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, :
Plaintiffs, : Civil Action
v. : Number
MILES INC., an Indiana Corporation, : 93-C-226 K
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 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 --

APPEARANCES (CONTINUED):

JOE G. HOLLINGSWORTH, ESQ.
BARBARA A. MILNAMOW, ESQ.
Spriggs & Hollingsworth
1350 Eye Street, N.W.
Washington, D. C. 20005-2305
On behalf of Defendant Miles Inc.

P R O C E E D I N G S

Whereupon,

DAVID H. GARABRANT

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

EXAMINATION

BY MR. HARTLEY:

Q Doctor, would you tell us your full name.

A David H. Garabrant.

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

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

A Yes. Actually, I prepared a list of what I

reviewed, which needs to have a couple of things added to it.
Q What does it need to have added?
A It needs to have added this tabulation of John Lavender's work assignments and this deposition by Herman Ring and this deposition by James Taylor.
Q Anything else?

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A No.
Q Is there a legend on here somewhere to describe what MR II is? Do you know what MR II is?
A I believe it is one of the plants in the Mobay facility in Atrium, West Virginia.
Q I figured that too, but do you know specifically what plant it is?
A I do not know -- I can point to it on a map. I do not know what is made in this plant.
Q How about in the polyol, p-o-l-y-o-l? Have you considered this at all in your determination for today?
A Yes, I have.
Q Can you tell me what the polyol, p-o-l-y-o-l area of the plant is?
A I believe that's a facility that handles polyols, which is a common term for polymers of alcohols, also called glycol ethers or polyethers.
Q Do you have a map someplace of the plant?
A I do not have a copy of the map.
Q Do you know what building polyols are in?
A I could point to it on the map.

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Q But sitting here, you have no independent recollection of what building it's in; correct, other than if you see a map it would refresh your recollection?
A I could draw it.
Q Okay. Give me a drawing.
MR. HOLLINGSWORTH: Dean, while he's drawing this diagram of the plant, he should mention -- I think also he's probably neglected to mention that he looked at the industrial hygiene data as we referred to it so far in these depositions, which, of course, would include the monitoring data

that we discussed most recently, I think, in Dr. Rose's deposition.

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

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

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

BY MR. HARTLEY:

Q Is the map on here? What is your

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

A North of the polyols.

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

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

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

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

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

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

A I reviewed industrial hygiene data that

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

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

A I do not recall if there were any personal

samples made on contract electricians. There were personal samples made on Mobay employees and area samples in a number of areas throughout the plant which are relevant to the determination of exposure of whoever is in the area.

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

A I do not understand.

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

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

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Q Quantitywise?

A I'm sorry?

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

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

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

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

Q Yes.

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

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

A Do you mean written notes?

Q Yes, sir.

A Written notes, no, I did not.

Q Do you recall the lowest?

A I do not recall the lowest either. I

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believe that the lowest was below the limit of detection and on some of those, I think that that was in the range of .01 parts per million.

Q What was the limit of detection for benzene?

A Using what method?

Q What methods were used at the Mobay plant?
A There were charcoal tubes, and there was an HNU detector.
Q Are you familiar with charcoal tubes?
A I am somewhat familiar with charcoal tubes.
Q Are you familiar with what the lowest amount of benzene a charcoal tube can detect?
A I do not know the literature on that. It certainly has some dependence on the volume of air that's drawn through the tube, and so I don't have a single number. To some extent, it depends on how the sample is taken as well as on the tube.
Q Do you have any recollection of whether a charcoal tube can detect benzene out to 3 decimals, decimal places, .001, .0012?
A What units?

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Q I'm sorry?
A What units are you referring to?
Q Units of parts per million.
A I do not know -- I do not know. The lowest numbers I recall seeing were .01 PPM. I didn't see anything lower than that, so I don't know if they can detect .003 or .009.
Q What did you find significant about the air monitoring at Miles in light of Mr. Lavender's disease process?
A I don't want to misinterpret your question. What do you mean by "significant"?
Q You reviewed all the monitoring; correct? Did you find anything significant that would apply to this particular case?
A I found things that were relevant, if that's the term.
Q Are you having a hard time with my word "significant"?
A Yes.
Q What did you find relevant?
A Okay, thank you. There were many, many

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

Q Did you find -- there were likewise samples above that, were there not?
A There were occasional samples that were in the few parts per million range.
Q Did you notice the particular study that was done by the trench on the day when an upset occurred in the MNB unit? Were you aware of that study?
A I do not recall that specifically. I'd be happy to look at that, if you want to direct me to it.
Q Do you have the monitoring data with you?
A I do not.
Q Did you rely on the monitoring data?
A Yes.
MR. HOLLINGSWORTH: Do you want us to get that?
MR. HARTLEY: See if I can do it this way because there's no sense in wasting time.

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MR. HOLLINGSWORTH: We can get it for you.
BY MR. HARTLEY:
Q Do you have a recollection that there were studies done indicating when an upset occurred at the MNB unit, the monitoring above the trench was between 5 and 50 parts per million of benzene?
A I do recall seeing some monitoring data that was in that range. I don't recall the incident around which the monitoring was done, but I do recall that in that range.
Q Would you agree that is a high level of benzene in the air?
A I don't know what you mean by "high."
Q Would you agree it's above the OSHA standard?
A Yes.
Q Do you agree that level of benzene could cause leukemia?
A No.
Q What level do you think, Doctor, is necessary to cause leukemia?
A I think that the epidemiologic literature

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

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

A Acute monocytic leukemia specifically?

Q Yes.

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

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

A For acute monocytic leukemia?

Q Yes.

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

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

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

A Yes.

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

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

A I don't recall that.

Q Have you searched the literature before the testimony today?

A I did.

Q You didn't come across that new update?

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

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

A I do not recall that article.
Q Do you think you'll have time to read

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Rinsky and Infante's cohort before trial?
A I would certainly like to.
Q When did you last search the literature?
A Last week.
Q Did you come across that?
A Missed it.
Q Let me show you, Doctor, pages 118 and 119 of your deposition in the -- titled Mary Montgomery versus Tricontinental Industries, et al., specifically to the pages that I have marked down at the bottom.
A Yes.
Q In that deposition, did you say there was an association between benzene and acute myelogenous leukemia?
A Yes, I did.
Q Did you include within the acute myelogenous leukemia acute monocytic leukemia?
A It is one of the subtypes that is included in the broader heading acute myelogenous leukemia.
Q During this deposition in May of 1994, did you say that acute monocytic leukemia was related to

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benzene or associated with benzene?
A I don't think that is what I said.
Q What do you think you said in that particular instance?
A I think I said that the broader heading, acute monocytic leukemia, is associated with benzene. I did not say that there was adequate evidence for each of those specific subtypes to establish that they're associated with benzene.
Q The question was asked "is it your opinion that there is sufficient evidence to show that benzene does cause some leukemias?
"Answer: I've answered that and the answer is yes."
That was your answer.
"Question: Which leukemia?
"Answer: Acute myelogenous.

"Question: Anything else?
"Answer: That families of leukemia, which means the leukemias that are derived from the monocytic series of cells.
"Question: Name them."

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You go on to name them and include monocytic. Are you telling me that doesn't mean that monocytic leukemia is related to benzene or associated with benzene?
A What I'm telling you is that the scientific evidence is adequate to establish the association for the family. It is not adequate to establish the association for each specific member of the family.
Q Did you say that here?
A Did I say what there?
Q Just what you told me. The question was asked "which means what?" And you said "the leukemias that derive from the myelocytic series of cells."
You didn't say anywhere in this -- and you can look at it again if you'd like -- that there is not adequate association between each of these individual types under the AML category.
Are you saying that's what your testimony is today?
A What I'm saying today is I consider the scientific evidence adequate to establish a causal

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association between benzene and acute myelogenous leukemia under circumstances of exposure that exceed 20 -- I should say 50 to 200 parts per million years assuming adequate latency is met. I am not saying that the scientific data are adequate to establish a causal association for each of the specific subtypes of leukemia that are categorized under the heading acute myelogenous leukemia.
Q That's what you're saying today?
A That is what I'm saying today, and I believe that's consistent with what I told Mr. Nace.
Q And you haven't reviewed Kenny Crump's article in '94 which discusses specifically acute monocytic leukemia?

A To my recollection, I have not seen that article. If you might let me look at it, I might recall having seen it.
Q Do you have a list of all the articles you have reviewed?
A I do not have a list. I have brought them with me.
Q Do you want to take a look through your

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binders to see if it's in there?
A I have.
Q It's not in there?
A No.
Q Would it be fair to say you've included every article you've read for today's deposition in these binders?
A Yes.
Q Does individual susceptibility have any part in the development of leukemia as it relates to benzene?
A I'm not sure what you mean by "individual susceptibility."
Q What does that term generally mean to you? Some people are more susceptible than others? Some people may have a development of a disease process, regardless of what the toxin is, at lower levels than the person next to him who may have a disease process or no disease process at a higher level? Do you understand individual susceptibility?
A Let me try to rephrase what I think you're saying, that people's susceptibility to the

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leukemogenic effect of benzene varies such that some people are more susceptible to that than others. Is that what you mean?
Q I'm asking you if -- what is your -- let me do it this way.
What is your understanding of "individual susceptibility," if you understand the term, and if you don't understand the term, we'll move on.
A Well, it's a vague term.
Q Is it in the literature?
A I don't know if it's in the literature or

not.
Q Is it in the benzene literature?
A I'm not aware of it in the benzene literature.
Q Do you think you'll be looking at the benzene literature between now and trial to see if that term is in there, because I'm representing to you that it is.
A Put it this way. In the epidemiologic literature, I'm not aware that there is any support for the notion that individual susceptibility to

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leukemia explains risk.
Q I understand that. I understand that completely. But what is your understanding of the term "individual susceptibility" since you've now used it in your own answer.
A Well, again, I'm not sure I understand what you mean by it. The question I asked you was whether you meant that individuals varied in their susceptibility to leukemia in terms of the dose of benzene that would cause that. That's what -- I don't know what you mean.
Q Is that your definition of individual susceptibility, what you just said?
A I don't have a definition of it in this setting because I'm not sure what you're asking me. There are myriad definitions of individual susceptibility, and I'm not sure what it is you're trying to ask me.
Q Have you ever used the term "individual susceptibility" before?
A I don't recall whether I've used that term or not.

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Q Have you used that term in the TDI cases?
A Have I used that term in the TDI --
Q You've done TDI cases in the past, haven't you?
A What do you mean "done TDI cases"?
Q You've testified as an expert in the TDI cases; correct?
A I don't recall testifying in a TDI case.

Perhaps you could refresh my memory.
Q We'll do it at a later time. How about in an MDI case? Have you testified in an MDI case?
A I don't recall testifying in an MDI case.
Q Have you testified in any isocyanide case?
A Not to my recollection.
Q Have you reviewed TDI cases for litigation purposes?
A I believe I have.
Q And you didn't testify in those cases?
A Not to my recollection.
Q What about the MDI cases?
A What about them?
Q You did not testify -- you reviewed those

cases for litigation purposes?
A I don't recall whether I've ever reviewed an MDI case for litigation purposes or not. I've seen such cases in clinic. And I've reviewed their records. I don't remember if I've done that in support of litigation or not.
Q Isocyanide cases, have you reviewed any isocyanide cases generally for litigation purposes?
A I think I just said I've reviewed TDI cases, and TDI is one of the isocyanides, so the answer is yes, I think I've reviewed records on TDI for litigation purposes.
Q Did you prepare reports in those cases?
A I do not recall whether I did or didn't. I know I've seen lots of them in clinic, and have prepared clinical summaries. I don't remember if I've prepared documents in addition to that. I've certainly done clinical summaries.
Q How does benzene damage the bone marrow, do you know? Let me ask you this first. Is benzene known to be a myelotoxic agent?
A Benzene is known to damage the bone marrow

under circumstances of very high exposure.
Q And how does that occur?
A Do you mean what are the cellular mechanisms and biochemical mechanisms?
Q Yes.

A I don't know.
Q What is your understanding, other than the fact that at high levels it causes bone marrow problems? Do you know anything else about how benzene affects the bone marrow?
A I know at high exposure levels, it can depress erythroparesis. It can cause pancytopenia. It can cause aplastic anemia.
Q Anything else?
A I think that's it.
Q Does it cause leukopenia?
A That would be part of pancytopenia, yes.
Q Can it cause leukopenia without total pancytopenia? Are you saying -- can it reduce the white blood count by itself?
A In the absence of reductions of the other cell lines?

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Q Yes.
A Actually, I've never studied that. I don't know.
Q Will that be something you'll look at before trial, too?
A Well, you've intrigued me. I'll probably take a look at that.
Q Is benzene a mutagenic agent?
A I'm sorry. I didn't quite understand the question. What agent?
Q Mutagenic. I can never pronounce the word.
A My understanding of that area, of the scientific literature is it's been very difficult for laboratory scientists to demonstrate that benzene is mutagenic. And I remember it took a long time to develop an appropriate animal model or model in animal cell lines to demonstrate that.
I do not actually know the details of the circumstances under which mutagenicity of benzene has and has not been demonstrated.
Q Are you making a note to yourself, Doctor?

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A I am.
Q Do you intend to review that subject before

trial?
A Briefly.
Q How about chromosome damage? Does benzene cause chromosome damage?
A I'd have to say again, that's an area where I don't know the literature in great detail. My recollection is that there have been reports of chromosome changes related to benzene, but again, I did not review the molecular and genetic literature on the effects of DNA in preparation for this deposition.
Q That doesn't come into your equation as to whether there's a causal relationship between Mr. Lavender's exposure to benzene at the Miles facility and his subsequent development of acute monocytic leukemia?
A The answer is it plays a fairly modest role. I'm certainly interested in whether there is biologic plausibility for the association between benzene and leukemia, and consideration of biological

plausibility does play a role in my assessment of whether that association is causal.
The actual mechanisms by which benzene might cause leukemia in humans and in other animal species are fairly detailed, and I did not review them in depth -- I did not review them in preparation for this deposition.
Q Have you ever reviewed them?
A I've read some of that literature, yes, over the years.
Q How long ago -- when was the last time you read that literature?
A I don't recall the last time I read any of that literature. I know I've seen that literature on any number of occasions over the past 15 years.
Q Did you review that literature for the Bradley deposition, the Mary Montgomery deposition by Mr. Nace?
A The literature on genetic damage and mechanisms of carcinogenicity of benzene?
Q That's correct.
A No.

Q Have you ever testified in a specific pure benzene case prior to Bradley?
MR. HOLLINGSWORTH: Objection. Bradley wasn't a pure benzene case.
MR. HARTLEY: I understand that. I'm not implying that it was. I'm looking to see if he testified in a pure benzene case --
MR. HOLLINGSWORTH: Even though your question implied that it was.
MR. HARTLEY: I apologize that it was.
THE WITNESS: I don't believe I have.
BY MR. HARTLEY:
Q You have never testified in a pure benzene case before this one; correct?
MR. HOLLINGSWORTH: Same objection.
THE WITNESS: To my knowledge, I've never testified in a pure benzene case.
BY MR. HARTLEY:
Q Is this a pure benzene case? Benzene is not part of a solvent. It's not part of a gasoline. It is basically released into the atmosphere by the process of making MNB. Would you agree this is a

pure benzene case?
MR. HOLLINGSWORTH: Objection.
THE WITNESS: I'm not sure whether this is a pure benzene case or not. I've been asked to review the epidemiologic literature on benzene and leukemia. I'm not sure I know the breadth of the issues in the case.
BY MR. HARTLEY:
Q Other than the Bradley gasoline case, can you tell me what other cases you've testified in that concern benzene in any way?
A To my recollection, none.
Q Have you testified in any cases concerning benzene in solvents?
A To my recollection, no.
Q Was Bradley the very first case that you've testified in concerning benzene exposure and the development of leukemia?
A Yes.

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

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A I believe so.
Q Was Bradley the first case that you testified in concerning benzene and any type of blood disorder?
A I believe so.
Q Have you written anything on benzene?
A I'd have to look at my CV.
Q Do you have it with you?
A I do.
Q Take a look at it and see.
A Yes.
Q What was that?
A I coauthored a report on electric and magnetic field exposures, chemical exposures and chemical risks in electrical occupations.
Q Which one is that?
A Number 49, to the Electric Power Research Institute. That study included some discussion of benzene, I believe.
Q Excuse me, Doctor. Just a second. When was that published?
A That was published in 1992.

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Q What page are you on?
A I'm on page 12.
Q I don't have that on this. What's the date of your CV?
A November '94.
Q I've got July of '94. That's what I was provided.
A Can I assist you?
Q Sure.
A 43.
Q 43 is now 49?
A Yes. I also authored an article looking at mortality in shoe and leather workers, cancer mortality in shoe and leather workers in Massachusetts.
Q What number was that?

A It's 5 on my version. I think it's probably 5 on yours. Those numbers don't change.
Q Yes. 1984?
A Yes. That's certainly relevant to the topic of benzene. I can't recall specifically what it says about benzene in that it's been 11 years.

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Q And the article or the study that you did for the Electric Power Research Institute in '92, that was not peer reviewed, was it?
A That version of it was published by EPRI with an internal review. We actually did publish a peer reviewed paper which came out in 1994, which is number 37 on your copy of my CV, and that derived from that same report.
Q When I compare the 37 on my CV and the 43, "chemical exposures" are missing from the peer review study. Can you explain that from the title anyway?
A Well, I don't know why we chose to edit the title, other than to make it shorter. The fact is that the study was designed to look at chemical exposures among people who held jobs that had electromagnetic field exposure to determine if those chemical exposures confounded the relationship between EMF and leukemia.
Q Did you come to any conclusions?
A Yes.
Q Which were?

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A That the chemical exposures did not confound that relationship.
Q Based upon what?
A Based upon an assessment of which occupations had chemical exposures and analyses that showed that they were not -- that they didn't appreciably confound the relationship.
Q Was it because the individuals who allegedly developed leukemia from electromagnetic fields were not exposed to chemicals that could cause or could be associated with leukemia?
A My recollection is that the prevalence of exposure was low and that it didn't act as a

confounder, and so there was not founding.
Q The chemical exposure?
A The chemical exposures did not act as confounders, that's correct.
Q Do you have a copy of that study, Doctor, that I can get from you, since it's not published in the open literature?
A The EPRI version of it?
Q Yes.

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A Yes, I do.
Q Do you happen to have it with you?
A No, I do not.
Q Can I get a copy of that from you?
A Yes, I'll have to Xerox it. It's a big thing.
Q That's okay. You can bill me for it.
A That can also be purchased directly from EPRI. You can phone him up --
Q In Palo Alto?
A Sure. It's one of their publications. You can call them up.
Q I'll do that. Don't worry about it. What do you have on your list so far --
A Of things to do?
Q Yes.
A I need to get the Crump article from the Journal of Toxicology and Environmental Health, 1994. I need to look and see whether benzene causes leukopenia in the absence of pancytopenia, and I need to review if benzene is mutagenic.
Q What specific chromosome, if any, does

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benzene affect?
A I don't know.
Q Does benzene cause -- have you looked at the literature at all with regard to benzene in chromosomes?
A Not in a systematic matter, no.
Q Have you done any of that review for today's deposition, systematic or nonsystematic?
A No, I haven't.
Q You're not aware of the new studies out

discussing chromosome damage from benzene that were published in '94 and '95?
A I'd have to say I don't believe I have.
Q Are you going to rely on any of the chromosome damage in this particular case to express an opinion that benzene did or did not cause or is associated with Mr. Lavender's AML?
A No.
Q Do you have any understanding at all of the significance of chromosome damage from benzene in exposed workers? I'll change "significant" to "relevant" for you.

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A I'd have to say that's not an area that falls within my -- that part of the literature doesn't fall within my area of expertise. I know I have seen some articles on that that discuss chromosomal changes in the various types of leukemia, but I would have to say I have very little knowledge of that area.
Q So you don't have an appreciation of the true significance or relevance of the literature concerning chromosome damage as it relates to benzene exposure, do you? You know it's out there, but you don't really know what the significance of it is in a cause-and-effect relationship? Go ahead. I'm listening.
A My knowledge of that area is limited, and I did not review that in preparation for this deposition.
Q Do you think an understanding of that area would help you determine whether there is a causal relationship between Mr. Lavender's benzene exposure and his development of acute monocytic leukemia?
A No.

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Q And why is that?
A Because Mr. Lavender doesn't satisfy the criteria that are necessary to think that benzene was a risk factor in his leukemia at all.
Q But then again, you don't know what the literature says on chromosomes, so you can't totally exclude that, can you, from your equation?

A I think I can based on the strength of the epidemiologic literature.
Q So because of the strength of the epidemiological literature, you don't need to know about the chromosome damage; correct?
MR. HOLLINGSWORTH: I object to the question on the ground that it proceeds from an inference that there is chromosome damage, evidence unavailable in this case at all.
MR. HARTLEY: Sure. Your own doctor said there was evidence of chromosome damage. He didn't think it was related to benzene, but he didn't know the literature on benzene and chromosomes either.
MR. HOLLINGSWORTH: He knew it better than anybody on this planet, but he didn't say there was

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damage. He said there was a translocation.
BY MR. HARTLEY:
Q With all that said, Doctor.
A Would you repeat the question.
Q Basically your opinion is based on the epidemiological study and the lack of association between Mr. Lavender's exposure and his development of acute monocytic leukemia; correct?
A My opinion is based on knowledge of the scientific literature that strongly supports the conclusion that he was not adequately exposed to place him at risk of leukemia.
Q And it is not that specific basis that permits you to exclude a review of the literature which discusses chromosome damage in your analysis?
A That is one of the reasons, and it is the major reason, yes.
Q What are the other reasons? One, that you don't understand it, the chromosome area in the literature?
A No. I don't think that would be one of my reasons.

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Q You do or don't think it would be?
A I don't think that would be. The exploration of a mechanism by which benzene might have caused Mr. Lavender's leukemia is conditional on

his having had adequate exposure to think that benzene caused it.

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

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

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

Q Benzene would cause leukemia?

A Excuse me, benzene would cause leukemia.

Q You don't understand how benzene causes

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

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

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

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

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

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

Q You're writing that one down, too?

A Yes.

Q Okay. How old are you, Doctor?

A How old am I?

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Q Yes.

A 44.
Q You look younger than I do. I'm only 39.
A Thank you.
Q It was a compliment.
A And I took it in that spirit.
Q Will you be looking at the studies that discuss low level exposure to benzene and chromosome damage before trial?
A Probably not.
Q If I would tell you that there are articles out there which discuss exposure to less than 3 parts per million causing chromosome damage, would you find that in any way relevant to the equation of Mr. Lavender's acute monocytic leukemia?
MR. HOLLINGSWORTH: I object to that.
MR. HARTLEY: Assume for purposes that there are.
MR. HOLLINGSWORTH: Are you talking about animal studies?
MR. HARTLEY: Yes.
THE WITNESS: I would find that of --

BY MR. HARTLEY:
Q Let me do it this way. You've reviewed other animal studies in other situations, haven't you?
A I'm not sure what you mean by "other situations."
Q In attempting to determine a causal association, you have reviewed animal studies and have testified that animal studies do provide some evidence of a causal association, have you not?
A I don't recall to what I've testified. However, it is my opinion that animal studies are relevant to the determination of carcinogenicity to humans, so they certainly can be relevant.
Q Do you think that there would be any relevance in animal studies which demonstrate chromosomal changes at below 3 parts per million?
MR. HOLLINGSWORTH: What kind of chromosome damage?
MR. HARTLEY: He doesn't understand the chromosome changes anyway, so it doesn't really

matter. Generally speaking, chromosome damage.

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MR. HOLLINGSWORTH: Object.
MR. HARTLEY: That's okay.
BY MR. HARTLEY:
Q That means you can answer.
A I think they are peripheral to the issue of whether benzene causes leukemia in that the human epidemiologic literature quite clearly does not show evidence of risk at levels of exposure that low.
Q That's because the studies don't have the power to do that, do they?
A No, I would not agree with that answer.
Q The studies are smaller studies, aren't they, that do show a relationship?
A I don't understand that question.
Q Number of people in the cohort.
A I still don't understand the question.
Q What studies have you reviewed for this particular case? Would you itemize them for me, the epidemiological studies.
A Well, I'd have to go through my books. Do you want everything, or do you want the important studies?

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Q I'd like to have the pertinent ones, the ones that you're basing your opinion on specifically that there needs to be 50 parts per million years with an adequate latency period.
A Okay.
Q Can you do that for me.
A Yes. I would include the study by Wong in 1987 of chemical workers, a study by Ott of chemical workers published in 1978. I would include the two studies by Paxton in 1994.
Q What did she study?
A Pliofilm workers.
Q She reevaluated the Rinsky and Infante study. Any other ones?
A Yes. I would also include the studies by Devine published in '85 and '87.
Q Of what cohort?
A Gasoline workers. I would include --

Q Gasoline workers?
A Yes, or I should say refinery workers exposed to gasoline. I would include the studies by Rushton, also of refinery workers and petroleum

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distribution workers.
Q What year was that?
A That was 1993.
Q Was that Rushton by himself or Rushton and Alderson?
A It's a she, I think, and I think it's with Alderson. I'd have to look, but I believe it's both.
Q Is that the study of the eight refineries in Great Britain?
A Yes, it's in Great Britain. I would include Wong in 1995, another reanalysis of the pliofilm workers. And those are the major ones.
I'm sure if we start to talk about some of the issues in detail, I may use other literature in addition but those are the principal studies upon which my answer rests.
Q You did not include the Bond update of the Ott study. Do you intend to utilize that?
A Yes, I should use that as well. Thank you.
Actually, let me add one other. There's a new update by Delzel of the Shell cohort.

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Q That was Thorpe's study earlier?
A I'm sorry?
Q Not Shell. That was Exxon. Go ahead.
A That's 1995.
Q Where is that published?
A JOM.
Q M, you did say M?
A JOM, yes -- excuse me, JOEM, Journal of Occupational Environmental Medicine.
Q Doctor, how is benzene removed from the body?
A I believe it's metabolized to phenol which is excreted by the kidneys.
Q Do you know what the half-life is?
A It's fairly short. I don't have a number,

but it's in the matter of days, at least.
Q Is that the only way for it to be removed from the body?
A Oh, I think you could exhale a small proportion of it unchanged. There are probably other metabolic pathways that are less important than phenol. I don't recall them offhand.

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Q Can the worker exposed to benzene eliminate all of the benzene that he is exposed to over a workweek?
A I would have to look that up. It certainly would eliminate a very large proportion of it.
Q Would some still remain in the bone marrow by the end of the workweek?
A I do not know the literature on that point. My understanding of the kinetics of excretion of benzene are that a very large proportion of it would be eliminated within a few days, which would imply that a very small proportion would be retained. Since it's a lipophilic solvent, I would expect it would be retained in fatty tissues of the body.
Q Do you have an opinion as to whether all the benzene is ever removed if the individual worker continues to be exposed? Do you understand the question?
A No.
Q In other words, is there a cumulative -- is there an accumulation of the benzene in the marrow

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over the course of the workweek or work-month or work-year?
A The answer is no, there's not an accumulation. It would be true that under a steady state exposure, you would reach equilibrium such that the absorbed amount and the amount eliminated by excretion, both of the parent compound and the metabolites, would be in balance, but I don't think that it actually accumulates in any sense.
Q At what level does the bone marrow become damaged?
A I'm not sure what you're referring when you

say "level." Are you talking about air level, blood level, cell level, fat level?
Q At what particular air level of benzene does the bone marrow become damaged?
A I don't think we know a specific number. The literature that addresses that issue comes largely from circumstances of very high uncontrolled exposure, such as in the shoe industry in Turkey, the reports by Aksoy and some from Italy. It's clear from the descriptions in those reports and the small

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amount of measurement of exposure, that the levels are well up in the many hundreds of parts per million.
Q But you have not reviewed the literature concerning lower level exposure and damage to bone marrow, have you? Not the epidemiological literature, but the other scientific literature.
MR. HOLLINGSWORTH: I object to that question. You're referring to animal data again or epidemiologic data?
MR. HARTLEY: Any data. I'm not referring specifically to epidemiologic data on that.
THE WITNESS: The human literature, I do know some of that. So the answer is yes, I do know some of that literature.
BY MR. HARTLEY:
Q Specifically what literature would you rely on concerning that specific point?
A I couldn't cite specific articles offhand. I know there have been a number of studies of hematologic changes among workers exposed to low levels of benzene in industry that do not show any

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affects on cell counts or bone marrow function, and so I can't cite the articles offhand. I could certainly pull those together for you.
Q Did you review them before today?
A I have reviewed that topic in the past. I did not specifically prepare that topic for today, but I'm generally familiar with the area.
Q I notice you didn't indicate the Wong '83 study. Did you review the Wong '83 study?

A I do not know offhand which study that is. What's the citation on that?
Q It was a study done for CMA.
A I don't believe I know that study.
Q Have you ever seen that study cited in the literature when you were doing the --
A Without having a citation to know exactly which study we're talking about, it's hard for me to know if I've seen that cited.
Q I'll see if I can get you a cite. It's not in the open literature. I think it's published by his group. Here it is. "Comments on the NIOSH study of leukemia in benzene workers technical report"

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submitted to Gulf Canada Limited by Environmental Health Associates, August '83.
A I do not know that particular document.
Q So you don't know what Wong said in that particular study, do you?
A No, I do not.
Q Have you reviewed Dr. Wong's testimony before OSHA?
A On what occasion?
Q Have you reviewed any of Dr. Wong's testimony before OSHA?
A I do not recall offhand. I know I've read a lot of things he's said. I don't recall offhand if I've read testimony before OSHA.
Q March 4, 1986 was the date of the submission.
A Was it published in the Federal Register?
Q You know, I don't know whether it was or not. I know it's on the OSHA docket.
A I probably did not read it if it did not come out in the Federal Register.
Q Doctor, do you agree that cytotoxicity is

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associated with the development of cancer?
A Sometimes. Sometimes not.
Q When would it not be?
A There are many instances. We could kill cells by making them hypoxic and that's cytotoxicity, and that's not associated with the development of

cancer.
Q Anything else?
A We could kill them with cyanide. That's not associated with cancer. We could physically burn them. We could disrupt them osmotically. We could come up with a long list of agents and circumstances that were cytotoxic that had nothing to do with cancer.
Q Could we damage them through benzene cytotoxically?
A I suppose you could manage that in the laboratory. I don't know that you can do that in living people, if we're talking about people. Which species are we talking about?
Q We'll talk about people right now. Does benzene cause cytotoxic damage, or is benzene a

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cytotoxic agent?
A In living, intact human beings? We're not talking human cell lines in a lab. I would have to say it can be cytotoxic to some of the cellular elements in the bone marrow under circumstances of extremely high exposure.
Q What do you base that on?
A What do I base that on?
Q Yes. What's the basis for your statement?
A The literature that shows that people who have extremely high levels of exposure can develop pancytopenia and anemia in response to benzene exposure.
Q And what literature would that be, the one that we've talked about here earlier?
A It would include the earlier literature by Aksoy and Vigliani. There's more. I can't recall names of articles offhand.
Q Since you've mentioned Aksoy's studies, do you recall Aksoy's study of leukemia in a family of shoemakers?
A Not offhand.

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Q Let me try to help you recollect a study that talked about a mother and either a son or a daughter -- I think it was a son -- who developed

leukemia very quickly working with their husband and father in the same setting and the father did not develop the leukemia for 12 or 15 years later. Do you recall that article?

A No.

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

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

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

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

a reliable manner.

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

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

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

A No.

Q Why not? It's questionable reliability.

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

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

THE WITNESS: No.

BY MR. HARTLEY:
Q They're not?
A No.
Q Which one is not?
A 1976 was a PMR study.
Q Other than that study, was everything else published because of the question of reliability?
A I'd have to look at everything he's published to see which were scientific studies and which were case reports. I would be reluctant to throw out valid scientific inquiries.
Q Does that mean that case studies can't have valid scientific -- a valid scientific place in the determination of whether benzene can cause acute monocytic leukemia?
MR. HOLLINGSWORTH: Objection; asked and answered.
MR. HARTLEY: He's changed his answer. His first answer was that they have questionable relevance. Now he's saying that they have a valid place in the determination. I just want to know which one it is.

MR. HOLLINGSWORTH: I object to your characterization of the testimony. My understanding of the testimony is that case reports are nonscientific and are of questionable reliability.
BY MR. HARTLEY:
Q Is that your answer, Doctor? If that's your answer, I'll accept that answer.
A That was half of my answer.
Q What's the other half?
A The other half is that they play an important role in generating systematic scientific inquiry.
Q When was the last time you looked at the Ott/Bond study?
A Yesterday.
Q Did you look at the exposure levels that were reported for the Ott/Bond study?
A I believe I did.
Q What did you find?
MR. HOLLINGSWORTH: Can we take a

five-minute break?
MR. HARTLEY: Can we take long enough for
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me to go down and smoke a cigarette?
THE WITNESS: Sure. That would be fine.
(Recess.)
BY MR. HARTLEY:
Q Did you pull out the Ott portion of the study?
A Yes.
Q Have you pulled out Bond's update of the study?
A Yes.
Q Would you look at Bond's table 5.
A Yes.
Q Is it your opinion that the Ott/Bond study is a reliable study?
A I'm not sure what you mean by "reliable," but it appears to be a well-conducted study.
Q Did you look at the exposure data on table 5, the summary of myelogenous leukemia?
A Yes.
Q Have you attempted to calculate what the parts per million months were in parts per million years?
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A Yes, we can do that as we look at it.
Q What is the first one, case number 1, 648 parts per million months?
A In years, that would be about 50-some -- 54 parts per million years.
Q I got 53, but we're close. What about the 18, case number 2?
A That would be 1.5 parts per million years.
Q Case number 3?
A Would be about 25 parts per million years.
Q Case number 4?
A Be about --
Q 346?
A I was going to say 350 parts per million years.
Q And in case number 5?
A About 29 parts per million.

Q I got 28.
A I'm just doing it in my head. You've got to give me a break.
Q I am. I'm confirming what you are saying. I'm in agreement with you.

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MS. MILNAMOW: You used a calculator?
MR. HARTLEY: I used a calculator. In fact, it's 21.89.
BY MR. HARTLEY:
Q Case number 2, age of death was 51. How old was Mr. Lavender when he died?
A 46 -- is that right? Yes, 46.
Q Young man?
A Middle-aged, I believe, unfortunately.
Q I guess it's all relative, isn't it?
A The standard definition says 40s are middle-aged.
Q Case number 2 had developed the leukemia with only 1.5 parts per million years; correct?
A Yes.
Q Can you explain that to me in light of your theory that you need 50 parts per million years and you need a latency period of five years and so forth?
A Yes, I can.
Q Would you.
A We expect to see leukemia in every population, even without benzene exposure, and so the

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fact that one case occurred with short duration of exposure and relatively low cumulative exposure probably has nothing at all to do with exposure.
If we look at table 4, we see that -- in fact, we expected to see two cases of leukemia based on general population rates without benzene exposure, so two of those cases would have been expected to have occurred on their own even if this cohort of chemical workers had never handled benzene at all.
Q So this one, in your opinion, is not a benzene-related leukemia?
A Well, there's no reason to think that -- I should say there is very strong reason to think that we should not conclude all cases of leukemia in a

cohort are due to exposure. If that was true, it would support the conclusion that leukemia doesn't occur in the population in the absence of benzene exposure, which is clearly false.

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

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

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

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

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

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

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

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substantial benzene exposure.

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

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

Q We'll take your hypothesis that you need 50 parts per million years to develop leukemia; correct? That is your hypothesis. Is it 50 or 60 now?
A I believe what I said was that the scientific literature indicates that there is a causal association between acute myelogenous leukemia and cumulative benzene exposure in the range of 50 parts per million years to 200 parts per million years.
Q There's also other literature out there

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that indicates that it's less than 50 parts per million years; correct?
A There is not adequate literature to conclude with reasonable -- with a high degree of certainty that that association is not due to change.
Q Didn't the NIOSH study find that?
A Which NIOSH study are we referring to?
Q The Rinsky study, that it was only 40 parts per million, one part per million for 40 years.
A I'd have to look at the Rinsky study. You and I are well aware that many people have reexamined that cohort and the exposure estimates upon which Rinsky's conclusions were based, and their findings are considerably different than Dr. Rinsky's, indicating that the risk estimates he derived were based on underestimation of the exposures.
Q Of course, the people who reevaluated the literature were sponsored by API, were they not?
A Some were.
Q Do you think that there would be some bias in the work that was done when it was sponsored by API?

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A I would make no such assumption.
Q Would API want the exposure limit to be higher than an independent NIOSH examination, based on your understanding?
A My understanding is that API would want to find the truth, not a biased representation of the truth.
Q Under your view of the literature, and that

is 50 parts per million years, would it be safe to assume, then, that case number 2, case number 3 and case number 5 are not benzene-induced leukemias and that the study is therefore invalid as relating to any showing of a causal relationship because you would only have two cases left and you're only expecting 2.1?

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

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

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

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

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

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

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

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determine a causal association, haven't you?

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

A I think there were two questions there. Could you give me one at a time.
Q Sure. Have you testified that statistical significance is not all-encompassing and not absolutely positively necessary to draw a causal association, yes or no? And then you can explain it.
A Statistical significance is a concept we use to evaluate the role of chance --
Q Let me interrupt you. Yes or no. Then you can explain it. Have you testified in the past that statistical significance is not absolutely necessary to determine a causal relationship, yes or no, and then you can explain it, but I want a yes or no answer. I'm entitled to that.
A I do not recall the exact wording of everything to which I've testified in the past. You

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would have to show me my testimony that says exactly that.
Q I do happen to have this one marked. Page 186 of your deposition.
A And what is the question?
Q Have you testified that statistical significance is not an absolute requirement to determine a causal association?
A I don't believe that characterizes my testimony accurately.
Q What does -- how would you characterize your testimony, Doctor?
A Why don't I read it into the record.
Q Sure.
A I said "when epidemiologists talk about studies, they commonly refer to positive studies as ones that show evidence of an association, whether it's significant or not, and again the context of how they're talking about it is probably more important than the term 'positive study.'"
Then the next question was "you mean you have a positive study that's not statistically

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significant?"
My answer was "there are any number of

references that I've reviewed and listed for you that show positive associations between various types of leukemia and various exposure" -- it should have said "exposures" -- "under study which are not statistically significant. So a positive association or positive study in my experience just means that there is some evidence of an association of whether it's statistically significant or not."

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

Q Okay. Do that.

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

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

Q But it is not the only one; correct?

A It is not the only one.

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

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

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

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

study, whether it's significant or not.

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

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

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

Q What did you say specifically, then?

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

Bond and his colleagues present some of

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

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

A I was referring to table 8.

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

A Yes, 499 parts per million months.

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

A About 41.

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

A Yes.
Q And we had one case over a thousand?
A That is correct.

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Q And it's your testimony that based on table 8, you need in excess of 500 parts per million months of benzene to develop a leukemia?
A It is my testimony that this paper does not support the view that there is increased risk below 50 parts per million years.
Q And the basis for that is?
A That in table 8, there is no -- there is only a weak and nonsignificant association between exposure below 500 parts per million months and leukemia.
Q Where is the association above 500 parts per million months? There isn't any, is there?
A This paper only has three leukemia deaths. It's a small study. It doesn't provide conclusive evidence that benzene is associated with leukemia. It's a small study.
Q How do we take the 50 parts per million years that you're suggesting and apply it to Mr. Lavender? It's an individual case, which -- you can't pick and choose which cases you want to apply it to and which ones you want to throw it out in.

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So how do we apply your 50 parts per million years to Mr. Lavender's case? You couldn't apply it to any one of these cases here and throw these cases out, so how do we apply it to Mr. Lavender, solely on the exposure issue?
A The first step is to determine how much exposure Mr. Lavender had in terms of his cumulative exposure. Then the second step is to examine the epidemiologic literature to determine whether there is sufficient evidence to say that exposures of that magnitude cause leukemia.
The answer to that second issue is there is not adequate literature to conclude that those exposures cause leukemia, and so there is simply no basis for saying that leukemia -- that benzene exposure caused Mr. Lavender's leukemia.

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

so they shouldn't be included in this study?

MR. HOLLINGSWORTH: Objection; asked and answered.

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

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

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

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

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

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

BY MR. HARTLEY:
Q When you do an epidemiological study of the Mobay facility, as you suggested could be done, do you include contract workers as well as Mobay employees in that study?
A You certainly could. There'd be no reason to exclude them. They could be included. They could -- the study could be done with or without them, depending on feasibility issues.

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Q Would the fact that they were not included in a study give you any enlightenment as to disease processes in the nonincluded group? Do you understand my question?
A I believe so. If the contract workers were similar to the workers who were included in terms of their other risk factors for leukemia and were similar in terms of their distribution of exposure, the findings from the workers who were included would be applicable to the contract workers.
Q But if the contract workers were not provided with respirators, were not provided with safety equipment when they were working around benzene and the hourly Mobay people or salary people additionally were, then there would be a difference in exposure, would there not? That way you could not utilize the epidemiological study of the Mobay people to support a lack of causation for the contract employees?
A Could I ask you to split that into two questions, and I'll try to answer them separately.
Q Sure. First question is, if the contract

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employees are not given safety equipment to protect against exposures to benzene, their rate of exposure, potential exposure would be higher than the hourly people who are given the adequate protection and use it; correct?
A Not necessarily, or I should say not necessarily appreciably higher. It's my impression from the industrial hygiene data that the exposures in the Mobay plant were well under 1 part per million virtually all the time.

Q What do you rely on for that?
A I'm sorry?
Q What do you rely on for that?
A The industrial hygiene data, and that that's an exposure level which would not cause people to wear their respiratory protective equipment.
Q How about when there were spills --
A I was still answering.
Q I didn't know that you were. I apologize.
A Insofar as the Mobay employees are not wearing respiratory protective equipment routinely nor are the contractors, their exposure levels in the

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same locations are expected to be identical or comparable.

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

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

Q What is your opinion of the parts per million of benzene in the air when you can smell it?
A Are you asking me what the odor threshold is?
Q Correct.
A I believe it's in the few parts per million range.

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Q Do you disagree with the American Industrial Hygiene Association's geometric mean of 61 parts per million?
A Are you referring to the odor threshold document they published in 1987?
Q That's correct.
A I think that estimate is high.

Q And what do you support that on?
A An estimate made by ASTDR and my own personal experience.
Q Have you seen Paustenbach's explanation of what the odor threshold is in 1992 or '3 in his rereview of the pliofilm?
A I do not recall his discussion of that.
Q The study was responsive by API in that particular study. Do you have it with you?
A Is that the one from Environmental Health Perspectives in '93?
Q The long paper.
A Looks to be.
Q It's toward the end. Did you find it yet, Doctor?

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MR. HOLLINGSWORTH: What page is it on?
MR. HARTLEY: It's toward the end. I know it's at the top. I didn't bring my own copy of the article, but it's at the top, I know, of one of the pages and I believe that he says that the odor threshold is 35 parts per million.
BY MR. HARTLEY:
Q You don't find it in there. Let's assume that he says it's 35 parts per million. Do you believe that would be too high as well?
A Yes.
Q Why would someone from API want the odor threshold to be higher than what it should be, then?
A I really don't know the motives of the people who wrote that.
Q It's in the section right after respirators, I think. That's okay. We'll move on. I don't think this is the right study.
A Oh.
Q It's a longer study. What year is this one?
A '93.

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Q That's not the right study. That's why you couldn't find it.
Why did you utilize the Devine study on refinery for gasoline workers?

A I'm sorry, why did I what?
Q Why did you utilize Devine when it's not a pure benzene situation, or is it?
A It's a compound question. It is not a pure benzene study. It is a study of refinery petrochemical and research workers and a separate study of producing pipeline workers.
The reason I included some of the studies of petroleum workers is that they have low level exposure to benzene in the course of handling petroleum products and so it is relevant to the issue of whether those low level exposures are associated with leukemia.
Q What do you -- how would you calculate a designation of benzene exposure, that is in milligrams per cubic meter, 18 milligrams per cubic millimeter?
A How would I do what with it?

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Q Change it to parts per million. 18.1 would be what?
A Roughly 6.
Q 6 parts per million?
A Yes.
Q Have you seen the Yin studies?
A I have seen studies by Yin, yes.
Q Did you utilize it for this particular case? And I'm talking about Yin 1987.
A Do I have Yin '87? Yes, I do. Industrial page 124?
Q No, 192.
A Yes.
Q Did you notice the geometric mean concentration of benzene in 50,255 workplaces was 6 parts per million, correct, summary of the abstract, Doctor?
A What number did you give?
Q The geometric mean concentration of benzene in 50,255 workplaces was 18.1 milligrams per cubic meter which, in your translation, was approximately 6 percent -- 6 parts per million?

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A That is what he said, yes. However, if you

look at the distribution of exposures, they go -- he only shows them from 10 to 2000 milligrams per cubic meter, so he shows them from roughly 3 to 600 parts per million, and there were appreciable numbers of them, well above 100 milligrams per cubic meter or 33 parts per million.

Q What would 40 milligrams per cubic meter be?

A Approximately 13.

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

A Yes.

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

A For end measurements, it's the product of all measurements to the one over nth power.

Q Which means what, in layman's terms?

A You take all the measurements, you multiply them together, and you take the nth root of that

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

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

Q Why would you use a geometric mean?

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

So for example, if you and I had exposure data where we had 100 values that were between 1 and 5 and two values of 2000, an arithmetic mean would be

moved up by those values. A geometric mean is not

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

Q What percent would that be, in your opinion?

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

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

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

Q Yes.

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

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

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

A What's the relevance of that?

Q Yes.

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

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

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

Q That's what I meant.

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

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

Q It was a peer reviewed paper, wasn't it?
A It's in a very fine journal that is a peer review journal.
Q Yin had follow-up, didn't he? Did you see

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the follow-up studies that were done on his cohort?
A To what are you referring?
Q For one, there was a 1994 update on leukemia and lymphoma. Do you have that one?
A Are you talking by Lee?
Q No, by Travis. Do you have that?
A I do not believe I have that one.
Q When you were doing your search, since you knew that the Yin study in your own opinion was somewhat questionable, did you do your Medline search to see if there was any further update on the Yin study?
A I did my Medline search to look for new literature on benzene and leukemia. If the Travis study had been assigned the appropriate key words, I probably would have found it.
Q "Benzene," "leukemia," "dysplasia," would those be key words you'd be searching for on Medline?
A I didn't use the word "dysplasia." I did use "benzene" and "leukemia."
Q Would "benzene" and "leukemia" have been picked up in this particular article and would you

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have found it on your Medline?
A I couldn't tell you without getting the article and seeing what the key words were under which it was indexed.
Q It was under -- "benzene," "leukemia" and "dysplasia" were the key words. I've got it in front of me.
A Those are the Medline key words? Those aren't the Medline key words. That's not necessarily what the National Library of Medicine entered for it. Those are -- I used a number of key words. I remember specifically using "leukemia" and "benzene." And I do not remember seeing a citation by Travis.
Q You didn't find that article either;

correct? That didn't come up in your search?
A That's correct.
Q Do you believe that Mr. Lavender's smoking history caused or contributed to his development of acute monocytic leukemia?
A Yes, I do.
Q And what is it in tobacco smoke that causes

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leukemia?
A I'm not sure we know that with certainty. The epidemiology points consistently toward an association between smoking and leukemia. We do know that benzene is present in cigarette smoke. I do not believe we know that that is the reason why smokers have elevated risk of leukemia, however.
Q Has anyone published an article saying what the cause may be in cigarette smoke for leukemia that you're aware of?
A I'm not aware that anyone has meaningful data on that. I wouldn't be surprised if lots of people have speculated as to the cause, but I don't know that anyone has meaningful data.
Q Do you know what the speculations are?
A We could look at some of the articles and see, if you want to do that.
Q Sure. Let's do that. Which articles do you want to look at on smoking? You're writing down something else. What are you looking for now?
A Travis.
Q Will you find the Travis article before

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trial?
A I'd like to see that.
Q Did you come across the Robert Snyder article in your Medline search?
A To which one are you referring?
Q 1994.
A I have Critical Reviews in Toxicology.
Q Yes.
A Yes. Well, now, one article by Michael Segal postulates "at least three chemical leukemogens (benzene, urethane and nitrosamines) are present in tobacco smoke." In addition, the concentration of

lead 210, a radioactive leukemogen, has been demonstrated to be significantly increased in the bones and soft tissues of smokers, so there are a number of plausible hypothesis for that association.

Q Benzene is plausible?

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

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

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

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

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

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

A I do not.

Q You didn't calculate that?

A I did not calculate that.

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

A I'll put it on the list.

Q What is the relative risk of leukemia in

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

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

Q What's the cite of Segal?

A American Journal of Epidemiology, volume 138, page 1, 1993, pages 1 to 9. The pooled odds ratio for myeloid leukemia in Segal based on a summary of nine studies is 1.23 with confidence

limits that go from 1.08 to 1.39.
Q 1.08 to --
A 1.39. Those are case control studies.
Q Well, we know that those are not really relevant when we're talking about epidemiological studies, are they, because we threw them out when we were talking about Aksoy. So the case control studies that Michael Segal bases his analyses on would not really have any relevance, would they, in good epidemiology?
A I suspect that you're mistaking case control studies for case reports. Case control studies are scientific studies, and they are

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different than case reports, so we would not throw them out.
There are also six cohort studies, four of which examine myeloid leukemia risk and find a pooled relative risk of 1.51 with 95 percent confidence interval that goes from 1.31 to 1.74. So both case control studies and cohort studies demonstrate a significantly elevated risk of leukemia associated with smoking.
Q Significant?
A Yes, statistically significant.
Q But it is really only a modest relative risk. You've only increased it how many, .23 above 1 and .5?
A I would call it a modest risk.
Q The reports are not discussing acute monocytic leukemia, but are rather zeroing in on acute myeloid leukemia; correct?
A They are zeroing in on myeloid leukemia, and that is correct.
Q And that is not the same as acute monocytic, is it?

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A It includes acute monocytic. Now, that -- I'm not sure the intent of your question. If it's to say that this literature is not relevant to the determination of smoking risk to acute monocytic leukemia, then the same argument is made of the benzene literature, that very little of it examines

acute monocytic leukemia specifically, and there's almost no data whatsoever showing that's associated with benzene.

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

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

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

A It references myeloid leukemia without specifying subtypes.

Q Did they try to break it down into subtypes?

A To the extent of my recollection, they did

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not break it down any finer than myeloid leukemia.

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

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

Q Why can't it be done?

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

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

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

A With respect to what outcome?

Q Any outcome.

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

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of different limits that we could talk about.

Q Let's talk about human disease or human cytogenetic studies or human changes within the body

as a result of benzene exposure, changes in the human body. Is there any safe level of benzene to the human as far as medical disease processes are concerned?

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

Q Are you familiar with McDonald studies, Gene McDonald?

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

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

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

Q Dr. McDonald is with NIEHS. Are you not

familiar with those studies?

A Is that published work?

Q Yes.

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

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

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

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

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

Q Have you read his literature?

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

Q Are lymphocytes affected by benzene exposure?

A I'm sorry?

Q Are lymphocytes affected by benzene

exposure?
A I'm not sure what you're asking. Benzene exposure is adequate to cause pancytopenia and certainly affects the lymphocytic series of cells. Other than that --
Q What is the medical term for a decrease in the number of lymphocytes?
A Lymphocytopenia.
Q Is lymphocytopenia associated with benzene exposure absent pancytopenia?
A I would have to look at that issue. I'm not aware that it is.
Q Is that another issue you're going to look at before trial?
A Yes.
Q Do you believe, Doctor, that one exposure to benzene can cause leukemia to evolving?
A I'm not aware that there is any support for that hypothesis in the scientific literature and so I'd say there's no basis for making that conclusion.
Q Is that a proposition that is floating out in the scientific community?

A I don't know whether it's floating out there or not. It's not floating in my office.
Q Can one molecule cause mutation of a particular stem cell?
MR. HOLLINGSWORTH: One molecule of benzene?
MR. HARTLEY: Benzene, yes.
THE WITNESS: The answer is in theory, one molecule can cause mutation. In practice, that's a speculation that doesn't have any meaning. We have no ability to observe the effects of one molecule of exposure or to examine the risk associated with one molecule of exposure, and I'd say that even in a laboratory setting, we can't do that, much less out in the world of free ranging human beings.
BY MR. HARTLEY:
Q Is there a biological marker for benzene that you're aware of?
A I'm not aware of a biological marker for benzene.

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

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

Q What do you understand the term to mean?

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

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

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

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

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in your search?

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

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

A Dr. Bernard Goldstein?

Q Yes.

A I have read some of his literature, yes.

Q Did you review any of that literature for today?

A No, I don't think I did.

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

A No, I don't.

Q What do you keep referencing there in front

of you?
A I have a partial listing of the literature, but it's not complete because I've added articles to it and have not updated this listing.
Q Are all the materials that you reviewed in

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front of you today?
A Yes.
Q What's your hourly rate, Doctor?
A $350.
Q How many hours do you have in this case?
A I do not know.
Q Have you billed yet?
A I don't think so.
Q When did you first get this case?
A I'm not sure. However, I have a letter of transmittal of documents. The earliest one is dated October 17, 1994. My recollection is I got it before that, but I don't know when.
Q Did you start working on it when you received it?
A I don't remember when I started working on it. I certainly didn't start the day I got the first documents, but I have worked on it over the past few months.
Q How many hours do you think you have in this case? Do you have any idea, estimate?
A Probably between -- excluding today?

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

Q You reviewed all that material in 25 hours in addition to doing the Medline search?
A Yes.
Q Have you taken the Evelyn Wood's speed reading course? Because I know I've read that material and I couldn't have read it in 25 hours.
A I have not taken the Evelyn Wood's speed reading course.

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Q When do you expect to bill in this case?
A I haven't set a date. Probably soon.
Q Did you meet with either Mr. Hollingsworth or Ms. Milnamow yesterday?
A Yes.
Q How long did you meet with them then?
A We met for most of the day.
Q Eight hours?
A Something like that.
Q In the 25 hour estimate, is the eight hours included in that 25 hours?
A No.
Q So we're over 33 hours. Have you met with them prior to yesterday?
A No, I don't believe so.
Q Telephone conferences?
A Yes.
Q Any long telephone conferences, an hour, two-hour conference?
A I don't think so. I don't recall, to be honest with you.
Q How do you keep track of your time?

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A I write it down.
Q Do you have a little ledger that you keep on each case?
A I have pieces of paper.
MR. HARTLEY: We might as well take a lunch break because I don't think I can get done in another hour. Take about a half hour.
THE WITNESS: I'm happy to work through.
MR. HARTLEY: If you want to work through, I'll work through.
THE WITNESS: It's only noon. If you can

do it in another hour, hour and a half, that's fine with me. Let's keep going.

(Recess.)

BY MR. HARTLEY:

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

A That is correct.

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Q Do you have that study?

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

Q To 213.

A Yes, I do.

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

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

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

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

Q Table 5?

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

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

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

A 3.25, yes.
Q Case number 9 would have had less than 30 parts per million cumulative exposure?
A Yes.
Q Case number 10 --
A Wait a minute. I'm sorry. No. That looks like it's actually case 8, but inexplicably, they've dropped down a line.
Q I guess so. So it's case 8, which is less than 30 parts per million years?
A Yes.
Q Case number 9 would be what, less than 12 parts per million years?
A Looks like about a little over 12

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actually -- no, I'm sorry, a little less than 12.
Q Case 11?
A A little less than 10.
Q Did you say a little less than 10?
A No. 37 divided by 3-1/4, a little more than 10.
Q Less than 11, a little more than 10?
A Somewhere between 10 and 11.
Q Case number 11, 110 milligrams per cubic meter?
A 35 maybe.
Q 15, which is 52, which is what --
A 17 and a third -- a little less than 17.
Q Case number 16 at 93 which would be 33, less than 31. Case number 18, 12 parts per million years?
A Uh-huh. What's the question?
Q All these numbers are below 50 parts per million years; correct?
A That's correct. However, there's no data in the paper that indicates that those exposures are associated with increased risk of leukemia. There's

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

Q Page 212, "findings of leukemia risk subsequent to relatively low estimated average accumulative lifetime of benzene exposure supports a recent report by Rinsky." Do you see that?
A I see that.
Q Is that an attempt by the author to correlate the low level exposures to the leukemia and risk?
A No. It appears to be a statement not based on any analyses. It appears to have no foundation. We can critique this paper in more detail, if you'd like to go through it. There are some weaknesses that lead me to think that this is not reliable.
Q Is it a peer reviewed article?
A Yes -- or I should say it's in a journal that does peer review and in fact, for which I've done peer review of articles.

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Q Would an article be published in Environmental Health Perspectives without being reviewed?
A I would think not.
Q What did you find deficient in this particular article?
A The exposure information --
Q Specifically?
A -- is one of the issues that I find to be inadequately described to make sense out of it.
Q In other words, you have problems with the way the authors have indicated the exposure data within the article itself or that it's lacking something?
A It is entirely lacking from the paper what exposure information existed and how it was obtained and what the numbers -- the exposure numbers actually looked like in a meaningful way.
It's also entirely lacking from the paper -- let me rephrase that sentence. That's awkward. The paper contains no results based on the exposure information, and so all of the conclusions

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that are stated in the discussion about risk at exposure levels are simply without foundation in the

paper.
Q Would you say that the paper has no benefit in attempting to associate benzene to low level exposures?
A I regard it as unreliable. I draw no conclusions from this because I don't think it's reliable.
Q You haven't seen Travis's update of that; correct?
A I have not.
Q What is your estimate of Mr. Lavender's benzene exposure?
A Based on the still hygiene data that was done in various areas of the plant, it appears that the exposure levels while he was in the MNB area were routinely in the .01 to -- I'm not sure what upper limit to state somewhere in the tenths of parts per million. His exposure levels for most of his work in the iron oxide area were not detectable. There was not benzene used in that area and so the exposures

would have been negligible.
Q Anywhere else?
A His exposure levels when he was in ECD were routinely down in the .01 parts per million to a few tenths of a part per million.
Q Anywhere else? Did you make an estimation as to the amount of benzene he was exposed to when he worked around the wastewater trench itself?
A My impression from reviewing the area samples throughout the plant is that the exposures in the areas in which benzene was used directly were, as I've stated, in the hundredths or tenths parts per million and that included working in the ECD area.
Based on that, I think it's unlikely that the exposures near the trenches were appreciably different than those levels.
Q And what do you base that on, the fact that all the other levels in the area were low?
A Yeah. The fact that the area level -- many of the measurements were area measurements and they showed low levels almost all the time with occasional excursions into the few parts per million, so it's

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

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

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

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

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

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

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

MR. HOLLINGSWORTH: What finding?

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

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

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

plant.
BY MR. HARTLEY:
Q First of all, you say that upsets or spills

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were uncommon at the Mobay plant?
A That is my impression.
Q And what do you base that on?
A Record of spills prepared by Mobay that tabulated them.
Q Do you have a copy of that with you?
A I do not, but I was shown that yesterday.
Q For what period of time did you have those record of spills?
A For every period of time for which Mr. Lavender was on the site.
Q What was that particular document called?
A I do not recall the title of it.
Q And it discussed all of the -- was it a control log or was it a computerized printout or some typewritten version? What did it look like?
A It was a computerized printout listing every date on which there was a spill of benzene, the amount spilled, the plant involved and the estimated cost of the spill.
Q Okay. I've seen that document. That document does not include all of the dates, does it?

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Does it include every date that there was a spill, or do you know?
A I was under the impression it included every date there was a spill.
Q Let me represent to you that it doesn't. Do you -- have you reviewed the depositions where the various management employees indicated that many times there were upsets that were not recorded?
A I do not recall that specific statement. You have the list of things that I have reviewed.
Q Let me represent to you Ray LeMasters, who was a foreman in that particular area; Kyle Earley, who was a particular foreman; William Thompson, who was a particular foreman in that department, all indicated that there were times when upsets occurred that were not recorded.

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

BY MR. HARTLEY:

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

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

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

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

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

A Yes.

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

(Garabrant Exhibit 1 identified.)

BY MR. HARTLEY:

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

A Yes.

Q And you reviewed this.

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

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

MR. HARTLEY: That will be 2.

(Garabrant Exhibit 2 identified.)

BY MR. HARTLEY:

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

A I have not.
Q Have you talked to anyone about exposure personally?
A I have not.
Q Did you talk to Dr. Irons about this case?
A I have not.
Q Have you talked to any physicians at the University of Michigan -- are you still at Michigan?
A I am.

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Q Did you talk to any physicians or anyone at the University of Michigan about this case?
A No.
Q What level do you feel -- I know you answered this before, but I want to refresh my recollection. What's the odor threshold?
A It's in the range of a few parts per million.
Q And a few to you is what?
A Less than 10.
Q More than five, less than 10?
A I'm not sure that any of us could defend a single number. People's odor thresholds vary. I think in the range of 3 to 10.
Q Is there a fatigue factor which is taken into account for exposures of any length of time?
A It's a general principle that our ability to smell things fatigues rapidly and we cease to smell things as time goes on, usually within minutes.
Q Does that occur on a day-to-day basis? If you are used to smelling something every day and every day and every day, do you become fatigued or

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just basically used to it is a poor choice of words or immune to it is a poor choice of words, but you recognize it and just keep on going and forget about it?
A Well, I think there are two things that happen. It's my understanding that the sensory nerve endings in the nose become -- I don't know if "fatigued" is the right term -- acclimated is probably a better term, to a smell fairly rapidly, but that leaving overnight would allow them to

reestablish their sensitivity to the smell.

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

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

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

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

A Yes.

Q Why would it be significant?

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

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

Q My question to you, though, was if someone

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

A If that were true, it would indicate that at the time the person was out smelling the trench, that there was benzene at or above the odor threshold.
Q Is there a latency period, Doctor, in your opinion for leukemia to develop from benzene exposure?
A Yes.
Q What do you believe that to be?
A I think that the scientific literature supports the conclusion that latency of 10 years or greater is necessary for leukemia to develop after benzene exposure.
Q Is it absolutely necessary for it to take 10 years?

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A Well, I think that's what I just said. The scientific literature does not show evidence of increased risk of leukemia within the first 10 years of exposure.
Q And what particular ones establish that you need 10 years? Would you reference me to those articles?
A Wong in his chemical workers study.
Q Is that Wong 1987-A, 1987-B? That's his latency period?
A Yes.
Q Anyone else?
A Ott also supports that.
Q What about the Bond review of Ott? Does that support it?
A I don't recall that Bond reexamined the issue. I would have to look at it to see. The Bond study would support that particular --
Q What portion of the Bond study are you referencing?
A Table 5.
Q Table 5?

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A Yes.
Q Three out of every five people have more than 10 exposure; two out of the five of them do not?
A I thought we were discussing latency.

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

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

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

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

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

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

which we have to go back and look at.

Q Does toluene have any effect on benzene exposure concomitantly?

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

Q Resulting in?

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

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

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

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

A Decreases.

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

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

the metabolism down a different pathway. If the

leukemogenic effect of benzene occurs through formation of metabolites in the pathway that goes via hydroquinone, then shifting it away from that pathway would be expected to reduce the risk associated with benzene exposure.

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

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

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

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

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

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

A Yes, I did.

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

A No.

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

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

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

A Yes.

Q And why is that?

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

Q When was it done?

A It was completed in 1988.

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

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

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

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

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

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

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

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

A Yes.

Q Did you do anything with that data, other

than look at it and say here's what happened?
A No, I did not. If I can add, there wasn't anything that could be done with the data in the interrogatories.

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Q When you look at carcinogenic risk, do you assume it operates multiplicatively or additively, in your own opinion?
A I don't actually assume either. That is usually one of the reasons we do epidemiology, to determine whether it acts to add to risk or to multiply the underlying risk.
Q Did you determine in your own opinion what benzene does?
A The epidemiologic literature, I don't think, is adequate to comment on whether it's a multiplicative or additive risk. There are certain methods in analyzing the data that assume multiplicative risk, so some of the discussions we discuss make that assumption, and it's probably a reasonable assumption.
Q Specifically which ones?
A Paxton, Rinsky and actually, I guess -- no, I guess that's it.
Q Why didn't you rely on the Rinsky and Infante studies for your cumulative dose requirements?

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A Because I think they have systematically underestimated the exposures.
Q Do you think there is a nonlinear or linear dose response relationship between benzene and leukemia?
A The best data at this point suggests that there is increased risk above 50 parts per million years and there is no evidence of increased risk below 50 parts per million years. Those two observations are consistent with a nonlinear dose response relationship.
Q Have you ever treated someone with leukemia?
A Yes.
Q Have you ever treated anyone with leukemia

who had been exposed to benzene?
A Not to my recollection, but I'm not sure I would know at this point.
Q If we assume -- did you read Dr. Rose's deposition?
A Vern Rose?
Q Yes.

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A Yes.
Q Do you have any quarrels with any of his statements, or did you disagree with any of his opinions, other than the odor threshold, which I'm aware of?
A I'd have to go back through it.
Q Did you make any notes on that?
A No, I did not.
MR. HOLLINGSWORTH: I object to that question on the ground that it's overbroad. And without reviewing the deposition in detail now or beforehand, it's not a fair question. Maybe if you could state what his opinions are and ask the doctor whether he agrees or disagrees with them, that would be a more efficient way to proceed on this issue.
BY MR. HARTLEY:
Q Did you make any highlighted pages in Dr. Rose's deposition? Did you highlight any pages?
A I don't recall whether I did or not. There's a fairly easy way to find out.
(Witness reviewed the document.)
I did.

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Q What did you highlight?
A On page 81, I highlighted "to me the description of these two odors are quite different and therefore, I would believe that they would appear to most people to be different odors." I actually disagree with that.
Q Have you smelled mononitrated benzene?
A Yes, I have.
Q Where did you smell it?
A I used to smell it cleaning guns.
Q And have you smelled benzene?
A Yes, I have.

Q And it's your opinion they smell alike?
A It's my opinion they smell enough alike that most people would have difficulty naming which one they were smelling.
Q Would that be true for someone who has worked in the area 20 years?
A I think that someone who has trained himself to recognize smells could reliably distinguish between them, but someone who is not trained to do that probably could not.

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Q Do you have any way to refute Mr. LeMasters's statement that he could tell the difference between benzene and MNB?
A I have no way to refute such a statement.
Q You haven't met with Mr. LeMasters, have you?
A No.
Q Have you put benzene and mononitrated benzene under his nose?
A No, I have not.
Q Other than your own conclusion that most people wouldn't, there's really no way to refute that Mr. LeMasters is able to differentiate between the two?
A I have no way to refute that, no.
Q If we assume that Dr. Rose's assessment of 61 parts per million any time you could smell it occurred on a daily basis for Mr. Lavender, as he described in his deposition, would that be sufficient in and of itself to cause his -- or to be associated with his AML?
MR. HOLLINGSWORTH: Objection. Lack of

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foundation. Misstates the evidence.
BY MR. HARTLEY:
Q That means you can answer, Doctor.
A First off, I think there are good reasons to think that the exposure levels were not that high and that Mr. Lavender couldn't tell what he was smelling, so the characterization that the exposure levels were 61 parts per million is contradicted by the evidence that I read in this case.

Q Well, let's assume that a jury in Marshall County accepts the fact that Mr. Lavender smelled benzene every day, and let's further assume that this jury accepts the fact that Dr. Rose will testify that when you smell it at 61 parts per million.

Given those two set of circumstances, do you have an opinion with reasonable medical probability as to whether Mr. Lavender's acute monocytic leukemia would be causally associated with that exposure?

MR. HOLLINGSWORTH: Same objection.

BY MR. HARTLEY:

Q Sure. I'm just asking you to assume that.

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I don't want you to change it for me. I want you to assume that fact.

A If I were to accept those assumptions, which I do not --

Q I understand that.

A -- it would still be my opinion that Mr. Lavender's exposure to benzene at the Mobay plant did not cause his leukemia.

Q Have you attempted to calculate his parts per million years?

A I've made some mental calculations of that.

Q And in your opinion, what would that be?

A In my opinion, his first exposure to benzene was in the 1990 to '92 employment period, and that out of the 6000 hours he worked in that period, the records indicate he was only in MNB for 48 hours and in ECD for 208. And so even if the exposures were at 60 parts per million while he was exposed, with which I disagree, his parts per million years would still be far below that that is known to cause leukemia.

Q Did you see in the depositions that the

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operators at ECD were complaining about solvent smells?

A I do remember that there were complaints of solvent smells from the trenches and from the wastewater, and I believe in ECD as well.

Q Did you notice that Mr. Myers, who was the

manager for ECD, testified about the content of benzene or the amount of benzene in the wastewater? Do you remember that?

A I do not recall that specific issue.

Q Do you remember him testifying that they had received on an average 50 pounds per day?

A I don't recall.

Q Have you seen any documents that would support that?

A Did I see any documents that would support that 50 pounds per day of benzene went into ECD?

Q Uh-huh.

A I don't recall those documents. I would be happy to look.

Q If we assume that to be true, I just want you to assume it for right now, that ECD received on

average 50 pounds per day of benzene every day from 1980 through 1993. Is that a significant amount of benzene coming to waste?

MR. HOLLINGSWORTH: Are you basing this question on the trench monitoring data? Is that what you're referring to?

MR. HARTLEY: I'm asking him to assume that there's 50 pounds of benzene making its way every day on average to ECD.

THE WITNESS: I don't know how to answer your question, is that significant. Whether that has any relevance to human exposure isn't clear. It depends on whether that wastewater stream is enclosed or whether it's open. It depends on the temperature of the water. It depends on how the water is handled and where the people are.

If 50 pounds a day reached ECD and if all 50 pounds a day evaporated into the air, there would be 50 pounds of benzene vapor in the air every day. It would also matter what the wind velocity was and wind direction and general ventilation. And so the relevance of that number to human exposure is not

clear.

BY MR. HARTLEY:

Q Do you understand the ECD process at Miles?

A I am not familiar with the details of the ECD process.
Q Do you have an understanding as to how the contaminants that are in the water are removed prior to induction into the organism pool?
A I do not know that specifically at the Miles plant.
Q Will you know that by the time we go to trial?
A I suspect I will.
Q That's another thing you ought to be looking at, then.
Can you calculate for me, Doctor, if 50 pounds of benzene is making it to the ECD per day, how much is being spilled in the MNB unit? Is that calculation possible?
A I cannot make that calculation.
Q You don't have the training to do it, or you don't have sufficient information to do it?

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A I don't have sufficient information to do it, and I'm not sure whether I have the training to do it.
Q Would you agree with me that if a certain amount of benzene was spilled into the trench at MNB, that a portion of that would evaporate by the time it made its way almost a half a mile down the road to ECD under normal conditions?
A It depends on the construction of the trench and whether it's a closed stainless steel pipe and what the fire traps and grates look like and how much of it is caught and removed in the course of going. It depends on wind velocity and temperature. It depends on lots of things.
Some of it would certainly evaporate, but the amount I cannot predict. It would take someone with different credentials than mine.
Q What do you understand this trench to be? Can you describe it for me?
A My recollection is there was a trench system that ran throughout the plant that basically had a branch that originated near or in each or most

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of the buildings and that it flowed south to the ECD area, which was at the south end of the plant.

The actually description of it is not entirely clear to me. I had the impression in some of the materials that I read that it was an open trench. I had the impression that some areas were covered by a grate, and I recall reading some materials that talked about a stainless steel pipe, and I do not know how all of that fits together, to be quite honest with you.

Q Have you tried to determine how it fits together?

A I have not.

Q Do you think that it would matter, the type of trench system that was being operated through the Miles facility when you attempt to determine benzene exposure, assuming benzene to be in the trench?

A I think that the physical construction of the trench system and the rate of water flow and temperature and ventilation and many such factors certainly will have an effect on the benzene concentration.

I do not represent myself to be an industrial hygienist or an environmental engineer, and I will rely on the expert opinion of other witnesses in this case for determination of what the exposure levels were along the trench.

Q Does that mean that you'll rely on Dr. Rose's evidence or testimony or only on Mr. Spencer's?

A I will certainly read Dr. Spencer's and have read Dr. Rose's and will rereview his to make my own determination of what exposures I think are relevant to Mr. Lavender.

Q Do you think there is enough air monitoring at the Miles facility to calculate cumulative exposure?

A The answer is we always want more exposure samples than we have. I've been involved in studies where we've had thousands of measurements and still wanted and needed more.

In the real world, it is clear that there

have been hundreds of benzene measurements made at Miles, and it's my impression that they are adequate

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to come up with an estimate of Mr. Lavender's exposure or a range of estimates that we can be confident of. The issue of having more samples would allow us to have greater precision in the estimate, but nonetheless, there are many samples that I believe are relevant to Mr. Lavender.

Q Did you determine what his cumulative parts per million years were?

A I think we've already discussed that --

Q We may have. I just forgot.

A My answer was I made mental calculations. I didn't give you a number.

Q I didn't think you did.

A My recollection is we started discussing it and moved on. His exposures were in the range of .01 to a few tenths of parts per million with rare excursions into the parts per million range, and his duration of exposure based on his work records appears to have been in the range of a few hundred hours.

The product of .01 parts per million times .1 working years, which would be the low end of my

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estimate, would be .001 parts per million years. The upper end of my estimate would be .3 parts per million times perhaps .2 years, which would be .06 parts per million years.

Q What did you base those calculations on?

A I based those calculations on the still hygiene measurements we've discussed, which showed many samples in the range of .01 to, as I said, a few tenths of a part per million, which I used as .3 in these calculations, so parts per million between .01 and .3 and years of exposure between .1 and .2.

So the product of .01 times .1 is .001. That's the low end of my estimate. The product of .3 PPM times .2 years is .06 parts per million years. That's the high end of the estimate.

Q Did you take into account any peak exposures?

A Yes, I did.
Q What peak exposures did you take into account?
A As we've already discussed, it's my understanding from Mr. Lavender's work records and

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descriptions in the depositions and the industrial hygiene monitoring that he was rarely exposed to spills or unusual occurrences. So even if he had a few peaks in the parts per million range, it would not appreciably alter his total parts per million years.

Maybe I can add to that. For example, if he worked at 10 parts per million for a full day, a full day is 1/2000 of a working year, so you can add to my estimate .001 times 10, which would be .01. It would only trivially change my upper limit estimate.

Q Did you take into account the fact that Miles routinely did not record samples in the MNB area?

MR. HOLLINGSWORTH: Objection. I don't think that accurately states what the record is.

BY MR. HARTLEY:

Q Let's put it this way then. Did you take into account Kyle Earley's testimony that many times they did not record the monitoring of benzene in the area of MNB?
A I am aware that there were instances in

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which a measurement was made and the results were not written down, if that's what you're referring to.
Q Yes. Did you take that into account in your calculation of parts per million years?
A The answer is -- the best estimate of a worker's exposure is based on full shift samples that are made for surveillance purposes, not based on brief grab samples or short duration samples that are made for the purposes of characterizing excursions or determining compliance with the law.

And therefore, the large number of samples that represent full shift time weighted averages I regard as far more reliable estimates of his real exposure than do measurements made during excursions

and accidents where there were very brief peaks that did not represent the usual working conditions.

Q In your opinion, how long would 2000 pounds of benzene need to be exposed to the air to be completely back below the OSHA level?

MR. HOLLINGSWORTH: Excuse me, 2000 pounds of benzene from a 55-gallon drum or from some container or from the trench?

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BY MR. HARTLEY:

Q Let's assume that we have 2000 pounds of benzene escaping from the water column. How long would it take that 2000 pounds -- first of all, what is your estimate of the parts per million that would be in the area for the 2000 pounds?

A There's no meaningful way to answer that question.

Q Do you think it would be pretty high?

A There's no meaningful way to answer the question as it's asked.

Q Why not? What are you lacking that would help you answer that question?

A I'm capable of calculating the concentration of benzene in air provided I know the volume of air we're talking about, and you've given me no sense for the volume of air we're talking about.

The area, as you've stated it, I don't know whether you're talking about the area around the entire Mobay facility, the area in Atrium, West Virginia, the area around the MNB plant, the area

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around ECD, the air inside a building.

If you release 2000 pounds of benzene in this room, the exposure would be very, very high. In fact, you probably couldn't vaporize it. So I can't answer the question as it's asked.

Q The one area you forgot was the entire state of West Virginia when we're dropping 2000 pounds. If we dropped 2000 pounds into the trench, open air trench, what would the levels be approximately when you're standing 5 feet from the trench?

A I have no way to estimate that based on the information that you've postulated.
Q Have you looked at those releases, the releases that Mr. Hollingsworth or whomever showed you yesterday, the spills?
A The amount spilled?
Q Yes.
A Yes, I have.
Q Have you attempted to calculate any level of benzene that would have been released during those spills?

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A The answer is I have not, nor do I think that I could do that reliably without knowing temperature, wind velocity, wind direction. And those calculations could be better made by someone with different credentials than mine.
Q What is your little paper there that you keep referring to?
A This is my notes on the chronology of events in Mr. Lavender's life.
Q Can we mark that as an exhibit? Is that your only copy? I don't want to take your only copy. Yes, it is your only copy or yes, we can mark it?
A It is my only copy, but it's on disk. I can generate another one.
MR. HOLLINGSWORTH: We'll copy it for you.
MR. HARTLEY: Do you want to mark that as 3.
(Garabrant Exhibit 3 identified.)
BY MR. HARTLEY:
Q Anything else you brought with you, Doctor?
A I brought everything else I was instructed

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to bring in that folder.
Q This is your notes on your studies --
A Yes.
Q -- that you reviewed in addition to whatever else was in there?
A Those are my notes on the studies I reviewed, except for some that I have recently added, which are not listed on that page.

Q But which are in your three-ring binder?
A Yes.
Q This is an extra copy of this?
A That's the only one I can put my hands on at the moment.
MR. HARTLEY: Can we make a copy of this?
MR. HOLLINGSWORTH: Yes.
BY MR. HARTLEY:
Q Doctor, I don't want a copy of all your articles there. If you would go through the articles and tell me what the titles are for the record and what the cites are so I know what you're utilizing at this juncture in the case.
A Could I suggest that I would leave these

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with Mr. Hollingsworth and that they could do that or copy the face pages.
Q That will be fine.
MR. HOLLINGSWORTH: There are dozens and dozens of papers that you're referring to there. It's probably a faster way to do it.
MR. HARTLEY: Whichever way you think is quicker, Doctor.
THE WITNESS: Either way is fine with me.
BY MR. HARTLEY:
Q How many documents are in these total so -- I'm not suggesting that he would intentionally not give me all the copies, but just so I know that there wasn't a mistake made or something like that. How many articles are in both these volumes?
A I do not know. I would estimate -- here. This will list everything except the ones that have yellow tags and the ones that are these. So this plus approximately -- because there are probably a couple that aren't tagged -- I'd say that with roughly a dozen more would be about right.
Now, I know a couple of these are tagged

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that are on that list. So it's that plus about a dozen.
Q Do you have an opinion as to what is the cause of Mr. Lavender's leukemia?
A My opinion is that the vast majority of

leukemia arises for reasons that are yet to be discovered and Mr. Lavender is one of those unfortunate cases.

Q So what you're basically saying is you don't know?

A What I'm saying is that I do not think that we -- there's no evidence that his leukemia arose as a result of any of the established risk factors for that disease with the exception of smoking, which I think increased his risk in a small way.

Q Outside of the increased risk, you don't know what really caused his cancer? I know you've said it three different ways. I just want to know yes or no, if we subtract the increased risk of leukemia from smoking, you do not know what caused his leukemia, yes or no?

A It's not a question that can be answered in

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that manner. The question presupposes there is a single cause and that is not the way most, if any, human disease arises. Almost every disease we've studied has multiple causes and the contributions of a number of causes are important in the causation of each case.

In Mr. Lavender's case, we've identified smoking as a factor that led to his leukemia, although admittedly it's not a strong factor. The other causes of his leukemia are not known to me or to medical science.

Q He didn't have chemotherapy that caused it, did he?

A Not to my knowledge.

Q He didn't have radiation therapy that caused it?

A He did not have radiation therapy.

Q That caused his leukemia?

A That's correct.

Q He was not exposed to the atomic fallout in Hiroshima?

A Not to my knowledge.

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Q Or Nagasaki?

A Not to my knowledge.

Q He was not at the testing ground at Los Alamos, was he?
A Not to my knowledge.
Q According to his own deposition, he was not exposed to electromagnetic fields because when he was working on the towers, they were deenergized. They had not even been energized at all, so he would not have been exposed to electromagnetic fields, would he?
A I suspect that's an area where his perceptions are simply wrong. Electricians are commonly exposed to substantial electromagnetic fields in the course of their work.
Q Do you have an opinion that his exposure to electromagnetic fields caused his leukemia?
A No, I do not.
Q Do you feel there is sufficient evidence in the epidemiological literature to support that relationship?
A No.

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Q His parents weren't known to have a genetically induced leukemia, were they?
A No.
Q So we've eliminated just about everything except for his smoking, correct, because we know you don't think he had sufficient exposure to benzene. So the only thing we can point to is the .31 to .5 elevation in smokers of developing leukemia, correct, that you supported with the Segal study, and that's the only thing we can put our finger on?
A Again, that has to be said in the right context. We do not know the causes of leukemia in the vast majority of instances and so for Mr. Lavender, although we know that smoking is a cause, he basically falls in the vast majority of cases of leukemia where we are unable to identify what caused it.
Q Did you review anything else, Doctor, that we haven't discussed today?
A I do not believe so.
Q Would you read for me what your notes are that you're going to do before trial.

MR. HOLLINGSWORTH: What he has written down that he may do before trial is not necessarily what we, on behalf of Miles or Miles's attorneys, would ask him to do in connection with preparing his testimony for trial.

These are just a few things that you have asked him about, and you have asked him whether he would perform certain tasks before trial or not, and he said that he would. I just want to make clear what he has been asked to do he has done and he has given you an opinion today in accordance with what I believe our 26(b)(4) statement was as to what his opinion would be.

He's provided you with, I think, at least in my opinion, what all the bases for his opinion are so these other things that you have asked him, whether or not he would do or look at before trial are things that he has taken down on his own initiative, at your suggestion, really. It's been by your suggestion.

It's certainly not clear to me that any one of those things, certainly not all of them, are

things that we would ask him to do in advance of his testimony at trial and certainly not any one of those things or all of them are necessary we believe or we believe the court would find to support his opinion at trial.

But having said that, you can ask him what those things are.

MR. HARTLEY: Thank you.

MR. HOLLINGSWORTH: Or we could make a copy of the list.

MR. HARTLEY: I'd rather him read it into the record.

BY MR. HARTLEY:

Q Would you make a list and tell me what you have written down there on your piece of paper?

A I've written down I need to get the Crump article that we've already discussed. I'm interested in reviewing whether benzene causes either leukopenia or lymphocytopenia in the absence of pancytopenia.

I'm interested in reviewing the issue of whether benzene is mutagenic. I'm interested in reviewing the mechanisms by which benzene causes leukemia.

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I've written down something about the Wong 1983 comments on NIOSH that were not in the open literature, but I don't intend to do anything about that since I have no access to those. I've written down to get a copy of the Travis update of the Yin study.

I've written down to calculate the benzene exposure from cigarettes for Mr. Lavender. I've written down the same, Gene McDonald at NIEHS with the intention of trying to find out what that person has published relevant to this issue.

I've written down the name Maltoni, although I don't think I'm going to do anything about it. I know a good deal of Dr. Maltoni's literature and have it. I don't think it's terribly important to this issue.

I've written down "review ECD process," and I've written down in my estimate of parts per million years between .001 and .06.

Q Have you ever acted as an occupational physician for a corporation, in-house?

A No.

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Q You've written on occupational medicine and workers' right to know, haven't you, at least a chapter in some book?

A Yes, I've written on communication in the Handbook of Occupational Medicine by Robert McCunney. I don't think the title is quite right.

Q '93 or '94 book?

A '94.

Q Did you read in the depositions where the only access Mr. Lavender had to hazards associated with the Miles job site was the fact that he was told about a phosgene MSDS and there were other MSDSs available if they wanted to review that? Have you reviewed that?

MR. HOLLINGSWORTH: Objection; mischaracterizes the record.

BY MR. HARTLEY:
Q That means you can answer.
A My recollection of that issue is the testimony indicated that Miles had supplied a complete set of MSDSs to Mr. Lavender's employer and Mr. Lavender's employer was responsible for health

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and safety training.
Q If Miles knew that the employer was not following through with health and safety training, did they have an obligation in your opinion to inform Mr. Lavender, either through signs in the areas concerning respirators being needed or through safety meetings or handouts, about the hazards associated with the various chemical processes?
MR. HOLLINGSWORTH: Objection. Calls for a legal conclusion.
BY MR. HARTLEY:
Q That means you can answer.
A I'm not a lawyer. I do not know the OSHA regulations in adequate detail to answer that.
Q From a purely industrial setting, as a medical physician, occupational physician, would it be good occupational medicine practice to inform all employees, whether they be your employees or someone else's employees, on the job site of the hazards associated with that particular facility?
MR. HOLLINGSWORTH: I object to the form. Same objection on the requirement for a legal

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conclusion.
BY MR. HARTLEY:
Q Do you understand the question, from an occupational medicine standpoint?
A I thought I did until you said that. I think I understand the question. While I would certainly agree that everyone at work should understand all of the hazards at work fully, it is extremely difficult to achieve that in the real world and that there are regulations written to ensure that that is -- that a system is built to do that.
It's my understanding that Miles did its part by providing the MSDSs and that it was

Mr. Lavender's employer's responsibility to see that he was appropriately trained in terms of the hazards that were present at Miles and in terms of the hazards that were uniquely present in the course of being an electrician.

Q What is the basis for your opinion that his employer was solely responsible for that training?

A My recollections of the OSHA regulations.

Q Are there any other opinions that we have

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not talked about today that you might testify to at trial?

A I cannot think of any.

MR. HOLLINGSWORTH: I object to that question. How can he answer that? He doesn't know what he's going to testify to at trial. The trial isn't here yet.

You can ask him about his opinion and the bases of his opinions and anything we've offered in the Rule 26(b)(4) statement, but when it comes to the trial, for him to testify at trial, a lot will come before him, I'm sure.

MR. HARTLEY: That's a typical question. I've been through 10,000 depositions.

BY MR. HARTLEY:

Q Is there any other opinion, Doctor, that you haven't --

MR. HOLLINGSWORTH: But you haven't heard me ask that.

MR. HARTLEY: You're right. I haven't. I don't think I have anything else, Doctor, that I can think of anyway.

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Do you want to read and sign the deposition -- we better mark this as number 4.

(Discussion off the record.)

MR. HOLLINGSWORTH: It appears, Mr. Hartley, that the number of papers that he's relied on which are contained in these two black binders that have been present here in this deposition room is about 100.

MR. HARTLEY: I don't want the entire articles now.

MR. HOLLINGSWORTH: I understand that. You want the names of the articles. And what we'll endeavor to do is put those on a list for you, which I realize it's not our duty to do. It's yours. We'll do that anyway to save time, and I assume you'll just return the favor to me sometime when you get a chance.

MR. HARTLEY: I always try to.

MR. HOLLINGSWORTH: Okay.

MR. HARTLEY: If we go to trial, will you bring these two notebooks with you?

THE WITNESS: Probably.

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MR. HARTLEY: Okay. That's fine.

(Whereupon, at 2:00 p.m., the deposition was concluded.)

DAVID H. GARABRANT

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C O N T E N T S

E X H I B I T S

GARABRANT DEPOSITION NUMBER	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