.

20 MR. HARTLEY: If we go to trial, will you
21 bring these two notebooks with you?

22 THE WITNESS: Probably.

1 MR. HARTLEY: Okay. That's fine.
2 (Whereupon, at 2:00 p.m., the deposition
3 was concluded.)

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DAVID H. GARABRANT

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2
3
4
5
6
7
8
9
10
11
12
13
14
15
16
17
18
19
20
21
22

CONTENTS

WITNESS

EXAMINATION

David H. Garabrant

by Mr. Hartley

4

EXHIBITS

NT DEP I NU

IDENTIFIED

Exhibit 1 - List of materials reviewed

in John D. Lavender v. Miles,

Inc. by David Garabrant

120

Exhibit 2 - 1980 Work history of John D.

Lavender, Jr. from Sargent

Electric Company Time Sheets

120

Exhibit 3 - Notes on chronology of events

in John D. Lavender's life

151

Exhibit 4 - Handwritten document

165

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I, JULIE BAKER, the officer before whom the foregoing deposition was taken, do hereby certify that **the** witness whose testimony appears in **the** foregoing deposition was duly sworn: that the testimony of said witness was taken in shorthand and thereafter **reduced** to typewriting by me or under my direction; that said deposition **is a true record of** the testimony given by said witness; that I am neither counsel **for**, related to, nor employed by any **of** the parties to the action in which this deposition **was** taken: and, further, that I am not a relative or employee of any attorney or counsel **employed by** the parties hereto, nor financially or otherwise interested in the outcome of this action.


Notary Public in and for the
District of Columbia

My Commission Expires **SEPTEMBER 30, 1997**

**Look-See
Concordance Report**

UNIQUE WORDS: 1,913
TOTAL OCCURRENCES:
8,340
NOISE WORDS: 385
TOTAL WORDS IN FILE:
24,981

**SINGLE FILE
CONCORDANCE****CASE SENSITIVE****NOISE WORD LIST(S):
NOISE.NOI****INCLUDES ALL TEXT
OCCURRENCES****DATES ON****IGNORES PURE
NUMBERS****POSSESSIVE FORMS
ON****- DATES -**

August [1]
52:2
July [1]
32:6
March 4, 1986 [1]
52:15
May [1]
41:14
May of 1994 [1]
16:21
November [1]
32:5
October 17, 1994 [1]
104:12

- \$ -

5350 [1]
104:4

- 1 -

1/2000 [1]
147:9
15th [1]
41:14
1987-A [1]
125:9
1987-B [1]
125:9

- 2 -

2:00 [1]
166:2

- 3 -

3-1/4 [1]
110:5

--- 4 ---
40s [1]
61:11
--- 5 ---
50-some [1]
60:4
55-gallon [1]
148:21
--- A ---
ability [3]
71:20; 101:11; 121:17
able [2]
124:1; 136:13
absence [6]
25:21; 35:20; 62:17;
67:10; 129:2; 159:19
absent [1]
100:10
absolute [1]
69:7
absolutely [3]
68:10; 17; 124:21
absorbed [1]
49:6
abstract [3]
84:16; 85:12; 109:3
accept [2]
58:7; 138:3
acceptable [1]
71:17
accepts [2]
137:11, 13
access [2]
160:4; 161:10
accidents [1]
148:15
acclimated [1]
122:8
accompany [1]
115:14
accordance [11]
158:11
According [1]
156:6
according [2]
85:12; 107:20
account [8]
121:16; 129:4, 12;
146:16, 20; 147:12,
19; 148:3
accumulates [1]
49:9
accumulation [2]
48:22; 49:4
accumulative [1]
111:8
accurately [3]
55:9; 69:10; 147:16
accustomed [1]
122:14
achieve [1]
163:9
act [2]
34:13, 16
acted [1]
160:20
acts 111

132:6
actual [1]
28:3
Acute [2]
14:9; 17:17
acute [26]
14:7, 18; 16:14, 17,
18, 20, 22; 17:6; 19:1,
8, 13; 27:17; 37:21;
39:8; 42:15; 57:14;
64:18; 90:20; 95:16,
18, 21; 96:1, 4, 7, 16;
137:17
add [7]
6:20; 46:21; 76:22;
131:20; 132:6; 147:7,
9
added [5]
4:17, 18, 19; 103:20;
152:7
addition [6]
24:17; 46:15; 68:3;
92:12; 105:16; 152:4
additional [1]
131:4
additionally [1]
78:15
additive [1]
132:12
additively [1]
132:2
address [1]
126:17
addresses [3]
49:18; 126:15, 20
adequate [20]
8:16, 19, 22; 17:7;
18:6, 7, 17, 22; 19:4,
5; 40:5; 45:4; 65:3;
75:13; 79:4; 97:13;
100:3; 132:11;
144:22; 162:14
adequately [5]
39:11; 40:7; 72:3;
128:10, 22
admittedly [1]
155:9
advance [1]
159:1
affect [1]
36:1
affected [2]
99:19, 22
affects [3]
25:10; 51:1; 100:4
age [1]
61:5
agent [4]
24:21; 26:8, 10; 54:1
agents [1]
53:12
agree [9]
13:11, 14, 17; 14:7;
29:22; 44:11; 52:22;
142:4; 163:7
agreement [1]
60:22
agrees [1]
134:14
air [17]
10:15; 11:8; 13:12;

49:13, 15; 80:17;
115:11; 116:10;
140:18, 19; 144:13;
148:18; 149:15, 16,
17; 150:1, 9
Aksoy [6]
49:21; 54:17; 94:16;
126:17, 19, 20
Aksoy's [3]
54:19, 20; 56:11
al [1]
16:9
Alamos [1]
156:4
alcohols [1]
5:18
Alderson [2]
46:5, 7
all-encompassing [1]
68:9
allegedly [1]
34:9
allow [2]
122:10; 145:4
alone [2]
67:10; 76:12
alter [2]
116:18; 147:5
alters [1]
127:5
Alto [1]
35:10
amend [1]
127:21
American [2]
81:1; 94:6
AML [4]
18:18; 36:17; 129:13;
136:21
amount [15]
10:13; 49:6; 50:1;
76:13; 92:22; 114:7;
117:19; 131:8, 15;
139:8; 140:2; 142:5,
16; 150:17
analyses [5]
34:5; 73:1; 94:17;
111:4, 15
Analysis [1]
15:18
analysis [5]
39:15; 75:18; 88:15;
109:2; 111:1
analyze [1]
108:18
analyzed [1]
116:10
analyzing [2]
72:20; 132:13
anemia [2]
25:13; 54:12
animal [10]
26:17, 18; 28:4;
42:20; 43:3, 8, 9, 12,
16; 50:9
Answer [3]
17:13, 17, 19
answer [48]
17:13, 15; 22:5;
24:11; 27:19; 44:4,
11; 46:16; 49:3;

50:14; 55:16; 57:18,
19; 58:6, 7, 8; 63:10,
17; 66:20, 21; 67:13;
68:20; 70:2; 75:12;
76:4, 22; 78:21;
93:11; 101:8; 102:18;
127:21; 128:19;
131:6; 137:3; 140:10;
144:16; 145:11;
148:5; 149:7, 10, 13;
150:5; 151:1; 161:18;
162:12, 14; 164:5
answered [7]
17:13; 57:17; 66:16;
76:3; 121:5; 130:15;
154:22
answering [2]
55:22; 79:18
answers [3]
105:6; 130:10; 131:14
anybody [1]
38:22
anyway [4]
33:12; 43:21; 164:22;
165:15
Anywhere [2]
114:2, 6
anywhere [1]
18:15
API [6]
65:18, 22; 66:2, 5;
81:15; 82:12
aplastic [1]
25:13
apologize [3]
6:20; 29:10; 79:19
appear [1]
135:4
appears [7]
59:16; 111:4, 14, 15;
113:16; 145:19; 165:4
applicable [1]
78:10
apply [6]
11:14; 74:19, 21;
75:1, 3, 4
appreciable [4]
85:5, 14, 15; 129:3
appreciably [5]
34:7; 79:7; 114:15;
116:18; 147:5
appreciation [1]
37:8
approach [1]
72:18
appropriate [7]
26:17; 41:3; 72:21;
87:1, 3; 89:15
appropriately [1]
163:15
Approximately [1]
85:10
approximately [3]
84:21; 150:10; 153:19
area [46]
5:15; 8:1, 11, 14, 20,
21; 9:5; 26:13; 27:7;
37:1, 3, 7, 15, 18;
39:19; 41:12; 51:7;
113:17, 21, 22; 114:9,
13, 15, 19, 20; 115:2,

5, 7; 118:12; 119:12; 123:18; 127:9; 128:14, 18; 135:18; 143:2; 147:14, 21; 149:6, 19, 20, 21, 22; 150:6; 156:12	128:5; 136:10; 137:18, 161:0; 162:7, 20	averages [1] 148:12	92:18; 97:17; 100:16; 106:15; 108:18; 118:18; 124:16; 126:15; 135:4; 139:5; 145:6; 157:20; 158:12; 159:3, 4	Bernard [1] 103:8
areas [7] 7:18; 8:4, 12; 113:16; 114:11; 143:6; 162:5	Associates [1] 52:2	aware [17] 12:10; 21:14, 21; 36:10; 65:11; 91:10; 11; 100:12, 18; 101:18, 19; 102:2; 126:18, 19; 128:7; 134:5; 147:22	benefit [1] 113:4	bias [1] 65:20
aren't [4] 44:12; 56:19; 90:9; 153:20	associates [1] 63:12	awkward [1] 112:21	Benzene [7] 24:22; 29:19; 40:20; 89:17; 92:17; 100:2; 101:7	biased [1] 66:6
argument [1] 96:5	association [38] 14:12, 16; 16:14; 18:6, 8, 17; 19:1, 6; 27:21; 28:2; 39:6; 43:8, 10; 64:18; 65:5; 67:1, 18, 19; 68:1, 11; 69:8, 17; 70:7, 9, 20; 71:3, 10, 16; 72:7; 73:2; 74:9, 12; 76:7; 77:10, 11; 91:4; 92:16		benzene [229] 7:16, 18; 8:19, 20; 9:5; 10:5, 13, 20; 12:3; 13:6, 12, 17; 14:8; 16:14; 17:1, 7; 9, 12; 18:3, 4; 19:1; 20:11; 21:1, 13, 14; 17; 22:10; 24:19, 20; 25:10; 26:8, 15, 20; 27:5, 10, 16, 22; 28:3; 20; 29:2, 4, 7, 13, 17; 19; 30:1, 4, 5, 11, 14; 17, 21; 31:3, 6, 19; 32:21, 22; 35:19, 21; 36:1, 3, 4, 11, 16, 20; 37:10, 20; 38:3, 19; 20; 40:3, 6, 9, 19, 21; 22; 41:6, 9; 42:8; 44:6; 47:10; 48:1, 2, 10, 17, 22; 49:15; 50:22; 51:22; 53:15, 22; 54:12; 55:14, 21; 57:14; 61:22; 62:6, 9, 17; 64:1, 19; 67:1, 10, 15; 72:19; 73:2; 74:3, 16; 75:15; 76:9, 11; 77:3, 13; 78:14; 79:2; 80:4, 17; 83:7, 9, 14, 19; 84:15, 19; 87:8, 22; 88:11; 89:14, 20, 21; 90:5, 13; 91:5; 92:11, 22; 93:3, 7, 14; 96:6, 9; 97:18; 98:4, 5; 99:19, 22; 100:9, 17; 101:6, 17, 20; 102:1, 3, 13, 14, 17; 103:3, 7; 111:8; 113:5, 14, 22; 114:7, 11; 115:6, 7, 11, 12; 116:18; 117:18; 122:21; 123:7, 8, 13, 17, 21; 124:2, 10, 13, 20; 127:2, 5, 6, 17, 18; 128:2, 6, 15, 22; 129:2, 9, 13; 132:9; 133:4, 16; 135:7, 11; 136:3, 8, 9; 137:12; 138:7, 14; 139:8, 17; 140:1, 3, 8, 19; 141:17; 142:5; 143:16, 17, 21; 144:21; 147:20; 148:18, 21; 149:3, 15; 150:2, 21; 157:6; 159:18, 21, 22; 160:7	biller [1] 104:7
arose [1] 154:12	Association's [1] 81:2		binder [1] 152:9	binders [3] 20:1, 7; 165:7
article [28] 15:21; 19:13, 16; 20:6; 32:12; 33:1; 35:17; 55:7; 68:3; 82:4; 89:22; 90:3, 15; 91:8, 22; 92:4, 9; 94:2; 96:10, 12, 15; 97:4; 107:19; 111:19; 112:1, 6, 13; 159:17	associations [1] 70:4		biochemical [1] 25:4	biologic [3] 27:21; 102:4, 9
articles [21] 19:18; 37:4; 41:15; 42:11; 50:19; 51:2; 54:18; 56:19; 91:16, 18; 94:3; 102:15, 19; 103:20; 111:22; 125:7; 152:18; 153:15; 165:10, 12	Assume [1] 42:17	Based [4] 34:3, 4; 113:15; 114:14	biomarkers [1] 102:21	biological [6] 27:22; 101:17, 19; 102:12, 16; 103:7
asking [8] 21:4; 22:15; 80:18; 100:2; 131:11, 12; 137:22; 140:7	assume [21] 62:19; 63:1, 3; 66:10; 82:8; 124:4; 126:7; 132:2, 4, 13; 133:19; 136:16; 137:10, 12, 22; 138:2; 139:21, 22; 140:7; 149:2; 165:15	based [31] 38:8; 39:5, 9; 56:20; 62:5; 65:13, 16; 66:3; 67:11; 71:19, 22; 72:10, 20; 73:9; 74:1; 76:13; 80:9; 94:8; 102:2; 111:14; 112:21; 115:1; 119:1, 4, 8; 122:19; 145:18; 146:6; 148:6, 7; 150:12	body [6] 47:11, 18; 48:15; 86:16; 98:3, 5	Bond [14] 46:17; 58:14, 17; 59:13; 72:9, 22; 75:18; 76:5, 12; 97:15; 125:14, 16, 17, 19
aspect [1] 127:13	Assuming [1] 116:8	bases [3] 94:17; 158:15; 164:9	Bond's [2] 59:8, 11	bone [11] 24:19, 22; 25:8, 10; 41:2; 48:6; 49:10, 16; 50:5; 51:1; 54:5
assess [3] 8:20; 71:22; 76:19	assuming [2] 19:4; 143:17	basic [1] 128:20	bones [1] 92:15	book [2] 161:3, 7
assessing [2] 71:3, 10	assumption [3] 66:1; 132:15, 16	Basically [1] 39:5	books [1] 44:20	bounded [1] 88:7
assessment [4] 28:1; 34:4; 70:18; 136:16	assumptions [1] 138:3	basically [6] 29:21; 56:18; 122:1; 142:21; 154:9; 157:15	Bradley [8] 14:15; 28:17; 29:2, 3; 30:9, 16, 20; 31:2	brain [1] 122:13
assigned [1] 89:15	ASTDR [1] 81:9	basing [4] 45:2; 76:10; 94:1; 140:4	branch [1] 142:22	breadth [1] 30:6
assignment [11] 119:9	atmosphere [1] 29:21	basically [13] 9:4; 39:13; 54:9; 74:7; 75:15; 80:8; 100:20; 118:22; 121:20; 122:22; 124:3; 136:18; 163:19	break [6] 58:21; 60:20; 96:20; 97:1, 10; 107:6	brief [3] 115:10; 148:8, 15
assignments [3] 4:20; 7:11; 129:17	atomic [1] 155:20	bear [1] 15:6	Briefly [1] 27:4	Britain [2] 46:9, 10
assist [1] 32:8	Atrium [2] 5:5; 149:21	becomes [1] 87:3	broad [3] 62:11	
associate [1] 113:5	attach [4] 115:9, 15; 116:3; 120:6	beforehand [1] 134:12		
associated [29] 14:2, 8; 17:1, 6, 9; 18:4; 34:11; 36:17; 53:1, 6, 10; 64:10; 72:19; 74:16; 77:3; 83:16; 95:8; 96:8; 98:11; 100:9; 101:12; 110:22; 115:11;	attempt [2] 111:11; 143:16	behalf [1] 158:3		
	attempted [3] 59:20; 138:9; 150:20	beings [2] 54:2; 101:15		
	attempting [2] 43:7; 113:5	believe [42] 5:4, 16; 6:17; 10:1; 14:20; 19:11; 23:17; 29:11; 31:1, 5, 19; 36:13; 46:7; 47:12; 51:13; 58:18; 61:9; 64:16; 69:9; 72:15; 78:5; 80:21; 82:5, 10; 89:7; 90:18; 91:6;		
	attorneys [1] 158:3			
	August [1] 52:2			
	author [2] 98:20; 111:11			
	authored [1] 32:12			
	authors [1] 112:12			
	available [1] 161:13			
	average [7] 8:20; 9:5; 108:7; 111:7; 139:12; 140:1, 9			

16:20; 17:5; 97:13
building [3]
 5:21; 6:2; 150:1
buildings [1]
 143:1
built [1]
 163:11
burn [1]
 53:10

- C -

calculate [9]
 59:20; 83:18; 93:17;
 18; 138:9; 141:16;
 144:14; 150:20; 160:7
calculating [1]
 149:14
calculation [3]
 141:19; 20; 148:4
calculations [6]
 138:11; 145:11;
 146:5, 6, 10; 151:4
calculator [2]
 61:1, 2
call [2]
 35:12; 95:15
Calls [11]
 162:9
Canada [11]
 52:1
cancer [7]
 30:22; 32:13; 53:1, 7,
 10, 14; 154:17
cancers [6]
 130:9, 17; 131:9, IS,
 16
capable [1]
 149:14
Caravaggio [1]
 105:12
carcinogenic [1]
 132:1
carcinogenicity [2]
 28:20; 43:13
Carlos [5]
 6:16; 129:19; 130:1,
 9, 17
Case [14]
 56:5, 8; 60:9, 11;
 61:5, 13; 94:21;
 109:10, 13, 20; 110:2,
 9, 14, 15
case [86]
 4:12, 14; 7:8; 11:15;
 14:16; 23:8, 11, 12,
 13; 24:3; 29:2, 4, 7,
 14, 17, 19; 30:1, 4, 7,
 9, 16, 20; 31:2; 36:15;
 38:16; 44:18; 55:9,
 19; 56:3, 16, 17, 19,
 20; 57:10, 12; 58:3;
 60:2, 7, 16; 62:1;
 63:5, 19; 66:10, 11;
 73:21; 74:20; 75:2,
 21, 22; 84:9; 94:12,
 16, 20, 21; 95:1, 6;
 104:5, 9, 21; 106:1;
 107:3; 109:1, 5, 15,
 17; 120:13, 18; 121:2;
 123:9; 124:4; 126:21;

127:14; 128:13, 21,
 130:11; 137:9, 14:4;
 152:21; 155:6, 7
cases [37]
 23:1, 3, 5, 7, 15, 18,
 20; 24:1, 4, 7, 8, 10,
 13; 30:10, 13; 62:5, 7,
 14; 63:8, 15; 66:14,
 18; 67:5, 9; 72:8, 16;
 73:12, 18; 74:21;
 75:3, 4; 87:13, 16;
 88:9; 154:8; 157:16
categorized [1]
 19:7
category [1]
 18:18
caught [1]
 142:12
causal [16]
 18:22; 19:6; 27:15;
 28:2; 37:19; 43:7, 10;
 64:18; 66:13; 68:1, 4,
 10, 18; 69:8; 71:3, 10
causality [2]
 70:15; 72:5
causally [1]
 137:18
causation [5]
 63:18; 71:19, 22;
 78:18; 155:5
causative [11]
 68:4
cause-and-effect [1]
 37:13
caused [13]
 40:4, 6; 75:16; 76:9;
 90:19; 93:4; 154:17,
 20; 155:12, 16, 18;
 156:17; 157:17
cease [1]
 121:18
cell [6]
 25:22; 26:18; 49:14;
 51:1; 54:3; 101:4
cells [4]
 17:21; 18:14; 53:5;
 100:4
cellular [2]
 25:3; 54:4
certainty [2]
 65:5; 91:2
chance [10]
 67:10; 68:14; 70:19,
 21; 71:1, 14, 21; 72:4,
 7; 165:17
Change [1]
 84:1
change [7]
 32:18; 36:21; 65:5;
 115:13; 129:3; 138:1;
 147:11
changed [2]
 57:18; 128:20
changes [7]
 27:10; 37:5; 43:17,
 21; 50:21; 98:3, 4
chapter [11]
 161:3
characterization [3]
 58:2; 118:18; 137:7
chnracterize [1]

69:11
characterized [1]
 72:12
characterizes [1]
 69:9
characterizing [1]
 148:9
charcoal [5]
 10:8, 10, 11, 13, 20
check [1]
 131:5
chemical [15]
 31:14, 15; 33:10, IS,
 18; 34:1, 5, 15, 16;
 45:8; 62:9; 92:10;
 125:8; 162:8
chemicals [1]
 34:10
chemotherapy [1]
 155:12
choice [3]
 122:1, 2; 126:11
choose [3]
 63:15; 72:15; 74:21
chose [1]
 33:13
chosen [2]
 86:4, 10
chromosomal [2]
 37:5; 43:17
chromosome [18]
 27:5, 6, 10; 35:22;
 36:11, 15, 20; 37:10;
 38:12, 15, 18; 39:15,
 19; 42:8, 13; 43:18,
 21, 22
chromosomes [4]
 36:5; 38:6, 20; 40:13
chronology [1]
 151:8
cigarette [6]
 59:1; 91:5, 9; 92:22;
 93:3; 94:1
cigarettes [1]
 160:8
circumstances [8]
 19:2; 25:1; 26:20;
 49:19; 53:12; 54:5;
 129:17; 137:15
citation [7]
 15:16; 51:11, 16;
 90:13; 98:21; 99:4, 6
cite [5]
 50:19; 51:2, 19; 94:5;
 128:10
cited [2]
 51:14, 18
cites [1]
 152:20
claimed [1]
 70:13
cleaning [1]
 135:10
clear [11]
 49:21; 73:5; 80:7;
 123:3; 131:12;
 140:12; 141:1; 143:4;
 144:20; 158:9, 21
clinic [2]
 24:4, 15
clinical [2]

24:16, 18
closed [1]
 142:10
CMA [1]
 51:12
coauthored [1]
 31:13
cognitive [1]
 122:12
cohort [15]
 15:8; 16:1; 44:15;
 45:17; 46:22; 62:8,
 15; 63:22; 65:12;
 67:2; 77:6; 89:1; 95:3,
 7; 130:20
colleagues [1]
 72:22
collected [2]
 116:9, 11
column [1]
 149:3
combined [1]
 128:16
coming [2]
 124:2; 140:3
comment [1]
 132:11
Comments [1]
 51:21
comments [1]
 160:2
common [2]
 5:17; 122:15
commoniy [2]
 69:16; 156:14
communication [1]
 161:4
community [1]
 100:22
comparable [1]
 80:2
compare [2]
 33:9; 130:8
complaining [1]
 139:1
complaints [1]
 139:3
complete [2]
 103:20; 161:21
completed [1]
 130:7
completely [2]
 22:3; 148:19
compliance [1]
 148:10
compliment [1]
 42:5
compound [2]
 49:7; 83:8
computerized [2]
 117:15, 17
concentration [6]
 84:15, 19; 87:22;
 92:12; 143:22; 149:15
concentrations [2]
 87:8; 123:16
concept [1]
 68:13
concern [1]
 30:11
concerned [1]

98:7
concerning [9]
 30:13, 17, 21; 31:3;
 37:10; 50:5, 18;
 122:20; 162:6
conclude [6]
 62:14; 65:4; 70:22;
 73:8; 75:13; 77:11
concluded [1]
 166:3
conclusion [10]
 14:1; 39:11; 62:16;
 76:15, 16; 100:20;
 124:18; 136:11;
 162:10; 163:1
conclusions [6]
 33:20; 63:4; 65:13;
 109:3; 112:22; 113:8
conclusive [1]
 74:15
concomitantly [1]
 127:3
concurrent [2]
 127:5, 17
condition [1]
 129:16
conditional [1]
 40:4
conditions [3]
 55:12; 142:8; 148:16
conference [11]
 106:19
conferences [2]
 106:16, 18
confidence [2]
 94:9; 95:5
confident [3]
 71:13; 127:12; 145:3
confirming [1]
 60:21
confound [2]
 34:2, 7
confounded [1]
 33:18
confounder [1]
 34:14
confounders [1]
 34:17
conjunction [1]
 108:16
connection [1]
 158:4
consider [5]
 18:21; 71:2, 11;
 127:13; 129:19
considerably [1]
 65:14
consideration [1]
 27:22
considered [3]
 5:11; 66:22; 129:18
considering [1]
 72:20
consisted [1]
 8:1
consistent [3]
 19:11; 97:11; 133:10
consistently [1]
 91:3
construction [2]
 142:9; 143:18

contained [1] 165:6	credentials [2] 142:17; 151:5	104:11	77:22; 102:7	131:7; 132:6, 8;
container [1] 148:22	criteria [3] 38:3; 75:22; 76:17	dates [1] 117:22	depends [7] 10:17; 140:13, 14, 15;	143:11, 16; 145:7
contains [1] 112:21	criterion [1] 72:16	daughter [1] 55:3	142:9, 13, 14	determining [5] 55:20; 68:4; 72:18;
contaminants [1] 141:7	Critical [1] 92:7	DAVID [2] 4:3; 166:6	deposed [1] 119:6	107:19; 148:10
content [1] 139:7	critique [1] 111:16	David [1] 4:9	deposition [27] 4:20, 21; 6:14; 15:2;	develop [9] 26:17; 54:11; 55:6;
context [2] 69:18; 157:12	Crum's [1] 105:9	day [20] 12:9; 104:17; 106:7;	16:8, 13, 21; 20:6;	64:13; 74:3; 99:11;
continues [1] 43:18	Crump [2] 35:17; 159:16	119:9; 121:21, 22;	27:13; 28:7, 17; 36:8;	124:13, 19; 126:6
continuous [1] 123:8	Crump's [3] 15:7; 19:12; 96:10	124:5; 137:12;	37:17; 69:4; 99:17;	developed [7] 34:9; 55:3, 11; 61:13;
contract [10] 7:20; 8:7, 10; 77:17;	cubic [14] 83:20; 84:20; 85:3, 6,	139:12, 17; 140:1, 8,	105:9, 13; 127:16;	developing [1] 157:8
78:5, 10, 11, 18, 22;	8:87:10, 14, 17; 88:1,	147:8, 9	133:20; 134:11, 18;	development [10] 20:10, 16; 27:17;
80:15	2, 8; 109:7; 110:9	day-to-day [1] 121:20	136:19; 156:6; 165:2,	30:18; 37:21; 39:7;
contractors [1] 79:22	cumulative [15] 14:1; 48:21; 62:2;	days [2] 47:16; 48:11	8; 166:2	53:1, 6; 56:6; 90:19
contradicted [1] 137:8	64:19; 67:15; 73:10;	deal [1] 160:14	depositions [12] 6:11; 80:7; 105:4, 6;	Devine [3] 45:16; 83:3, 6
contribute [1] 72:4	75:7; 80:13; 108:8,	dealing [1] 7:20	118:6; 119:5, 16;	diagnostic [1] 97:14
contributed [1] 90:19	13; 109:11; 116:18;	Dean [2] 4:11; 6:7	129:15; 138:22;	diagram [1] 6:8
contributions [11] 155:4	132:21; 144:14; 145:7	death [5] 61:5; 126:8, 9, 12;	147:1; 161:9; 164:14	died [1] 61:6
control [6] 94:12, 16, 21; 95:7;	current [1] 41:16	131:3	depress [1] 25:12	difference [5] 78:15; 123:20; 124:1,
117:15	CV [4] 31:7; 32:4; 33:7, 9	deaths [6] 74:14; 130:8, 20;	depth [1] 28:6	6; 136:3
controlling [1] 56:3	cyanide [1] 53:9	131:2, 4, 8	derive [1] 18:13	differentiate [1] 136:13
copies [1] 153:13	cytogenetic [1] 98:3	decimal [1] 10:21	derived [5] 17:20; 33:7; 65:15;	difficult [3] 26:14; 71:13; 163:9
copy [19] 5:20; 33:7; 34:18;	cytotoxic [4] 53:13, 22; 54:1, 4	decimals [1] 10:20	67:13; 93:7	difficulty [2] 71:12; 135:15
35:4; 82:3; 117:6;	cytotoxically [1] 53:16	decrease [2] 100:6; 127:18	describe [2] 5:3; 142:19	direct [3] 12:13; 87:19; 130:1
120:5, 8; 151:11, 12,	cytotoxicity [2] 52:22; 53:5	Decreases [1] 127:19	described [3] 88:17; 112:10; 136:19	direction [2] 140:21; 151:3
14, 16; 152:11, 14,		deenergized [1] 156:8	description [2] 135:3; 143:3	disagree [4] 81:1; 134:3; 135:6;
17; 153:2; 159:9;		defend [1] 121:12	descriptions [2] 49:22; 147:1	138:19
160:5		definition [5] 22:12, 14; 61:11;	descriptive [1] 111:3	disagrees [1] 134:14
corporation [1] 160:21		85:16; 102:9	designation [1] 83:19	discard [2] 63:16; 66:18
correlate [1] 111:12		definitions [2] 22:16; 102:7	designed [1] 33:15	disclosed [1] 130:18
corresponding [1] 7:13		degree [1] 65:4	detail [8] 27:8; 46:14; 56:13;	discovered [1] 154:7
cost [1] 117:20		Delzel [1] 46:22	27:8; 46:14; 56:13;	discuss [5] 4:11; 37:4; 42:8, 12;
count [1] 25:20		demonstrate [5] 26:15, 18; 43:16;	97:14; 111:16;	132:15
counts [1] 51:1		64:7; 95:7	131:10; 134:11;	discussed [7] 6:13; 117:14; 145:9;
County [1] 137:11		demonstrated [2] 26:21; 92:14	162:14	146:7, 21; 157:19;
couple [3] 4:16; 153:20, 22		demonstrating [1] 40:18	detailed [1] 28:5	159:17
course [10] 6:12; 49:1; 56:21;		department [1] 118:14	details [2] 26:19; 141:4	discusses [4] 19:13; 39:15; 40:12;
65:17; 83:14; 105:19,		dependence [1] 10:15	detect [3] 10:13, 20; 11:7	115:17
22; 142:12; 156:15;		depending [2]	detectable [1] 113:21	discussing [8] 36:11; 40:13; 93:8;
163:17			detection [2] 10:2, 4	95:16; 96:15; 107:17;
court [1] 159:4			detector [1] 10:9	126:4; 145:14
covered [1] 143:7			determination [13] 5:11; 8:13; 43:13;	Discussion [1] 165:3
coworkers [1] 119:5			57:14, 21; 71:19;	discussion [3] 31:18; 81:14; 113:1
			72:5; 96:4; 126:9, 22;	discussions [1] 132:14
			129:8; 144:4, 11	disease [15]
			determine [22] 8:15, 18; 9:4; 33:17;	
			37:19; 43:7; 68:1, 18;	
			69:8; 75:6, 9; 76:8;	
			92:19; 102:12;	
			129:12; 130:14;	

- D -

11:10; 20:16, 18, 19; 63:12, 13; 71:9; 78:2; 98:2, 6; 126:6, 10; 154:14; 155:3	6:5, 68:10; 13:7 drawing [2] 6:6, 8 drawn [1] 10:16 drinking [1] 129:5 dropped [2] 109:16; 150:8 dropping [1] 150:7 drum [1] 148:21 due [5] 62:15; 65:5; 71:14; 130:20; 131:2 duly [1] 4:4 duration [3] 62:1; 145:18; 148:8 duty [1] 165:14 dysplasia [3] 89:17, 19; 90:6	54:5 elevated [4] 71:8, 18; 91:7; 95:8 elevation [1] 157:8 eliminate [2] 43:1, 5 eliminated [3] 43:11; 49:6; 157:4 else's [1] 162:19 EMF [1] 33:19 emphasized [1] 108:15 employees [9] 8:11; 77:18; 78:19; 79:1, 20; 118:7; 162:18, 19 employer [4] 161:21, 22; 162:2; 163:20 employer's [1] 163:14 employment [1] 138:14 enclosed [1] 140:13 end [10] 48:7; 70:11; 81:21; 82:2; 85:18; 143:2; 145:22; 146:2, 13, 15 endeavor [1] 165:13 endings [1] 122:7 energized [1] 156:9 engineer [1] 144:2 enhance [1] 127:18 enlightenment [1] 78:2 ensure [1] 163:10 entered [1] 90:10 entitled [1] 68:20 entity [1] 71:9 Environmental [8] 15:19; 35:18; 47:9; 52:1; 81:17; 107:18; 108:2; 112:2 environmental [1] 144:2 epidemiologic [11] 13:22; 21:20; 30:5; 38:9; 44:7; 50:10, 12; 75:9; 76:14; 77:1; 132:10 epidemiological [10] 38:11; 39:6; 44:19; 50:6; 77:15; 78:17; 94:14; 102:11; 129:19; 156:20 epidemiologists [1] 69:15 Epidemiology [1]	94:6 epidemiology [5] 71:22; 91:3; 94:19; 102:1; 132:5 EPRI [3] 33:4; 34:21; 35:9 equation [3] 27:14; 38:7; 42:14 equilibrium [1] 49:5 equipment [5] 78:13; 79:1, 16, 21; 80:12 Ernest [1] 105:10 erythroparesis [1] 25:12 escaping [1] 149:3 establish [6] 17:9; 18:6, 7, 22; 19:5; 125:5 established [1] 154:13 establishing [1] 70:15 estimate [18] 81:7, 9; 104:21; 106:10; 113:13; 145:1, 4; 146:1, 2, 13, 15; 147:10, 11; 148:5; 149:5; 150:12; 153:16; 160:18 estimated [3] 67:15; 111:7; 117:19 estimates [5] 14:4; 65:12, 15; 145:2; 148:13 estimation [1] 114:6 et [1] 16:9 ethers [1] 5:18 evaluate [1] 68:14 evaporate [2] 142:6, 15 evaporated [1] 140:18 Evelyn [2] 105:18, 21 events [11] 151:9 eventually [1] 127:11 evidence [29] 14:11; 17:8, 11; 18:6, 22; 38:15, 18; 43:10; 44:8; 63:12; 64:10; 69:17; 70:9; 73:5, 6; 74:16; 75:10; 77:2, 11; 93:6; 125:2; 127:4; 130:2; 133:8; 137:1, 9; 144:7; 154:12; 156:19 evolving [1] 100:17 exact [1] 68:21 exactly [2]	51:16; 69:1 EXAMINATION [1] 4:6 examination [1] 66:3 examine [4] 75:8; 76:6; 95:4; 101:12 examined [2] 4:5; 92:19 examines [1] 96:6 example [2] 86:19; 147:7 exceed [1] 19:2 Except [11] 96:10 except [3] 152:7; 153:17; 157:5 exception [1] 154:14 excess [2] 14:2; 74:2 exclude [9] 38:7; 39:14; 63:7; 70:18; 71:20; 72:3, 9, 17; 77:20 excluded [1] 63:20 excluding [1] 104:22 excreted [1] 47:13 excretion [2] 48:9; 49:7 excursion [1] 115:10 excursions [5] 80:3; 114:22; 145:17; 148:9, 14 Excuse [3] 31:20; 40:21; 148:20 excuse [1] 47:s exercise [2] 63:20; 72:18 exhale [1] 47:19 Exhibit [4] 119:20, 21; 120:10; 151:19 exhibit [3] 119:17; 120:6; 151:10 existed [1] 112:16 existing [1] 92:20 expect [3] 48:14; 61:21; 106:1 [4] 62:5, 7; 80:1; 128:5 expecting [1] 66:15 experience [2] 70:8; 81:10 expert [2] 23:6; 144:3 expertise [1] 37:3 explain [5]
- E -				
docket [1] 52:19 Doctor [31] 4:8; 7:9; 13:20; 15:4; 16:7; 26:22; 31:20; 34:18; 39:3; 41:21; 47:10; 52:22; 58:6; 64:8; 69:12; 81:22; 84:17; 85:16; 92:21; 97:17; 100:16; 104:3; 124:12; 137:3; 141:16; 151:21; 152:17; 153:8; 157:18; 164:16, 21 doctor [2] 38:17; 134:13 document [6] 52:3; 81:5; 117:12, 21, 22; 134:21 documents [8] 24:17; 104:11, 18; 119:2; 139:14, 16, 19; 153:11 doesn't [16] 18:2; 27:14; 37:3; 38:2; 43:20, 21; 62:16; 64:7; 66:20; 74:15; 97:10; 101:10; 109:3; 118:5; 131:3; 164:s dose [5] 22:9; 67:15; 132:21; 133:4, 10 dozen [2] 153:21; 154:2 dozens [2] 153:4, 5 Dr [22] 6:14; 52:7, 10; 65:14; 98:22; 103:6, 8; 105:6, 7, 8, 9; 120:18; 133:19; 134:18; 136:16; 137:13; 144:7, 9, 10; 160:14 draw [3]	Earley [3] 105:10, 11; 118:12 Earley's [1] 147:19 earliest [1] 104:11 easy [1] 134:20 ECD [17] 114:3, 13; 138:17; 139:1, 5, 7, 17, 22; 140:9, 17; 141:3, 5, 17; 142:8; 143:1; 150:1; 160:17 edit [1] 33:13 effect [8] 21:1; 86:15; 127:2; 128:2, 16; 129:8; 143:21 effects [2] 27:12; 101:11 efficient [1] 134:15 Eight [1] 106:8 eight [2] 46:8; 106:10 Electric [2] 31:17; 33:2 electric [1] 31:13 electrical [1] 31:15 electrician [1] 163:18 Electricians [1] 156:13 electricians [5] 7:20, 2i; 8:7, 8, 10 electromagnetic [6] 33:17; 34:9; 156:7, 10, 14, 17 elements [1]	54:5 elevated [4] 71:8, 18; 91:7; 95:8 elevation [1] 157:8 eliminate [2] 43:1, 5 eliminated [3] 43:11; 49:6; 157:4 else's [1] 162:19 EMF [1] 33:19 emphasized [1] 108:15 employees [9] 8:11; 77:18; 78:19; 79:1, 20; 118:7; 162:18, 19 employer [4] 161:21, 22; 162:2; 163:20 employer's [1] 163:14 employment [1] 138:14 enclosed [1] 140:13 end [10] 48:7; 70:11; 81:21; 82:2; 85:18; 143:2; 145:22; 146:2, 13, 15 endeavor [1] 165:13 endings [1] 122:7 energized [1] 156:9 engineer [1] 144:2 enhance [1] 127:18 enlightenment [1] 78:2 ensure [1] 163:10 entered [1] 90:10 entitled [1] 68:20 entity [1] 71:9 Environmental [8] 15:19; 35:18; 47:9; 52:1; 81:17; 107:18; 108:2; 112:2 environmental [1] 144:2 epidemiologic [11] 13:22; 21:20; 30:5; 38:9; 44:7; 50:10, 12; 75:9; 76:14; 77:1; 132:10 epidemiological [10] 38:11; 39:6; 44:19; 50:6; 77:15; 78:17; 94:14; 102:11; 129:19; 156:20 epidemiologists [1] 69:15 Epidemiology [1]	94:6 epidemiology [5] 71:22; 91:3; 94:19; 102:1; 132:5 EPRI [3] 33:4; 34:21; 35:9 equation [3] 27:14; 38:7; 42:14 equilibrium [1] 49:5 equipment [5] 78:13; 79:1, 16, 21; 80:12 Ernest [1] 105:10 erythroparesis [1] 25:12 escaping [1] 149:3 establish [6] 17:9; 18:6, 7, 22; 19:5; 125:5 established [1] 154:13 establishing [1] 70:15 estimate [18] 81:7, 9; 104:21; 106:10; 113:13; 145:1, 4; 146:1, 2, 13, 15; 147:10, 11; 148:5; 149:5; 150:12; 153:16; 160:18 estimated [3] 67:15; 111:7; 117:19 estimates [5] 14:4; 65:12, 15; 145:2; 148:13 estimation [1] 114:6 et [1] 16:9 ethers [1] 5:18 evaluate [1] 68:14 evaporate [2] 142:6, 15 evaporated [1] 140:18 Evelyn [2] 105:18, 21 events [11] 151:9 eventually [1] 127:11 evidence [29] 14:11; 17:8, 11; 18:6, 22; 38:15, 18; 43:10; 44:8; 63:12; 64:10; 69:17; 70:9; 73:5, 6; 74:16; 75:10; 77:2, 11; 93:6; 125:2; 127:4; 130:2; 133:8; 137:1, 9; 144:7; 154:12; 156:19 evolving [1] 100:17 exact [1] 68:21 exactly [2]	51:16; 69:1 EXAMINATION [1] 4:6 examination [1] 66:3 examine [4] 75:8; 76:6; 95:4; 101:12 examined [2] 4:5; 92:19 examines [1] 96:6 example [2] 86:19; 147:7 exceed [1] 19:2 Except [11] 96:10 except [3] 152:7; 153:17; 157:5 exception [1] 154:14 excess [2] 14:2; 74:2 exclude [9] 38:7; 39:14; 63:7; 70:18; 71:20; 72:3, 9, 17; 77:20 excluded [1] 63:20 excluding [1] 104:22 excreted [1] 47:13 excretion [2] 48:9; 49:7 excursion [1] 115:10 excursions [5] 80:3; 114:22; 145:17; 148:9, 14 Excuse [3] 31:20; 40:21; 148:20 excuse [1] 47:s exercise [2] 63:20; 72:18 exhale [1] 47:19 Exhibit [4] 119:20, 21; 120:10; 151:19 exhibit [3] 119:17; 120:6; 151:10 existed [1] 112:16 existing [1] 92:20 expect [3] 48:14; 61:21; 106:1 [4] 62:5, 7; 80:1; 128:5 expecting [1] 66:15 experience [2] 70:8; 81:10 expert [2] 23:6; 144:3 expertise [1] 37:3 explain [5]

120:1

- G -

GARABRANT [2]

4:3; 166:6

Garahrant [4]4:9; 119:21; 120:10;
151:19**Gasoline [2]**

45:18, 19

gasoline [4]29:20; 30:9; 45:21;
83:4**gave [3]**

105:5; 119:17; 130:10

gear [1]

80:6

Gene [2]

98:13; 160:9

generate [1]

151:15

generating [1]

58:11

genetic [2]

27:11; 28:19

genetically [1]

157:2

Geometric [1]

86:1

geometric [10]

81:2; 84:14, 19;

85-15, 17; 86:1, 13,

22; 87:11, 22

Give [1]

6:6

give [12]

7:10; 9:4; 15:15;

60:20; 68:7; 70:14;

78:2; 84:18; 97:2;

98:20; 145:12; 153:13

Given [1]

137:15

given [6]

79:1, 4; 122:21;

131:5; 149:16; 158:11

glycol [1]

5:18

goal [2]

76:7, 20

goes 131

95:6; 121:19; 128:3

Goldstein [1]

103:8

Goldstein's [1]

103:6

grab [1]

148:8

grate [1]

143:7

grates [1]

142:11

Great [2]

46:9, 10

great [2]

27:8; 115:10

greater [2]

124:19; 145:4

grossly [1]

86:5

ground [3]33:11; 61:16; 68:11,
16, 19**explains [1]**

22:1

explanation [3]

70:19; 71:1; 81:11

exploration [1]

40:3

explosion [11]

97:21

explosions [1]

97:22

exposed [23]

34:10; 36:21; 39:11;

40:7; 45:21; 48:1, 2,

18; 50:21; 62:21;

93:5; 114:7; 116:15;

128:22; 129:9;

133:16; 138:18;

147:2; 148:18;

155:20; 156:7, 10, 14

exposure [161]

8:13, 20; 9:5, 9, 11,

15; 12:1; 19:2; 25:1,

11; 27:16; 30:17, 21;

33:17; 34:13, IS;

37:11, 20; 39:7; 40:5,

9, 10, 12; 41:3, 9;

42:8, 12; 44:8; 49:5,

20; 50:1, 5; 54:6, 11,

13; 58:16; 59:17;

61:22; 62:2, 3, 6, IS,

18; 63:13; 64:1, 3, 8,

19; 65:12; 66:2; 67:1,

2, 3, 5, 8, 11, 15, 18;

70:5; 72:19; 73:3, 10;

74:10; 75:5, 7, 8, 16;

76:6, 11, 17, 20; 77:3,

13; 78:8, 16; 79:2, 3,

15, 22; 80:3, 13;

83:14, 19; 86:19;

87:16; 88:11; 93:9;

97:18; 98:4, 9; 99:10,

20; 100:1, 3, 10, 16;

101:12, 13; 102:3;

108:8, 9, 12, 13, 19,

20; 109:1, 2, 11;

111:2, 8; 112:7, 12,

16, 17, 22; 113:2, 14,

17, 20; 114:3; 115:13;

116:16, 17, 19;

120:13, IS; 123:19;

124:14, 20; 125:4;

126:3, 5, 7; 127:3, 5,

17; 128:6; 129:2, 12;

137:5, 7, 19; 138:7,

13; 140:12, 22;

143:17; 144:5, 15, 16;

145:2, 18; 146:11;

148:6, 14; 150:3;

156:16; 157:6; 160:8

exposures [36]

7:18; 14:1; 31:14;

33:10, 16, 18; 34:1, 5,

16; 40:17; 65:16;

70:6; 75:10, 14; 79:2,

8; 80:10; 83:16; 85:2;

110:21; 111:3, 12;

113:6, 22; 114:10, 15;

121:16; 128:17;

129:17; 133:2;

138:17; 144:11;

145:15; 146:7, '9

express [1]

36:15

extent [4]

10:17; 96:22; 97:10;

103:2

extra [1]

152:11

extreme [1]

86:17

extremely [3]

54:6, 11; 163:9

Exxon [1]

47:3

Eyler [1]

105:10

- F -

face [1]

153:2

facility [12]

5:5, 16; 8:16; 27:17;

77:2, 4, 16; 119:1;

143:16; 144:14;

149:21; 162:20

fact [22]

25:8; 33:14; 61:3;

62:1, 5, 20; 78:1;

87:21; 111:21;

114:17, 19; 115:1;

123:7, 18; 128:13;

130:14; 137:11, 13;

138:2; 147:12; 150:4;

161:11

factor [5]

38:4; 121:15; 129:7;

155:8, 9

factors [6]

68:4; 71:20; 78:7;

93:10; 143:20; 154:13

fail [1]

122:17

fair [4]

20:5; 72:12; 127:22;

134:12

fairly [5]

27:19; 28:5; 47:15;

122:9; 134:20

fall [1]

37:3

fallout [1]

155:20

falls [2]

37:2; 157:15

false [1]

62:18

familiar [11]

10:10, 11, 12; 51:7;

86:7; 98:12, 17, 19;

99:1; 103:6; 141:4

families [1]

17:19

family [3]

18:7, 8; 54:20

fashion [1]

72:21

faster [1]

153:6

fat [1]

49:14

father [4]

55:5, 12; 56:16

fatigue [1]

121:15

fatigued [2]

121:22; 122:8

fatigues [1]

121:18

fatty [1]

48:14

favor [1]

165:16

feasibility [1]

77:22

Federal [2]

52:17, 21

feel [2]

121:4; 156:19

feet [1]

150:10

fell [3]

88:7, 10, 12

field [2]

31:14; 33:17

fields [5]

34:10; 156:7, 10, IS,

17

figured [1]

5:6

find [25]

7:19; 8:6; 9:9; 11:8,

14, 21; 12:4; 42:13,

22; 58:19; 65:6; 66:6;

81:21; 82:8; 83:2;

90:15; 91:22; 95:4;

96:11; 112:5, 9;

115:4; 134:20; 159:4;

160:10

finding [5]

70:20; 71:2, 18;

116:4, 5

findings [5]

65:13; 71:13; 72:4;

78:9; 111:6

fme [6]

59:2; 88:20; 107:12;

153:3, 9; 166:1

finer [1]

97:1

finger [1]

157:10

fire [1]

142:11

First [5]

78:22; 108:17;

116:22; 126:5; 137:4

first [17]

4:4; 14:22; 24:20;

30:16, 20; 31:2;

57:19; 60:2; 75:6;

104:9, 17; 115:16;

125:3; 126:7, 10;

138:13; 149:4

fits 131

92:19; 143:9, 11

five [9]

61:18; 67:4, 9; 72:8;

73:9; 103:4; 121:11;

126:2, 3

five-minute [1]

58:21

floating [3]

100:21; 101:1, 2

flow [1]

143:19

flowed [1]

143:1

focus [1]

98:8

focused [1]

102:14

folder [1]

152:1

follow [1]

131:1

follow-up [2]

88:22; 89:1

following [1]

162:3

follows [1]

4:s

38:14; 134:10; 156:3 group [4] 51:21; 77:5; 78:3; 131:1 guess [6] 61:10; 102:3, 18; 109:17; 132:18, 19 Gulf [1] 52:1 guns [1] 135:10 guy's [2] 77:7, 8	161:10; 62:7, 9; 163:8, 15, 17 He's [2] 57:18; 158:14 he's [9] 6:7, 9; 52:13, 57, 8, 20; 99:8, 18; 164:6, 165:5 head [1] 60:19 heading [3] 16:20; 17:5; 19:7 Health [7] 15:19; 35:18; 52:2; 81:17; 107:18; 108:3; 112:2 health [2] 161:22; 162:3 heard [1] 164:18 held [1] 33:16 help [3] 37:19; 55:1; 149:13 hematologic [1] 50:21 hematopoietic [1] 130:9 hemopoietic [4] 30:22; 130:17; 131:8, 16 here's [1] 131:19 Herman [1] 4:21 high [20] 9:9; 13:11, 13; 25:1, 8, 11; 40:9, 12; 49:19; 54:6, 11; 64:8; 65:4; 80:4; 81:7; 82:10; 137:5; 146:15; 149:9; 150:3 higher [7] 20:19; 66:3; 79:3, 7; 82:13; 86:12, 16 highest [2] 9:11, 14 highlight [2] 134:18; 135:1 highlighted [2] 134:17; 135:2 Hiroshima [1] 155:21 history [5] 7:7; 63:1; 90:19; 93:13; 129:5 HNU [1] 10:9 HOLLINGSWORTH [44] 6:7, 17; 12:19; 13:1; 29:3, 8, 15; 30:2; 38:13, 21; 42:16, 19; 43:18; 44:1; 50:8; 56:15; 57:16; 58:1, 20; 76:2; 82:1; 101:5; 116:5; 118:17; 120:7; 134:9; 136:22; 137:20; 140:4; 147:15; 148:20; 151:16; 152:15;	53:4; 158:1; 159:9; 51:15; 162:9, 21; 54:4, 18; 65:4, 11, 19 Hollingsworth [4] 106:3; 119:3; 150:15; 153:1 honest [2] 106:21; 143:10 hour [6] 106:10, 18; 107:7, 12 hourly [3] 78:14; 79:3; 104:3 hours [12] 104:5, 20; 105:3, 15, 20; 106:8, 10, 11, 13; 138:15, 16; 145:20 human [13] 44:6; 50:13; 54:2, 3; 98:2, 3, 4, 6; 101:15; 140:12, 22; 155:3 humans [3] 28:4; 43:14; 93:7 hundred [1] 145:19 hundreds [2] 50:2; 144:21 hundredths [1] 114:12 husband [1] 55:4 hydroquinone [2] 127:11; 128:4 Hygiene [1] 81:2 hygiene [11] 6:10; 7:14, 15, 19, 22; 9:17; 79:8, 14; 113:15; 146:7; 147:2 hygienist [1] 144:2 hypothesis [5] 64:12, 14; 92:16, 19; 100:19 hypoxic [1] 53:5	18; 161:4; 164:14 idea [1] 104:21 identical [2] 80:1, 14 identification [1] 109:6 identified [4] 119:21; 120:10; 151:19; 155:7 identify [3] 123:4, 6; 157:16 ignore [1] 122:13 II [2] 5:3 immune [1] 122:2 implied [1] 29:9 imply [1] 48:12 implying [1] 29:6 important [9] 44:21; 47:21; 56:5; 58:11; 69:19; 72:6; 128:12; 155:5; 160:15 impression [11] 9:7; 41:7; 79:7; 114:9; 117:2; 118:3, 22; 119:11; 143:4, 6; 144:22 improper [2] 75:17, 21 in-house [1] 160:21 inadequately [2] 88:17; 112:10 incident [1] 13:8 include [18] 6:12; 16:17; 18:1; 45:7, 9, 15, 18, 21; 46:11, 17; 54:16; 77:4, 9, 17; 105:3; 117:22; 118:1; 126:20 included [14] 16:19; 20:5; 31:18; 63:19; 76:1; 77:20; 78:1, 6, 9; 83:12; 103:2; 106:11; 114:13; 118:3 includes [1] 96:1 inconclusive [1] 73:6 increase [2] 63:21; 131:7 increased [12] 74:5; 76:19; 77:12; 92:14; 95:13; 110:22; 125:3; 133:7, 8; 154:15, 16, 19 independent [2] 6:1; 66:3 indexed [1] 90:4 indicate [4] 51:8; 123:1; 124:8; 138:16	indicated [5] 12:1; 112:12; 118:7, 15; 161:20 indicates [3] 64:17; 65:1; 110:21 indicating [3] 13:4; 65:15; 119:7 indication [1] 97:2 individual [13] 18:18; 20:9, 12, 20; 21:6, 22; 22:4, 12, 16, 19; 48:17; 55:13; 74:20 individuals [3] 22:8; 34:8; 64:11 induced [1] 157:2 induction [1] 141:8 Industrial [2] 81:2; 84:10 industrial [11] 6:10; 7:13, 15, 19, 22; 9:16; 79:8, 14; 144:2; 147:1; 162:15 Industries [1] 16:9 industry [2] 49:20; 50:22 inexcusable [1] 77:6 inexplicably [1] 109:15 infamous [1] 6:15 Infante [3] 15:8; 45:13; 132:21 Infante's [1] 16:1 inference [1] 38:15 inform [2] 162:4, 17 information [9] 7:10; 56:6; 86:16; 112:7, 16, 22; 141:22; 142:1; 150:13 infrequent [1] 80:8 initiative [1] 158:19 inquiries [1] 57:11 inquiry [3] 58:12; 66:18; 76:5 inside [1] 150:1 Insofar [1] 79:20 instance [2] 17:4; 116:17 instances [4] 53:4; 116:17; 147:22; 157:13 Institute [2] 31:18; 33:2 instructed [1] 151:22 intact [1] 54:2
---	--	--	---	--

intend [3] 27:2; 46:18; 160:3	161:11, 162:2	149:12	97:15; 98:2; 107:13, 16; 119:19; 147:18; 149:2	150:20
intent [1] 96:2	jobs [1] 33:16	large [4] 43:5, 10; 76:13; 148:11	let's [3] 14:22; 137:10, 12	levels [31] 12:1; 20:17; 25:8, 11; 40:10, 12; 44:8; 50:1, 22; 54:11; 58:16; 79:22; 80:4; 93:9; 98:9; 113:2, 17, 20; 114:3, 16, 18, 21; 115:1, 19; 123:19; 129:2; 137:5, 8; 144:5; 150:9
intention [1] 160:10	JOEM [1] 47:8	largely [3] 41:7; 49:19; 56:9	letter [1] 104:10	Library [1] 90:10
intentionally [1] 153:12	John [4] 4:1A, 20; 105:13; 120:412	larger [2] 87:4	Leukemia [1] 108:6	life [1] 151:9
interested [4] 27:20; 159:17, 20, 21	Johnson [1] 105:11	Last [1] 16:4	leukemia [159] 13:18, 21; 14:3, 7, 9, 12, 18, 21; 16:15, 18, 20, 22; 17:6, 16, 19; 18:3; 19:2, 7, 8, 14; 20:10; 22:1, 9; 27:18, 22; 28:4; 30:6, 18; 33:19; 34:9, 11; 37:5, 21; 38:4; 39:8, 12; 40:4, 9, 18, 20, 21; 41:1, 2, 6, 9; 42:15; 44:6; 51:22; 54:20; 55:4, 6, 11, 21; 57:15; 59:18; 61:13, 21; 62:5, 11, 14, 16, 20; 63:2, 9, 22; 64:2, 4, 5, 6, 9, 13, 18; 67:1, 19; 70:5; 72:19; 73:4; 74:3, 11, 14, 16; 75:11, 14, 15, 16; 76:6, 9, 12; 77:3; 78:7; 83:17; 89:4, 14, 17, 20, 21; 90:5, 12, 20; 91:1, 4, 7, 9; 93:4, 7, 11, 22; 94:8; 95:4, 8, 17, 18, 19; 96:5, 7, 16, 18; 97:1, 11; 98:8, 11, 17; 99:11; 100:17; 102:2, 15, 17; 103:3; 108:10; 110:22; 111:6, 12; 124:13, 19; 125:3; 129:1; 130:2, 4; 133:5, 13, 15; 137:18; 138:8, 21; 154:4, 6, 12, 20, 21; 155:8, 10, 18; 156:17; 157:2, 8, 12, 16; 159:22	lifetime [3] 108:9, 13; 111:8
internal [1] 33:5	Johnston [1] 105:12	last [4] 16:3; 28:11, 13; 58:13	leukemias [4] 17:12, 20; 18:13; 66:11	light [3] 11:9; 61:16; 128:13
interpretation [1] 93:9	JOM [2] 47:6, 8	latency [9] 19:4; 41:3; 45:4; 61:18; 124:12, 18; 125:10; 126:4, 9	leukemogen [1] 92:13	liked [1] 87:10
interpreted [1] 119:7	Journal [4] 15:18; 35:18; 47:8; 94:6	Lavender [26] 4:12; 7:11; 38:2; 61:6; 74:20; 75:5, 7; 76:8, 19; 77:5, 12; 88:15; 93:13; 117:11; 119:12; 128:14; 136:18; 137:6, 11; 144:12; 145:6; 154:7; 157:14; 160:8; 161:10; 162:5	leukemogenic [2] 21:1; 128:2	likewise [1] 12:4
interrogatories [3] 130:10, 16; 131:22	journal [3] 88:20, 21; 111:20	Lavender's [29] 4:20; 11:9; 27:16; 36:17; 37:20; 39:7; 40:4; 42:15; 75:2, 16; 77:1; 90:18; 93:4; 105:13; 113:13; 116:14; 119:8; 128:21; 129:4; 137:17; 138:7; 145:1; 146:22; 151:9; 154:4; 155:7; 161:21, 22; 163:14	leukemogens [1] 92:10	limit [6] 10:1, 4; 66:2; 97:21; 113:19; 147:11
interrogatory [3] 105:5; 131:6, 14	judgment [1] 63:18		leukopenia [4] 25:16, 18; 35:20; 159:18	Limited [1] 52:1
interrupt [1] 68:15	July [1] 32:6		level [40] 13:11, 17, 20; 20:19; 42:8; 49:10, 13, 14, 15; 50:5; 64:3, 8; 79:15; 83:13, 16; 93:14; 97:18; 98:5; 99:10; 108:19, 20; 109:1, 2; 111:2, 12; 113:5; 114:19; 115:11; 116:10, 16, 17; 121:4; 122:12, 13; 123:14; 148:19;	limited [1] 37:15
interval [1] 95:6	juncture [1] 152:21			limits [2] 94:10; 98:1
intrigued [1] 26:6	jury [2] 137:10, 13			line [2] 15:19; 109:16
invalid [1] 66:12	justify [1] 76:17			linear [1] 133:3
investigate [1] 56:7				lines [3] 25:22; 26:18; 54:3
involved [3] 117:19; 119:14; 144:17				lipophilic [1] 48:13
iron [2] 7:5; 113:21				list [16] 4:15; 6:18; 19:18, 20; 35:14; 53:12; 93:21 = 103:14; 105:5; 118:10; 120:1; 153:17; 154:1; 159:10, 14; 165:13
Irons [1] 120:18				listed [4] 70:3; 119:17; 131:15; 152:8
Isocyanide [1] 24:7				listening [1] 37:14
isocyanide [2] 23:13; 24:8				listing [3] 103:19, 21; 117:17
isocyanides [1] 24:10				literature [84] 10:14; 13:22; 15:11; 16:3; 21:10, 11, 13, 15, 17, 21; 26:14; 27:8, 11; 28:9, 12, 14, 16, 19; 30:5; 34:20; 36:4; 37:2, 9; 38:6, 9, 11, 20; 39:10, 14, 20; 40:8, 11, 14, 15, 16; 41:8, 17; 44:7; 46:14; 48:8; 49:18; 50:4, 7, 13, 15, 17; 51:15, 20; 54:10, 14, 16; 55:15; 64:17, 22; 65:3, 18; 66:8; 67:7; 72:10; 75:9, 13; 76:14; 89:14; 96:3, 6; 97:6,
issue [20] 41:13; 44:5; 49:18; 75:5, 12; 83:15; 100:11, 13; 101:22; 102:17; 125:17; 126:16; 128:21; 134:15; 139:10; 145:3; 159:20; 160:11, 16; 161:19				
issues [8] 30:7; 46:14; 56:7; 68:2; 77:22; 88:16; 112:9; 129:11				
Italy [1] 49:21				
itemize [1] 44:18				

9: 99:7, 14, 15; 100:19; 102:11; 103:10, 11, 19; 105:4; 124:17; 125:2; 132:10; 156:20; 160:3, 14	Maltoni [2] 99:7; 160:14	meaningful [5] 63:20; 91:11, 14; 112:18; 149:7, 10	method [3] 10:6; 63:17; 66:7	121:19
litigation [6] 23:15; 24:1, 3, 6, 8, 12	man [4] 61:8; 64:4, 5, 6	means [10] 17:20; 18:12; 44:4; 70:8; 85:20; 86:2; 137:3; 161:18; 162:12	methods [2] 10:7; 132:13	mischaracterizes [1] 161:16
living [2] 53:19; 54:2	manage [1] 53:7	meant [2] 22:8; 88:13	Michael [2] 92:9; 94:17	misinterpret [1] 11:11
locations [2] 7:17; 80:1	management [2] 118:7; 119:6	measure [1] 86:10	Michigan [3] 120:21; 121:2	Missed [1] 16:6
log [1] 117:15	manager [1] 139:7	measured [1] 115:2	Middle-aged [1] 61:9	missed [1] 15:15
Looks [2] 81:20; 109:22	manner [4] 56:1; 86:5; 97:12; 155:1	measurement [2] 50:1; 148:1	middle-aged [1] 61:12	missing [11] 33:10
looks [2] 109:14; 111:1	map [7] 5:9, 19, 20, 22; 6:3, 22; 7:2	measurements [14] 85:18, 19, 21; 86:8, 9; 88:7, 12; 114:20; 115:8; 144:18, 21; 146:7; 148:14	mile [1] 142:7	Misstates [1] 137:1
Los [1] 156:4	March [1] 52:15	mechanism [1] 40:3	Miles [16] 11:9; 27:16; 120:2; 130:10; 141:3, 10; 143:16; 144:14, 22; 147:13; 158:3; 161:11, 20; 162:2; 163:12, 16	mistake [1] 153:14
lot [4] 52:13; 87:9; 88:16; 164:11	mark [5] 119:19; 151:10, 12, 17; 165:2	mechanisms [7] 25:4; 28:3, 20; 40:8, 18; 159:22	Miles's [1] 158:3	mistaking [1] 94:20
lots [3] 24:15; 91:12; 142:14	marked [2] 16:10; 69:3	mechanistic [2] 41:5, 9	milligram [1] 88:2	MNB [15] 8:20; 12:10; 13:5; 29:22; 113:17; 123:18, 20; 124:2; 136:3; 138:16; 141:18; 142:5; 147:13, 21; 149:22
low [20] 34:13; 40:12; 42:8; 44:8; 50:21; 62:2; 64:3; 70:20; 83:13; 16; 111:7, 12; 113:5; 114:18, 21; 115:2; 123:15, 19; 145:22; 146:13	marker [5] 101:17, 19; 102:4, 9, 13	medical [7] 98:6; 100:6; 129:14, 16; 137:16; 155:11; 162:16	milligrams [14] 83:20; 84:20; 85:3, 6, 8; 87:10, 14, 17; 88:1, 2, 8; 109:7; 110:9	Mobay [18] 5:5; 7:12; 8:3, 11; 10:7; 77:2, 16, 17; 78:14, 17; 79:9, 20; 116:19; 117:1, 4; 130:4; 138:7; 149:21
lower [3] 11:6; 20:17; 50:5	markers [3] 102:3, 16; 103:7	Medicine [3] 47:9; 90:10; 161:5	millimeter [1] 83:21	model [2] 26:17
lowest [5] 9:21, 22; 10:1, 12; 11:4	marrow [12] 24:19, 22; 25:8, 10; 41:2; 48:6, 22; 49:10, 16; 50:6; 51:1; 54:5	medicine [3] 161:1; 162:17; 163:4	million [1181] 10:3; 11:3; 12:3, 7; 13:6; 14:2, 5, 6; 19:3; 40:17; 42:13; 43:17; 45:3; 50:3; 59:21; 60:3, 5, 8, 10, 14, 17; 61:14, 17; 62:21, 22; 63:6; 64:4, 5, 6, 13, 20; 65:2, 9; 66:9; 67:16, 17; 72:11; 73:3, 4, 6, 13, 14, 15; 74:2, 6, 10, 13, 18; 75:2, 19, 20; 76:11; 79:9; 80:17, 21; 81:3; 82:6, 9; 84:1, 4, 16, 22; 85:5, 7, 12; 93:5, 10, 14; 98:10, 16; 99:11; 107:20; 108:7, 8, 12; 109:11, 18, 21; 110:15, 19; 113:20; 114:4, 5, 13, 22; 115:6, 18; 116:6; 121:8; 123:14; 133:7, 9; 136:17; 137:8, 14; 138:10, 18, 19; 145:8, 16, 17, 21; 146:1, 3, 4, 9, 10, 14; 147:4, 5, 8; 148:4; 149:5; 160:18	modest [7] 27:19; 55:20; 63:21 = 66:22; 67:18; 95:12, 15
lunch [1] 107:5	Marshall [1] 137:10	meet [4] 72:16; 75:22; 106:3, 6	millions [1] 10:3; 11:3; 12:3, 7; 13:6; 14:2, 5, 6; 19:3; 40:17; 42:13; 43:17; 45:3; 50:3; 59:21; 60:3, 5, 8, 10, 14, 17; 61:14, 17; 62:21, 22; 63:6; 64:4, 5, 6, 13, 20; 65:2, 9; 66:9; 67:16, 17; 72:11; 73:3, 4, 6, 13, 14, 15; 74:2, 6, 10, 13, 18; 75:2, 19, 20; 76:11; 79:9; 80:17, 21; 81:3; 82:6, 9; 84:1, 4, 16, 22; 85:5, 7, 12; 93:5, 10, 14; 98:10, 16; 99:11; 107:20; 108:7, 8, 12; 109:11, 18, 21; 110:15, 19; 113:20; 114:4, 5, 13, 22; 115:6, 18; 116:6; 121:8; 123:14; 133:7, 9; 136:17; 137:8, 14; 138:10, 18, 19; 145:8, 16, 17, 21; 146:1, 3, 4, 9, 10, 14; 147:4, 5, 8; 148:4; 149:5; 160:18	molecular [1] 27:11
lymphatic [3] 130:16; 131:8, 15	Mary [2] 16:8; 28:17	meetings [1] 162:7	Mehlmán [1] 105:7	molecule [5] 101:3, 5, 9, 11, 13
lympho [1] 130:8	Massachusetts [1] 32:15	Mehlman [1] 105:7	member [1] 18:8	moment [1] 152:13
lymphocytes [3] 99:19, 22; 100:7	material [4] 105:15, 20; 122:20; 123:15	member [1] 18:8	members [1] 77:5	monitoring [16] 6:12; 8:6, 19; 9:17; 11:9, 13; 12:15, 17; 13:5, 7, 9; 115:4; 140:5; 144:13; 147:2, 20
lymphocytic [1] 100:4	materialized [1] 126:10	member [1] 18:8	memo [4] 115:17, 20, 21; 116:1	monocytic [23] 14:7, 9, 18; 16:18, 22; 17:6, 21; 18:2, 3; 19:14; 27:18; 37:21; 39:8; 42:15; 57:15; 90:20; 95:17, 22; 96:1, 4, 7, 16; 137:18
Lymphocytopenia [1] 100:8	materials [5] 103:22; 128:11; 129:15; 143:5, 8	memory [1] 23:9	memory [1] 23:9	mononitrated [4] 115:7; 123:8; 135:7; 136:8
lymphocytopenia [2] 100:9; 159:19	matter [6] 36:6; 43:22; 47:16; 67:4; 140:20; 143:14	mental [3] 9:14; 138:11; 145:11	ment [2] 6:9, 10	mononitrobenzene [1] 123:12
lymphoma [1] 89:4	May [2] 16:21; 41:14	mention [2] 6:9, 10	mentioned [1] 54:19	Montgomery [2] 16:8; 28:17
- M -	McCunney [11] 161:6	metabolism [2] 127:6; 128:1	metabolites [2] 49:8; 128:3	months [10] 59:21; 60:3; 67:17; 73:3, 14; 74:2, 10, 13; 98:16; 104:19
magnetic [1] 31:14	McDonald [4] 98:12, 13, 22; 160:9	metabolized [1] 47:12	meter [12] 83:20; 84:21; 85:4, 6, 8; 87:10, 14, 17; 88:1, 8; 109:7; 110:10	mortality [5] 32:13, 14; 67:11, 14; 126:11
magnitude [11] 75:11	MDI [5] 23:11, 12, 20; 24:3	metabolic [2] 47:21; 127:6	metabolized [1] 47:12	mother [3] 55:2, 10; 56:16
main [1] 86:16	mean [42] 8:22; 9:11, 18; 11:12; 13:13; 18:2; 20:12, 14; 21:3; 22:7, 11; 23:5; 25:3; 43:5; 56:11; 57:12; 59:15; 69:21; 81:2; 84:14, 19; 85:15, 17; 86:1, 5, 6, 7, 11, 13, 18, 21, 22; 87:11, 12, 22; 88:9; 96:14; 102:4, 6; 108:14; 144:6	metabolized [1] 47:12	metabolized [1] 47:12	
major [2] 39:17; 46:12	meaning [2] 63:14; 101:10	metabolized [1] 47:12	metabolized [1] 47:12	
majority [4] 88:6; 154:5; 157:13, 15		metabolized [1] 47:12	metabolized [1] 47:12	
Maltoni [2] 99:10; 160:12		metabolized [1] 47:12	metabolized [1] 47:12	

motives [1] 82:14	nerve [1] 122:6	85:4 5; 90:11; 92:16; 93:12; 94:3; 99:18; 100:7; 109:10, 13, 20; 110:9, 14, 15; 121:13; 140:22; 145:12; 148:11; 155:5; 165:2, 5	offer [1] 164:9	outlying [2] 87:2, 3
move [2] 21:8; 82:17	NIEHS [2] 98:22; 160:9	numbers [9] 11:5; 32:18; 85:5; 88:10; 93:2; 109:6; 110:18; 112:17	offhand [8] 47:22; 50:19; SI 2, 10; 52:12, 13; 54:18, 22	Outside [1] 154:16
moved [2] 86:22; 145:15	nine [1] 94:9		office [1] 101:2	outside [1] 102:10
MS [1] 61:1	NIOSH [5] 51:21; 65:6, 7; 66:3; 160:2		Oh [2] 47:19; 82:19	overall [1] 56:2
Ms [1] 106:4	nitrate [2] 128:15		Okay [8] 6:6; 11:22; 41:21; 45:5; 70:16; 117:21; 165:19; 166:1	overbroad [1] 134:10
MSDS [1] 161:12	nitrobenzene [1] 7:2		okay [4] 7:6; 35:7; 44:2; 82:17	overnight [1] 122:10
MSDSs [3] 161:12, 21; 163:13	nitrosamines [1] 92:11		old [4] 41:21, 22; 61:6; 127:20	oxide [2] 7:5; 113:21
multiple [11] 155:4	noncausal [1] 71:4	Object [1] 44:1	ones [14] 45:1, 2, 14; 46:12; 63:15, 16; 66:18, 19; 69:17; 74:22; 125:5; 132:17; 153:17, 18	
multiplicative [2] 132:12, 14	nonetheless [11] 145:5	object [8] 38:13; 42:16; 50:8; 58:1; 118:17; 134:9; 162:21; 164:4	open [6] 34:20; 51:20; 140:14; 143:5; 150:9; 160:2	- P -
multiplicatively [1] 132:2	nonincluded [1] 78:3	Objection [8] 29:3; 30:2; 57:16; 76:2; 136:22; 147:15; 161:15; 162:9	operated [1] 143:15	P-a-c-h-t-e-r [1] 105:8
multiply [2] 85:21; 132:7	nonlinear [2] 133:3, 10	objection [3] 29:15; 137:20; 162:22	operates [1] 132:2	p-o-l-y-o-l [2] 5:10, 15
Mutagenic [1] 26:11	nonscientific [2] 56:9; 58:4	obligation [1] 162:4	operators [1] 139:1	P-r-z-y-h-y-z [1] 105:8
mutagenic [4] 26:8, 16; 35:21; 159:21	nonsignificant [2] 73:2; 74:9	observations [1] 133:10	opinion [42] 14:14; 17:10; 36:16; 39:5, 9; 43:12; 45:2; 48:16; 59:13; 62:10; 72:11; 75:19; 76:10; 13:80:16; 87:6; 89:9; 122:19; 124:13; 128:20; 132:3, 8; 135:13, 14; 137:16; 138:6, 12, 13; 144:3; 148:17; 154:3, 5; 156:16; 158:11, 13, 15; 159:4; 162:4; 163:19; 164:8, 16	p.m. [1] 166:2
mutagenicity [1] 26:20	nonstatistically [11] 71:17	observe [1] 101:11	operated [1] 143:15	Pachter [1] 105:8
mutation [2] 101:3, 9	nonsystematic [11] 36:8	observed [1] 70:19	opinion [42] 14:14; 17:10; 36:16; 39:5, 9; 43:12; 45:2; 48:16; 59:13; 62:10; 72:11; 75:19; 76:10; 13:80:16; 87:6; 89:9; 122:19; 124:13; 128:20; 132:3, 8; 135:13, 14; 137:16; 138:6, 12, 13; 144:3; 148:17; 154:3, 5; 156:16; 158:11, 13, 15; 159:4; 162:4; 163:19; 164:8, 16	pack-year [1] 93:13
myelocytic [11] 18:13	noon [1] 107:11	obtained [11] 112:16	occasional [2] 12:6; 114:21	Page [2] 69:3; 111:6
myelogenous [8] 16:14, 18, 20; 17:17; 19:1, 8; 59:18; 64:18	normal [1] 142:8	occasion [1] 52:9	occasions [1] 28:15	page [10] 15:19; 32:1, 2; 82:11 84:11; 88:3; 94:7; 108:3; 135:2; 152:8
myeloid [7] 94:8; 95:4, 18, 19; 96:18; 97:1, 11	North [1] 7:4	occasional [2] 12:6; 114:21	Occupational [2] 47:9; 161:5	pages [7] 16:7, 10; 82:5; 94:7; 134:17, 18; 153:2
myelotoxic [3] 24:21; 55:14; 129:8	north [2] 7:1, 5	occasions [1] 28:15	occasional [2] 12:6; 114:21	Palo [1] 35:10
Myers [2] 105:13; 139:6	nose [3] 122:7, 16; 136:9	Occupational [2] 47:9; 161:5	occasions [1] 28:15	pancytopenia [8] 25:12, 17, 19; 35:20; 54:12; 100:3, 10; 159:19
myriad [11] 22:16	note [2] 9:14; 26:22	occasional [5] 160:20; 161:1; 162:16, 17; 163:4	occasions [1] 28:15	paper [20] 33:6; 63:21; 66:21; 74:4, 14; 81:19; 88:17, 19; 107:4; 109:4; 110:21; 111:1, 16; 112:15, 20, 21; 113:3, 4; 151:6; 159:15
myself [1] 144:1	notebooks [1] 165:21	occupations [2] 31:15; 34:5	occasions [1] 28:15	papers [2] 153:5; 165:5
	notes [8] 9:16, 18, 20; 134:7; 151:8; 152:2, 6; 157:21	occur [5] 25:2; 62:17; 121:20; 123:2; 130:21	occurrences [2] 80:11; 147:3	parent [1] 49:7
- N -	notice [5] 12:8; 51:8; 84:14; 108:15; 139:6	occurred [11] 12:10; 13:4; 62:1, 8; 67:10; 70:21; 80:8; 108:6, 10; 118:15; 136:18	occurs [1] 128:2	parents [1] 157:1
Nace [3] 15:1; 19:11; 28:18	noticed [1] 123:19	October [1] 104:12	odds [1] 94:7	Parmer [1] 105:7
Nagasaki [1] 156:1	notion [1] 21:22	odds [1] 94:7	odor [11] 80:18; 81:4, 12; 82:5, 12; 121:6, 13; 123:8, 13; 124:10; 134:4	Part [1] 56:2
Name [1] 17:22	November [1] 32:5	odors [2] 135:3, 5	Od [13] 45:8; 44:18; 58:14, 17; 59:5, 13; 72:9; 75:18; 76:4, 12; 97:15; 125:13, 14	part [12] 12:3; 20:10; 25:17; 29:20; 37:2; 40:17; 65:9; 79:9; 114:5; 146:9; 163:13
name [4] 4:8, 10; 18:1; 160:12	nth [2] 85:19, 22		ought [1] 141:14	partial [1] 103:19
names [2] 54:18; 165:12	Number [2] 31:17; 44:15		outcome [2] 97:19, 20	parts [112] 10:3; 11:3; 12:7;
naming [1] 135:15	number [50] 7:16; 8:2, 12; 10:17; 28:15; 32:16; 33:7; 47:15; 49:17; 50:20; 60:2, 7, 9, 11, 16; 61:5, 13; 63:5; 66:10, 11; 70:2; 75:21, 22; 84:18; 85:14, 15;			
National [1] 90:10				
nature [1] 55:14				
needs [13] 4:16, 19; 45:3				
neglected [1] 6:9				
negligible [11]				

13:6; 14:2, 4, 6; 19:3; 42:12; 43:17; 45:3; 50:2; 59:21; 60:3, 5, 8, 10, 14, 17; 61:14, 17; 62:21, 22; 63:6; 64:3, 4, 6, 13, 20; 65:1, 8; 66:9; 67:16, 17; 72:11; 73:3, 4, 6, 12, 14, 15; 74:2, 6, 10, 12, 18; 75:1, 19, 20; 76:11; 80:16, 21; 81:3; 82:6, 9; 84:1, 4, 16, 22; 85:4, 7, 12; 93:5, 10, 14; 98:9, 16; 99:11; 107:20; 108:7, 8, 11, 12; 109:11, 18, 21; 110:15, 18; 113:19; 114:4, 12, 22; 115:6, 17; 116:6; 121:7; 123:14; 133:7, 9; 136:17; 137:8, 14; 138:9, 18, 19; 145:7, 16, 17, 21; 146:1, 2, 4, 10, 14; 147:4, 5, 8; 148:4; 149:5; 160:18	period [11] 41:4; 45:4; 61:18; 93:15; 117:8, 10, 124:12; 125:10; 131:2; 138:14, 15 peripheral [1] 44:5 permits [1] 39:14 person [4] 20:18; 124:5, 9; 160:10 personal [5] 8:1, 9, 11; 81:10; 129:16 personally [1] 120:16 Perspectives [4] 81:18; 107:18; 108:3; 112:2 pertinent [1] 45:1 petrochemical [1] 83:10 petroleum [3] 45:22; 83:13, 15 phenol [3] 47:12, 22; 127:11 phone [1] 35:9 phosgene [1] 161:12 physical [1] 143:18 physically [1] 53:10 physician [3] 160:21; 162:16 physicians [2] 120:20; 121:1 pick [4] 63:15; 72:15; 74:21; 77:7 picked [1] 89:22 picture [2] 56:2; 108:22 piece [2] 56:4; 159:15 pieces [1] 107:4 pipe [2] 142:10; 143:8 pipeline [1] 83:11 place [4] 39:12; 57:13, 21; 122:15 places [1] 10:21 planet [1] 38:22 plant [23] 5:7, 9, 15, 19; 6:8; 7:17; 8:4, 12; 10:7; 77:13; 79:9; 113:16; 114:10; 116:20; 117:1, 19; 130:3, 5; 138:7; 141:10; 142:21; 143:2; 149:22 plants [3]	4:4; 8:2 plausibility [2] 27:21; 28:1 plausible [2] 92:16, 17 play [3] 28:1; 56:5; 58:10 128:22 plays [1] 27:19 Plioform [1] 45:12 plioform [2] 46:12; 81:13 plus [2] 153:19; 154:1 PMR [2] 57:5; 126:22 point [11] 5:8, 22; 48:9; 50:18; 126:14, 15, 17, 22; 133:6, 18; 157:7 points [1] 91:3 polyethers [1] 5:18 polymers [1] 5:17 polyol [2] 5-10, 14 polyols [4] 5:17, 21; 7:3, 4 pool [1] 141:8 pooled [2] 94:7; 95:4 poor [2] 122:1, 2 population [5] 61:22; 62:6, 17; 67:11; 130:22 portion [3] 59:5; 125:19; 142:6 positive [6] 69:16, 20, 22; 70:4, 7, 8 positively [1] 68:10 postulated [1] 150:13 postulates [1] 92:10 potential [1] 79:3 pounds [16] 139:12, 17; 140:1, 8, 17, 18, 19; 141:17; 148:17, 20; 149:2, 4, 6; 150:2, 8 Power [2] 31:17; 33:2 power [2] 44:10; 85:19 PPM [2] 11:5; 146:14 practice [3] 56:21; 101:9; 162:17 precise [1] 97:14 precision [1]	145:4 predict [1] 142:16 predictable [1] 130:22 preparation [5] 27:12; 28:6; 37:16; 99:17; 127:15 prepare [2] 24:13; 51:6 prepared [4] 4:15; 24:16, 17; 117:4 preparing [1] 158:4 presence [1] 129:1 present [8] 72:22; 91:5; 92:11; 119:12, 14; 163:16, 17; 165:7 presented [2] 86:12; 108:19 presupposes [1] 155:1 pretty [1] 149:9 prevalence [1] 34:12 primarity [1] 115:1 principal [1] 46:15 principle [1] 121:17 printout [2] 117:15, 17 prior [4] 29:2; 93:19; 106:14; 141:7 probability [3] 70:21; 87:7; 137:17 problems [2] 25:9; 112:11 proceed [1] 134:15 proceeds [1] 38:14 process [8] 11:10; 20:16, 18, 19; 29:22; 141:3, 5; 160:17 processes [3] 78:3; 98:6; 162:8 producing [1] 83:11 product [5] 85:18; 86:1; 145:21; 146:12, 13 products [1] 83:15 progress [1] 41:12 prolonged [1] 40:10 pronounce [1] 26:11 properly [1] 116:10 proportion [4] 47:20; 48:5, 10, 12 proposition [1]	100:21 protect [1] 79:1 protection [1] 79:4 protective [4] 79:16, 21; 80:6, 12 provide [2] 43:9; 74:15 provided [6] 32:7; 78:12; 129:15; 149:15; 158:14 providing [1] 163:13 Przybysz [1] 105:7 publications [1] 35:11 publish [1] 33:5 published [19] 15:8; 31:21, 22; 33:4; 34:19; 36:12; 45:9, 16; 47:5; 51:20; 52:17; 57:7, 9; 81:5; 91:8; 93:1; 99:2; 112:1; 160:11 pull [2] 51:3; 59:5 pulled [1] 59:8 purchased [1] 35:8 pure [10] 29:1, 4, 7, 13, 17, 19; 30:1, 4; 83:7, 8 purely [1] 162:15 purposes [8] 23:16; 24:1, 3, 8, 12; 42:17; 148:7, 9 Putting [1] 119:10 puzzle [1] 56:4
- Q -				
				quality [1] 9:3 Quantity [1] 9:3 Quantitywbe [1] 9:1 quarrels [1] 134:2 Question [3] 17:16, 18, 22 question [41] 8:5; 11:12; 17:10; 18:11; 22:7; 26:10; 29:9; 38:14; 39:4; 44:14, 16; 48:19; 50:9; 55:16; 57:7; 66:17; 69:5, 21; 78:4, 22; 83:8; 87:15; 96:2; 102:5; 110:17; 118:21; 123:22; 134:10, 12; 140:5, 11; 149:8, 11, 13; 150:5; 154:22; 155:1; 163:3,

6; 164:5, 13
questionable [5]
 56:9, 14; 57:19; 58:4;
 89:10
questions [5]
 55:22; 63:18; 68:6;
 78:21
quicker [2]
 55:11; 153:8
quickly [1]
 55:4

- R -

radiation [2]
 155:15, 17
radioactive [1]
 92:13
ran [1]
 142:21
range [21]
 10:3; 12:2, 7; 13:8,
 10; 14:3, 6; 40:18;
 64:19; 80:22; 88:1, 7,
 12; 121:7, 14; 145:2,
 15, 17, 19; 146:8;
 147:4
ranging [1]
 101:15
rapidly [2]
 121:18; 122:9
rare [1]
 145:16
Rarefy [1]
 71:21
rarely [3]
 80:13; 116:15; 147:2
rate [3]
 79:2; 104:3; 143:19
rates [3]
 62:6; 67:12; 131:5
ratio [1]
 94:8
Ray [1]
 118:11
Raymond [1]
 105:11
reach [2]
 49:5; 63:4
read [27]
 15:22; 20:6; 28:9, 12,
 13; 41:11; 52:12, 14,
 20; 69:13; 99:14, 15,
 16, 17; 103:10;
 105:19, 20; 128:10;
 133:19; 137:9; 143:5;
 144:9, 10; 157:21;
 159:11; 161:9; 165:1
reading [6]
 80:6; 105:19, 22;
 108:17; 123:3; 143:7
real [4]
 72:7; 144:20; 148:13;
 163:9
realize [1]
 165:14
reanalysis [1]
 46:11
reason [7]
 39:17; 40:8; 62:12,
 13; 77:19; 83:12; 91:6

reasonable [3]
 65:4; 112:16; 137:16
reasons [5]
 39:16, 18, 22; 132:5;
 137:4; 154:6
recall [43]
 8:9; 9:21, 22, 11:5;
 12:12; 13:7, 8, 9;
 15:10, 21; 19:17;
 22:21; 23:8, 12; 24:2,
 14; 28:13; 32:21;
 43:11; 47:22; 52:12,
 13; 54:17, 20; 55:7;
 68:21; 81:14; 93:2;
 97:5; 103:2, 3;
 106:20; 115:8;
 117:13; 118:9;
 119:13; 125:16;
 128:9; 134:19;
 139:10, 13, 19; 143:7
received [3]
 104:15; 139:12, 22
recent [1]
 111:9
recently [2]
 6:13; 152:7
Recess [2]
 59:3; 107:14
recognize [3]
 122:3, 17; 135:20
recollect [1]
 55:1
recollection [19]
 6:2, 4; 10:19; 13:3;
 14:20; 19:15; 23:14,
 19; 27:9; 30:12, 15;
 34:12; 96:22; 104:12;
 121:6; 133:17;
 142:20; 145:14;
 161:19
recollections [1]
 163:21
Record [1]
 117:4
record [11]
 69:13; 107:18; 117:9;
 119:4; 147:13, 16, 20;
 152:19; 159:12;
 161:16; 165:3
recorded [2]
 118:8, 16
records [10]
 24:5, 11; 116:14;
 119:8; 123:1, 3;
 129:14; 138:16;
 145:18; 146:22
reduce [2]
 25:19; 128:5
reductions [1]
 25:21
reestablish [1]
 122:11
reevaluated [2]
 45:13; 65:17
reevaluation [1]
 15:7
reexamined [2]
 65:11; 125:16
refer [2]
 69:16; 107:19
reference [2]

95:16; 125:6
references [2]
 70:3; 56:18
referencing [2]
 103:17; 125:20
referred [1]
 6:11
referring [17]
 11:2; 49:12; 50:9, 11;
 65:7; 73:11; 81:4;
 89:2; 92:5; 98:20;
 115:9, 21; 116:2;
 140:6; 148:2; 151:7;
 153:5
refineries [1]
 46:8
refinery [4]
 45:20, 22; 83:4, 9
refresh [3]
 6:3; 23:9; 121:5
refute [4]
 136:1, 4, 12, 15
regard [6]
 36:4; 55:19; 56:9;
 113:7; 122:14; 148:13
regarding [2]
 7:11; 130:2
regardless [1]
 20:17
Register [2]
 52:17, 21
regular [1]
 80:8
regulations [3]
 162:14; 163:10, 21
relate [2]
 7:17; 40:16
related [7]
 14:15; 16:22; 18:3;
 27:10; 38:19; 55:10;
 102:17
relates [3]
 20:10; 37:10; 70:18
relating [1]
 66:12
relationship [14]
 27:15; 33:18; 34:2, 7;
 37:13, 20; 44:13;
 64:7; 66:13; 68:5, 18;
 133:4, 11; 156:21
relative [4]
 61:10; 93:22; 95:5, 12
relatively [4]
 14:13; 55:20; 62:2;
 111:7
release [1]
 150:2
released [2]
 29:21; 150:21
releases [2]
 150:14, 15
relevance [10]
 37:9; 43:16; 57:20;
 88:4; 94:18; 115:3;
 124:3, 6; 140:12, 22
relevant [16]
 8:3, 13; 11:16, 21;
 32:20; 36:22; 42:14;
 43:13, 14; 83:15;
 94:14; 96:3; 126:16;
 144:12; 145:6; 160:11

reliability [4]
 56:10, 14; 57:7; 58:4
reliable [10]
 56:1; 59:14, 15;
 66:20; 76:18; 88:18;
 111:18; 113:9;
 126:21; 148:13
reliably [5]
 123:4, 6, 20; 135:20;
 151:2
relied [2]
 7:10; 165:6
reluctant [1]
 57:10
rely [10]
 7:7; 12:17; 36:14;
 50:17; 71:9; 79:11,
 13; 132:20; 144:3, 6
relying [1]
 119:16
remain [1]
 48:6
remained [1]
 88:16
remember [12]
 15:1, 5, 6; 24:5, 16;
 26:16; 90:12, 13;
 104:16; 139:3, 9, 11
removed [5]
 47:10, 17; 48:17;
 141:7; 142:12
repeat [1]
 39:4
rephrase [4]
 8:5; 20:21; 112:20;
 131:11
report [5]
 31:13; 33:8; 51:22;
 56:16; 111:9
reported [2]
 58:17; 64:3
reports [17]
 24:13; 27:9; 49:21,
 22; 55:19; 56:3, 5, 8,
 17, 19, 20; 57:10;
 58:3; 94:21; 95:1, 16;
 126:21
represent [8]
 86:5; 116:10; 118:5,
 11, 21; 144:1; 148:12,
 16
representation [1]
 66:6
representing [1]
 21:18
requirement [3]
 67:22; 69:7; 162:22
requirements [1]
 132:22
requires [1]
 72:11
rereview [2]
 81:13; 144:10
Research [2]
 31:17; 33:2
research [3]
 83:10; 101:21; 105:4
respect [1]
 97:19
respirators [3]
 78:12; 82:17; 162:6

respiratory [4]
 79:16, 21; 80:5, 12
response [3]
 54:12; 133:4, 11
responsibility [1]
 163:14
responsible [2]
 161:22; 163:20
responsive [1]
 81:15
rests [1]
 46:16
result [3]
 88:18; 98:4; 154:13
Resulting [11]
 127:7
results [5]
 9:17; 88:18; 108:20;
 112:21; 148:1
retained [2]
 48:13, 14
return [1]
 165:16
review [34]
 6:15; 9:10; 27:2, 11;
 28:5, 6, 16; 30:5;
 33:5, 11; 35:21; 36:7;
 37:16; 39:14; 40:8;
 41:13, 15; 51:4, 9;
 63:10; 72:10; 88:21;
 96:12; 99:5; 103:11;
 105:4; 111:21, 22;
 122:20; 125:14;
 127:15; 157:18;
 160:17; 161:13
reviewed [43]
 4:13, 16; 7:15, 22;
 11:13; 19:12, 19;
 23:15, 22; 24:2, 4, 7,
 9, 11; 28:8; 33:3, 6;
 40:14; 41:8, 10; 43:2,
 8; 44:17; 50:4; 51:5;
 52:7, 10; 70:3; 88:19;
 103:14, 22; 105:15;
 111:19; 112:3; 118:6,
 10; 120:4; 127:9;
 129:14; 134:21;
 152:4, 7; 161:14
reviewing [5]
 114:9; 134:11;
 159:18, 20, 21
Reviews [1]
 92:7
Right [1]
 73:15
right [15]
 53:21; 61:7; 82:16,
 18; 83:1; 86:3; 96:11;
 122:8; 126:6; 139:22;
 153:21; 157:11;
 161:2, 6; 164:20
Ring [1]
 4:21
Rinsky [8]
 15:8; 16:1; 45:13;
 65:8, 10; 111:9;
 132:18, 20
Rinsky's [2]
 65:13, 14
Risk [1]
 15:17

risk [50] 14:4; 22:1; 38:4; 39:12; 44:8; 63:22; 64:2, 10; 65:15; 71:8; 73:5; 74:5; 76:19; 77:3, 12; 78:7; 91:7; 93:10, 22; 95:4, 5, 8, 13, 15; 96:4; 98:8, 10; 101:12; 109:2; 110:22; 111:1, 6, 13; 113:1; 125:3; 128:5; 130:2, 3; 132:1, 6, 7, 12, 14; 133:7, 8; 154:13, 15, 16, 19	50:11; 67:3; 68:3; 75:5; 91:8; 154:9, 11 science [3] 76:21; 77:8; 155:11 scientific [27] 18:5, 22; 19:5; 26:14; 39:10; 50:7; 55:22; 56:6, 8; 57:9, 11, 13; 58:11; 63:11, 17; 64:17; 66:17; 72:17; 18; 76:5; 94:22; 100:19, 22; 124:17; 125:2; 130:3 scientists [1] 26:15 search [11] 16:3; 89:8, 10, 13; 90:16; 92:4; 102:12, 14, 19; 103:1; 105:16 searched [1] 15:11 searching [1] 89:18 second [7] 15:6; 31:20; 67:13; 75:8, 12; 97:16; 107:16 section [1] 82:16 Segal [8] 92:10; 94:1, 4, 5, 8, 17; 96:15; 157:9 sense [4] 12:22; 49:9; 112:10; 149:17 sensitivity [1] 122:11 sensory [1] 122:6 sentence [1] 112:20 separate [1] 83:10 separately [1] 78:21 series [3] 17:21; 18:13; 100:4 setting [5] 22:15; 55:5; 76:17; 101:14; 162:15 Shaddock [1] 105:6 Shell [2] 46:22; 47:3 shift [2] 148:6, 12 shifting [1] 128:4 shifts [2] 127:10, 22 shoe [3] 32:13, 14; 49:20 shoemakers [1] 54:21 show [13] 14:4; 16:7; 17:11; 44:7, 13; 50:22; 69:1, 17; 70:4; 71:8; 72:2; 73:1; 125:2 showing [4] 14:11; 66:13; 96:8;	15:8 shows [8] 14:10; 63:21; 64:2, 10; 66:22; 67:16; 85:3, 4 sign [1] 165:1 significance [16] 36:20; 37:9, 12; 67:22; 68:9, 13, 17; 69:7; 70:15, 17; 87:21; 115:3, 10, 15; 116:3; 129:20 Significant [1] 95:10 significant [19] 11:8, 12, 14, 19; 36:21; 67:20; 69:18; 70:1, 7, 10, 22; 71:8, 18; 72:1; 95:11; 123:9, 11; 140:2, 11 significantly [2] 92:14; 95:8 signs [1] 162:5 single [5] 10:17; 71:22; 102:9; 121:13; 155:2 sir [1] 9:19 site [6] 7:12; 8:3; 117:11; 119:9; 161:11; 162:19 sites [1] 7:14 sitting [1] 6:1 situation [1] 83:7 situations [2] 43:3, 6 six [1] 95:3 size [1] 130:21 skewed [1] 87:1 smaller [1] 44:12 smell [20] 80:17; 121:18, 19; 122:9, 11, 15, 21; 123:5, 6, 12, 13, 15, 16; 124:1; 135:9, 10, 13, 14; 136:17; 137:14 smelled [4] 123:4; 135:7, 11; 137:11 smelling [4] 121:21; 124:9; 135:16; 137:7 smells [5] 123:1; 124:5; 135:20; 139:2, 4 smoke [8] 59:1; 90:22; 91:5, 9; 92:12, 22; 93:3; 94:1 smokers [3] 91:6; 92:15; 157:8 smoking [11] 90:18; 91:4, 15; 93:15; 95:9; 96:4; 154:14, 20; 155:3; 157:5, 14 Snyder [1] 92:3 soft [1] 92:15 solely [2] 75:5; 163:20 solvent [4] 29:20; 48:13; 139:1, 4 solvents [1] 30:14 someone [9] 82:12; 123:22; 133:12; 135:17, 19, 21; 142:16; 151:4; 162:18 someplace [1] 5:19 somewhat [2] 10:11; 89:10 Somewhere [1] 110:8 somewhere [3] 5:2; 14:5; 113:19 son [3] 55:2, 3, 10 sorry [11] 9:2; 11:1; 15:18; 26:9; 47:2; 79:12; 83:5; 87:15; 99:21; 109:14; 110:1 sort [1] 108:22 sorts [1] 97:22 south [3] 7:2; 143:1, 2 sparse [1] 14:13 speaking [1] 43:22 species [2] 28:5; 53:20 specific [16] 8:6; 14:12, 21; 17:8; 18:8; 19:6; 29:1; 35:22; 39:13; 49:17; 50:18, 19; 115:4, 8; 118:9; 139:10 Specifically [3] 50:17; 112:8; 132:17 specifically [19] 5:7; 7:20; 8:7; 12:12; 14:9, 16; 16:10; 19:13; 32:21; 45:2; 50:12; 51:6; 72:14; 90:12; 96:7, 14; 102:21; 115:22; 141:9 specifying [1] 96:19 speculated [1] 91:13 speculation [1] 101:10 speculations [1] 91:15 speed [2] 105:18, 21	spelling [1] 15:5 Spencer [1] 120:12 Spencer's [2] 144:8, 9 spill [9] 115:11, 14; 116:12; 117:18, 20; 118:1, 4; 119:13, 15 spilled [5] 115:12; 117:19; 141:18; 142:5; 150:17 Spills [1] 116:13 spills [10] 79:17; 80:10; 116:15, 22; 117:4, 9; 119:4; 147:3; 150:16, 22 spirit [1] 42:6 split [1] 78:20 sponsored [2] 65:18, 21 springs [1] 41:2 stack [1] 115:22 stainless [2] 142:10; 143:8 standard [2] 13:15; 61:11 standing [1] 150:10 standpoint [1] 163:4 start [3] 46:13; 104:14, 17 started [2] 104:16; 145:14 state [5] 41:16; 49:5; 113:19; 134:13; 150:7 stated [4] 109:3; 113:1; 114:12; 149:19 statement [7] 54:9; 111:14; 118:9; 136:2, 4; 158:12; 164:10 statements [1] 134:3 states [1] 147:16 Statistical [2] 68:13; 70:17 statistical [6] 67:21; 68:8, 17; 69:6; 70:14; 131:7 statistically [7] 67:20; 69:22; 70:7, 10, 22; 71:7; 95:11 statistics [1] 92:21 steady [1] 49:4 steel [2] 142:10; 143:8 stem [1] 101:4
---	--	--	--

- S -

safe [3]
66:9; 97:17; 98:5
safety [6]
78:13; 79:1; 97:22;
162:1, 3, 6
salary [1]
78:14
sample [4]
10:18; 116:8, 9, 11
samples [20]
7:16; 8:1, 3, 10, 11,
12; 9:7; 12:1, 4, 6;
114:10; 144:17;
145:3, 5; 146:8;
147:13; 148:6, 8, 11
satisfy [1]
38:2
save [1]
165:15
spying [15]
18:19, 21; 19:4, 9, 10;
20:22; 25:19; 57:20;

From step to translation

translocation [1] 39:1	116:13; 117:1	152:20	154:18	134:21
transmittal [1] 104:11	uncontrolled [1] 49:19	- V -	We'll [7] 23:10; 53:21; 64:12; 82:17; 120:7; 151:16; 165:15	witness [1] 4:4
traps [1] 142:11	underestimated [1] 133:2	vague [1] 21:9	we'll [2] 21:8; 165:12	witnesses [1] 144:4
Travis [6] 89:6, 14; 90:14; 91:21, 22; 160:5	underestimation [1] 65:16	valid [6] 57:11, 13, 20; 63:4; 66:17	We're [3] 4:11; 54:2; 108:2	Wong [8] 45:7; 46:11; 51:8, 9; 52:4; 125:8, 9; 160:1
Travis's [1] 113:10	underlying [1] 132:7	value [3] 55:20; 86:15, 17	we're [11] 51:17; 53:19; 60:6; 72:6; 86:7; 94:14; 106:13; 149:16, 17; 150:7	Wong's [2] 52:7, 10
treated [2] 133:12, 15	undermine [1] 62:22	values [7] 86:15, 20, 21, 22; 87:2, 3, 9	we've [12] 54:15; 93:8; 96:15; 144:18; 145:9; 146:7; 21; 155:3, 7; 157:4; 159:17; 164:9	Wood's [2] 105:18, 21
trench [26] 12:9; 13:5; 114:8; 115:5, 13, 18; 116:7; 122:21; 123:2; 124:2, 9; 140:5; 142:5, 10, 18, 20; 143:6, 15, 17, 19; 144:5; 148:22; 150:8, 9, 11	underrepresents [1] 86:6	vapor [1] 140:19	weak [2] 73:1; 74:9	word [3] 11:18; 26:12; 89:19
trenches [2] 114:15; 139:4	understand [26] 8:17; 20:20; 21:7, 8; 22:2, 6; 26:9; 29:5; 39:19; 40:22; 43:20; 44:14, 16; 48:18; 73:7; 78:4; 102:5, 6; 138:5; 141:3; 142:18, 163:3, 6, 8; 165:11	vaporize [1] 150:4	weaknesses [1] 111:17	wording [1] 68:21
trial [25] 16:1; 21:17; 26:5; 27:3; 41:14; 42:9; 55:17, 18; 92:1; 93:20; 100:14; 141:12; 157:22; 158:2, 5, 8, 17; 159:2, 5; 164:2, 6, 11; 165:20	understanding [17] 7:1; 21:6; 22:3; 25:7; 26:13; 36:19; 37:18; 48:9; 58:2; 66:4, 5; 80:10; 116:13; 122:6; 141:6; 146:22; 163:1	varied [1] 22:8	wear [2] 79:16; 80:12	words [14] 48:21; 70:22; 73:4; 89:15, 18; 90:3, 6, 8, 9, 11; 112:11; 122:1, 2, 13
Tricontinental [1] 16:9	unfortunate [1] 154:8	varies [1] 21:1	week [1] 16:4	work [20] 4:20; 7:7; 56:11; 65:21; 77:5; 99:2; 107:8, 9, 10; 113:20; 116:14; 119:8, 9; 129:17, 18; 145:18; 146:22; 156:15; 163:7, 8
trivially [1] 147:11	unfortunately [1] 61:9	vary [1] 121:13	weighted [1] 148:12	work-month [1] 49:1
true [8] 37:9; 49:4; 62:15; 63:4; 68:5; 124:8; 135:17; 139:21	uniquely [1] 163:17	vast [4] 88:6; 154:5; 157:13, 15	well-conducted [1] 59:16	work-year [1] 49:2
truth [3] 66:6, 7; 67:4	unit [5] 12:10; 13:5; 123:8; 124:3; 141:18	velocity [3] 140:20; 142:13; 151:3	weren't [1] 157:1	worked [6] 104:18; 114:8; 130:4; 135:18; 138:15; 147:8
tube [4] 10:13, 16, 18, 20	Units [1] 11:3	ventilation [2] 140:21; 143:20	West [3] 5:5; 149:21; 150:7	worker [2] 48:1, 17
tuhes [3] 10:8, 10, 11	units [2] 10:22; 11:2	Vern [1] 133:21	What's [9] 32:3; 51:11; 54:9; 58:9; 88:4; 94:5; 104:3; 110:17; 121:6	worker's [1] 148:6
Turkey [1] 49:20	University [2] 120:21; 121:2	version [4] 32:17; 33:4; 34:21; 117:16	whatsoever [2] 96:8; 129:1	workers [30] 32:13, 14; 36:21; 45:8, 9, 12, 18, 19, 20, 22; 46:1, 12; 50:21; 51:22; 62:9; 77:17; 78:5, 6, 9, 10, 11; 80:15; 83:4, 10, 11, 13; 108:6, 11; 125:8; 161:2
two-hour [1] 106:19	unknown [1] 41:7	versus [2] 16:9; 72:7	Where's [1] 7:5	working [13] 8:4; 55:4, 12; 78:13; 104:14, 16; 114:13; 120:2; 128:14; 145:22; 147:9; 148:16; 156:8
type [4] 14:12, 21; 31:3; 143:14	unlikely [2] 71:1; 114:14	via [1] 128:3	Whereupon [2] 4:2; 166:2	workplaces [3] 84:15, 20; 85:11
types 131 18:18; 37:5; 70:4	unreliable [1] 113:7	view [4] 66:8; 70:14; 74:5; 93:6	Whichever [1] 153:7	works [1] 67:17
typewritten [1] 117:16	unusual [2] 80:11; 147:3	Vigliani [1] 54:17	white [1] 25:20	workweek [3] 48:3, 7; 49:1
typical [1] 164:13	update [8] 15:14; 46:17, 22; 59:8; 89:3, 11; 113:10; 160:5	Virginia [3] 5:5; 149:22; 150:7	who's [1] 102:7	world [3] 101:15; 144:20; 163:9
- U -	updated [1] 103:21	virtually [1] 79:10	whoever [1] 8:14	worry [1] 35:13
Uh-huh [2] 110:17; 139:18	upper [3] 113:18; 146:2; 147:1	volume [5] 10:15; 94:6; 108:3; 149:16, 17	whomever [1] 150:15	wouldn't [3] 72:12; 91:12; 136:12
unable [1] 157:16	upset [3] 12:9; 13:4; 115:7	volumes [1] 153:15	William [2] 105:12; 118:13	write [1] 107:1
unavailable [1] 38:16	upsets [4] 116:22; 118:8, 15; 119:1	- W -	wind [5] 140:20, 21; 142:13; 151:3	writing [2] 41:19; 91:19
unchanged [1] 47:20	urethane [1] 92:11	Wait [1] 109:14	window [1] 56:12	
uncommon [2]	usual [1] 148:16	wanted [2] 144:19; 161:13	WITNESS [16] 6:19; 29:11, 16; 30:3; 42:22; 50:13; 56:22; 59:2; 76:4; 101:8; 107:8, 11; 116:8; 140:10; 153:9; 165:22	
	utilize [7] 46:18; 71:17; 78:17; 83:3, 6; 84:8; 88:14	waste [1] 140:3	Witness [1]	
	utilizing [1]	wastewater [4] 114:8; 139:5, 8; 140:13		
		wasting [1] 12:22		
		water [5] 140:15; 141:7; 143:19; 149:3		
		ways [1]		

Written [1] 9:20	95:13; 161:1		
written {18} 9:18; 31:6; 99:9, 18; 148:2; 158:1; 159:15, 16; 160:1, 4, 7, 9, 12, 17, 18; 161:1, 4; 163:10	you've [12] 4:13; 20:5, 6; 22:4; 26:6; 30:10, 16; 54:19; 149:16, 19; 150:13; 154:17		
wrong [1] 156:13	Young [1] 61:8		
wrote [1] 82:15	younger [1] 42:3		
- X -	yours [2] 32:18; 165:14		
Xerox [1] 35:5	yourself [1] 26:22		
- Y -	- Z -		
Yeah [1] 114:19	zero [1] 73:18		
yeah [1] 126:6	zeroing [2] 95:17, 19		
year [8] 46:2; 62:22; 82:20; 126:5, 7, 8; 147:9			
years [72] 14:2, 5, 6; 19:3; 28:10, 15; 32:22; 41:11; 45:3; 55:6; 59:22; 60:4, 5, 8, 10, 15; 61:14, 17, 18; 62:21; 63:7; 64:4, 5, 6, 13, 20, 21; 65:2, 9; 66:9; 67:18; 72:11; 73:4, 6, 16; 74:6, 19; 75:2, 19, 20; 76:11; 93:5, 10; 98:10; 99:11; 107:20; 108:8, 12; 109:18, 21; 110:16, 19; 124:18, 22; 125:3, 6; 133:8, 9; 135:18; 138:10, 19; 145:8, 22; 146:1, 3, 4, 11, 14; 147:6; 148:4; 160:19			
yellow [1] 153:18			
Yesterday [1] 58:15			
yesterday [5] 106:4, 14; 116:1; 117:7; 150:16			
Yin [11] 84:6, 7, 9, 10; 88:14, 22; 89:9, 11; 107:17; 126:15; 160:5			
Yin's [2] 126:13, 14			
You'd [1] 131:10			
you'd [4] 18:16; 89:18; 109:5; 111:16			
You'll [1] 87:19			
you'll [6] 15:22; 21:16; 26:4; 41:13; 144:6; 165:16			
You've [6] 23:3, 6; 43:2; 60:19;			