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IN THE DISTRICT COURT FOR JEFFERSON COUNTY, TEXAS
172nd JUDICIAL DISTRICT

Cause No. E-170.351

YOLANDA McCARTY ROBERTS, et al.,

Plaintiffs,

vs.

SHELL OIL COMPANY, et al.,

Defendant.

VIDEOTAPE DEPOSITION OF RICHARD D. IRONS, Ph.D.

August 29, 2006

Pursuant to Notice taken on behalf of the Plaintiffs at 2115 13th Street, Boulder,

Colorado 80301, at 10:48 a.m., before Joanne Blair, Registered Professional Reporter and Notary Public within Colorado.

APPEARANCES:

DARREN L. BROWN and RODNEY BARNWELL, Attorneys at Law, from the Provost Umphrey Law Firm LLC, 490 Park Street, Beaumont, Texas 77704, appearing on behalf of the Plaintiff.

ROBERT SCOTT, Attorney at Law, from the Law Firm of Abrams Scott & Bickley, L.L.P., 700 Louisiana, Suite 4000, Houston, Texas 77002-2727, appearing on behalf of the Defendant Univar U.S.A, Inc.

Also Present: Tom Koetting, videographer

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I N D E X

WHEREUPON, the following proceedings were taken pursuant to the Texas Rules of Civil Procedure:

THE VIDEOGRAPHER: We are now on the record. Today's date is August 29, 2006, and the time is approximately 10:48 a.m. The location is the Hotel Boulderado, Boulder, Colorado. This is the video-captured deposition of Dr. Richard Irons in the matter of Roberts, et al., versus Shell Oil, et al., Case No. E-170.351 filed in District Court, Jefferson County, Texas. The court reporter is Joanne Blair of Esquire Deposition Services. My name is Tom Koetting, legal videographer, and this deposition was requested by Darren Brown. Counsel, would you please identify yourselves for the record.

MR. BROWN: I'm Darren Brown for the plaintiff.

MR. BARNWELL: Rodney Barnwell for the plaintiff.

MR. SCOTT: Robert Scott for the Defendant Univar USA, Inc.

THE VIDEOGRAPHER: Joanne, would you please swear him in.

RICHARD D. IRONS, Ph.D.,
having been duly sworn to state the whole truth,
testified as follows:
MR. BROWN: Bob, before we get going, as far as agreements, pursuant to Texas Rules of Civil Procedure?
MR. SCOTT: Yes.
MR. BROWN: Signature, what do you want to do?
MR. SCOTT: I assume you want to sign -- read and sign it?
THE DEPONENT: Yes.
MR. BROWN: Can I have an agreement if it's not back within the required time by the rules, we can use an unsigned copy?
MR. SCOTT: Yes. I think the rules say 30 days, but whatever they say, that's fine.
MR. BROWN: Also, it's my understanding --

MR. SCOTT: Darren, let me just say, there may be -- I don't know what Dr. Irons' current travel schedule is, but he is out of the country a fair bit. So there may be some issue there. It may be delayed and mailed to China, but that would be the only thing I can imagine.

This shouldn't take any great time to get it there.

MR. BROWN: My only concern, we're No. 6 on the docket, I just found out today. So if we ended up having to go, we would like to be able to use what we get here today.

MR. SCOTT: Sure.

MR. BROWN: In addition, it's my understanding that you thought you had about 20 minutes of direct?

MR. SCOTT: Correct.

MR. BROWN: Okay.

EXAMINATION

BY MR. BROWN:

Q. Good morning, Dr. Irons.

A. Good morning.

Q. Would you please state your full name.

A. Richard D. Irons.

Q. What's your home address, sir.

A. 330 16th Street, Boulder, Colorado, in the United States.

Q. Where is a daytime phone where you can be reached?

A. (303) 315-7170.

Q. Where does that ring?

A. University of Colorado Health Sciences Center.

Q. Is that your employer?

A. Yes.

Q. You have been retained by Mr. Bob Scott as an attorney for the defendant Univar, formerly known as Van Waters & Rogers; is that correct?

A. That's correct.

Q. When were you first retained by him to act as an expert in this case?

A. I believe it was February or March of this year.

Q. In what areas -- strike that. What was your assignment in the case that you got from Mr. Scott?

A. To evaluate the records and testimony in this case and to ascertain the

likelihood as to whether or not Mr. McCarty's disease could have been caused or contributed to by exposure to benzene.

Q. Were you asked to do anything else?

A. I don't believe so.

Q. And in what areas of science or

medicine do you intend to act as an expert in this case?

A. Toxicology and the associated fields in toxicology, epidemiology, industrial hygiene, and pathology.

Q. You are not an epidemiologist, are you, sir?

A. No, sir, I'm not.

Q. You are not an industrial hygienist, are you, sir?

A. No.

Q. And you are not a medical doctor, are you?

A. No, I'm not.

Q. My name is Darren Brown. We met briefly before this deposition, correct?

A. I don't recall.

Q. You don't recall meeting me just now?

A. Oh, just now. I thought you meant some other time.

Q. I'm sorry. Let's go back.

MR. SCOTT: He thought you meant on a prior occasion. Are you taking any medications today that we should be aware of?

MR. BROWN: Feeling better now.

Q. (BY MR. BROWN) You and I met for the first time briefly before this deposition this morning?

A. Yes, sir.

Q. We've never met before, to my knowledge. Have we met before?

A. Not to my knowledge.

Q. I represent the family of Oliver McCarty, and you realize that he is the man who passed away from acute myelogenous leukemia and has brought a lawsuit against Van Waters & Rogers and Univar, who are represented by Mr. Scott and by whom you are testifying on behalf as an expert?

A. Yes.

Q. You've given many depositions in the past, correct?

A. Several, yes.

Q. During the last -- excuse me -- during the last year have you testified in any case involving benzene or diseases related to alleged benzene exposure?

A. No.

Q. How about since you last testified

for Mr. Dillard in the Cowey case back in November of 2003, have you given a deposition in a benzene-related case since then?

A. I believe so.

Q. Do you know the name of that?

A. As I sit here, I can't recall. It would have been probably last year.

Q. Who was the attorney who hired you?

A. I believe the last time I gave a deposition was for Mr. Lou Woolf, but I'm not certain. It's been a while.

Q. Was that deposition in Tennessee or here in Colorado?

A. It wouldn't have been in Tennessee. I believe it was here.

Q. All right. Do you keep copies of your prior depositions?

A. No.

Q. And you've been acting as a

consultant for defendant oil companies and chemical companies in litigation for about 16 years, as I understand; is that correct?

A. I've been involved in expert consultation and testimony for -- 16 years? Yes, that's probably right.

Q. During the entire course of that 16 years, sir, have you ever testified that a plaintiff's alleged disease was caused by exposure to benzene?

A. No.

Q. Can you give us a ballpark estimate of the number of times you've testified in cases where benzene exposure was alleged to cause a disease in which you had testified that it did not cause that disease?

A. Maybe 40. I'm not certain of that number.

Q. That's as close as you can get it as you sit here; is that right?

A. Yeah, either less or more.

Q. Having given numerous depositions before, you realize what a deposition is, correct?

A. Yes.

Q. You realize that we are videotaping today's deposition, and your deposition can be played before the judge and jury in the trial of this case just as though you were sitting in front of the judge and jury testifying yourself, correct?

A. That's correct.

Q. You realize that you are under oath to tell the truth today?

A. Yes.

Q. During the deposition if I ask you a question that you don't understand, would you stop me and tell me that you don't understand what I've asked you?

A. I will.

Q. If you answer my question, may I and the jury assume that you've understood what I've asked you?

A. Yes.

Q. If you need to take a break for any reason, just let us know we'll be happy to accommodate you, right? Is that agreeable?

A. Yes.

Q. What documents did you review to refresh your recollection in preparation for

your testimony here today, sir?

A. The medical records and testimony that I was provided by Mr. Scott in this case, some publications that I have brought with me, and other documents that I've also brought with me.

Q. The medical records that you have reviewed, what are those?

A. They are the medical records of Mr. McCarty.

Q. And you have received those from Mr. Scott; is that correct?

A. Yes.

Q. All right. The testimony that Mr. Scott provided you with, whose testimony have you seen and been provided with by Mr. Scott?

A. Frank Parker; Joseph Julius Beverly, also his affidavit; as well as the documents that I reviewed that include transcripts that are in Dr. Parker's exhibits.

Q. All right. Other than the testimony of Frank Parker and the testimony in deposition and by affidavit of Mr. Beverly, and exhibits to Mr. Parker's deposition, have you reviewed any

other testimony or affidavits?

A. Just those, as I mentioned, those that are in Frank Parker's exhibits. There's several depositions and transcripts in that.

Q. Can you tell me the ones that, as you sit here today, that you can recall that

were significant by name of the person who was doing the testifying?

A. No.

Q. With regard to any of the depositions that you have looked at, do you feel like any of those were significant to your opinions in this case?

A. With respect to relying on them for my opinions, no, sir.

Q. With respect to relying upon them for any matters, which depositions or testimony do you feel were significant in your consultation as an expert in this case?

A. As I mentioned, I reviewed Dr. Parker's deposition and Mr. Beverly's.

Q. Okay. Now, you also said that you have reviewed some publications; is that correct?

A. Yes.

Q. Do you have those publications with you here today?

A. I brought just a few of them as examples. I didn't bring everything that I've reviewed in terms of the medical epidemiologic literature pertaining to benzene or acute

myelogenous leukemia.

Q. Have you brought with you the articles that you felt were significant to the formulation of your opinions in this case?

A. Yes.

Q. And then you said you brought -- you had reviewed some other documents in preparation for your testimony. What category or what documents were those?

A. The medical records of Mr. McCarty, which I have provided both -- I have brought everything that I reviewed. I've also selected some of those that I think are pertinent to my opinions in this case, specifically.

Q. Okay.

A. I brought a few documents that I think are illustrative of the state-of-the-art with respect to photo thresholds related to benzene. I also brought my notes, which largely

pertain to a telephone conversation I had with Mark Plisker concerning an exposure assessment relating to Mr. McCarty's alleged exposure.

Q. Do you have copies of what you have there in front of you in your notebook? Is that the only copy that you have?

A. That's the only copy.

Q. What do you call that notebook that's there in front of you? Is that your file for the case?

A. I guess you could call it that.

Q. Well, what would you call it so when I refer to it we are referring to the same thing?

A. My notebook.

Q. Notebook on the case?

A. Yes.

Q. All right. I notice in some of the -- behind some of the dividers in your notebook on the case, you selected certain medical records, correct?

A. Yes.

Q. We've got a box of medical records and a box or actually three boxes of documents -- five boxes of documents, it looks

like here. Have you selected the documents that you felt were pertinent to your opinions in this case and put them in your notebook on the case?

A. Yes.

Q. All right. And then you also have a blue folder here in front of you. What is that?

A. These are items that I've produced for this deposition which includes my -- a current copy of my curriculum vitae, publications relating to the study that I direct in Shanghai, and some descriptions of my laboratory in Shanghai that I had.

Q. Okay. Why don't we do this. Would you mind if we mark your case notebook and the documents that you have in the blue folder, along with your blue folder, as exhibits to your deposition today?

A. Not at all.

Q. Let's have the court reporter mark his file notebook as Exhibit 1.

MR. SCOTT: Do you want to go off the video and get this organized? I don't know how many separate exhibits you plan to mark.

MR. BROWN: I don't either.

MR. SCOTT: However you want to

handle it.

THE VIDEOGRAPHER: We could go off if you like.

MR. BROWN: Let's go off for a second.

THE VIDEOGRAPHER: The time is

approximately 11:04 a.m. and we are now off the record.

(Irons Deposition Exhibits 1, 2, and 3 were marked.)

(Discussion off the record.)

THE VIDEOGRAPHER: The time is approximately 11:07 a.m., and we are back on the record.

Q. (BY MR. BROWN) Dr. Irons, we've marked your black notebook as Exhibit 1. We put the exhibit sticker on the first page. That black notebook contains medical records from Texas Oncology, M.D. Anderson. It looks like BM post chemo; is that correct?

A. Yes.

Q. What is that?

A. That's bone marrow -- that's pathology records relating to evaluation of Mr. McCarty's bone marrow post chemotherapy.

Q. This is death summary, and then this says express. What is that?

A. Exposure.

Q. Exposure. And then you have dividers numbered 1 through 14 with pages behind each divider; is that correct?

A. Yes.

Q. And you've got the deposition notice with the subpoena duces tecum over in the pocket part, and what is this back here in the back pocket part?

A. I think those are certification of the records. They are not of much importance to me. Yeah, these are records -- documentation of the medical records.

Q. Would you find for me in Exhibit 1 the notes that you referred to from Mark Plisker?

A. Yes.

MR. SCOTT: I think it may be Plisko. P-l-i-s-k-o, I believe it is.

A. Yes.

Q. (BY MR. BROWN) And that's behind the exposure tab; is that correct?

A. Yes.

Q. And that's the only document that you have behind the exposure tab in your case notebook, correct?

A. Yes.

Q. You mentioned earlier that you thought his name was Mark Plisker. It looks

like you have written down Mark Plisko?

A. Plisko.

Q. Had you ever met Mark Plisko before?

A. No.

Q. How did you get in touch with him so that you could make notes?

A. Mr. Scott suggested that I call him to discuss the nature of his assessment of the quantitative exposure that Mr. McCarty may have incurred as an employee of Univar.

Q. Did Mr. Scott ask you to talk to anybody else besides Mr. Mark Plisko about potential exposures of Mr. McCarty?

A. No.

Q. When did you make these notes?

A. Yesterday or the day before.

Q. Well, do you recall?

A. It was either yesterday or the day before. I can't think. I think it was

yesterday morning, but I could be mistaken.

Q. Your best estimate is you made those notes yesterday, August 28th, in the morning?

A. Yes.

Q. Did you call Mr. Plisko or did he call you?

A. I called him.

Q. Where did you call him from?

A. From my office.

Q. Is that the same number you gave me earlier?

A. Generally, yes, same switchboard.

Q. How long did you and Mr. Plisko talk?

A. Roughly, I would say half an hour.

Q. Who was present when you were talking with Mr. Plisko?

A. No one. Just of the two of us.

Q. You were the only one on your call --

A. Yes.

Q. -- on your end? And as you understood it, he was the only one in his end?

A. To my knowledge.

Q. Where was he when you were speaking

to him?

A. My assumption is he was at Environmental Profiles.

Q. Do you know where that is?

A. No.

Q. Do you know what area code that you

dialed?

A. I think it's East Coast.

Q. Do you know what city?

A. No.

Q. Had you ever heard of Mark Plisko before?

A. No.

Q. Do you think that based on your 30-minute conversation you were able to develop an idea about his reputation and ability to assess worker exposures to chemicals?

A. It's my understanding that he is associated with John Spencer, and I'm familiar with Mr. Spencer's credentials. To elaborate, I had asked Mr. Scott if he had a -- obtained an estimate of quantitative exposure in this case, and he referred me to Mr. Plisko.

Q. Just for the record, I need to object to responsiveness. My question was with

regard to your half-hour conversation of Mark Plisko, do you think that you had a sufficient ability to evaluate his qualifications and his reputation to be assessing worker exposures?

A. I believe I had sufficient opportunity to evaluate his expertise with

respect to this particular analysis, and I was satisfied with that.

Q. What is his expertise?

A. His expertise in terms of his credentials, I do not know.

Q. You don't even know if he is an industrial hygienist, correct?

A. That, I don't know.

Q. Do you typically rely upon people who give you industrial hygiene information when you don't even know if they are an industrial hygienist?

A. I rely on quantitative exposure assessments that are provided to me. I don't make quantitative assessments myself. I prefer to rely on exposure assessments by others, either engineers or industrial hygienists.

Q. So we are clear on that, you have not made independently on your own any

quantitative exposure assessment on Mr. McCarty, correct?

A. No.

Q. Do you feel like the data that you have, this one page note, and your half-hour conversation, provides you with sufficient

information to enable you as a scientist and health professional to accurately assess Mr. McCarty's exposure to benzene in his work at Van Waters & Rogers?

A. I feel like this provides me with an adequate basis to assess his exposure as it's outlined in the testimony that I have reviewed.

Q. So you feel like you have sufficient information from what Mr. Plisko has told you about Mr. McCarty's exposure; is that correct?

A. As I said, based upon the limited testimony that I have reviewed, I believe that this provides an adequate assessment of the conditions that are described in that testimony.

Q. All right. Without the testimony and just relying upon Mr. Plisko's notes that you took in that half-hour conversation yesterday, do you think Mr. Plisko's conversation with you and the notes you took

from that were sufficient in and of themselves to allow you to make a quantitative assessment of Mr. McCarty's exposure to benzene?

MR. SCOTT: Object to form.

A. Not without some basis in testimony or description to outline the conditions under

which the assumptions are made for the quantitative assessment. Quantitative assessment is only as good as the description of the conditions that have been provided either in written -- written form or testimony.

Q. (BY MR. BROWN) Do you know the information, the data and the facts, that Mr. Plisko relied upon to provide you with the notes that you have here in your Exhibit 1?

A. I know the assumptions that he outlined to me in describing his assessment, and I provided those summarized in my notes.

Q. Let me object to responsiveness. My question is, do you know what information he had, the data that he had, that he relied upon to give you the information that you wrote in your notes?

MR. SCOTT: Object to form.

A. As I said, the assessment that he

provided me is based on certain assumptions. Those assumptions are outlined in my notes.

Q. (BY MR. BROWN) What I'm asking you is, do you know what all assumptions that he had?

MR. SCOTT: Object to form.

A. I believe these are the assumptions that he had. These are certainly the assumptions that I had in evaluating his analysis and relying upon it.

Q. (BY MR. BROWN) Do you know if he had anything other than the testimony of Mr. Beverly, affidavit testimony of Mr. Beverly, and/or the affidavit testimony of Mr. McCarty regarding their exposures?

MR. SCOTT: Object to form.

A. He certainly had ACGIH modeling data and publications, which were the basis for the model that he used. With respect to exposure, I don't know anything more than what I've told you.

Q. (BY MR. BROWN) Again, I need to object to responsiveness. Forgive me. You can ignore them. Do you know if he had Mr. Beverly's affidavit, for instance?

A. I believe he did, yes.

Q. I'm asking, do you know that for sure?

A. From the conversation that we had, I believe that Mr. Beverly's testimony is one of the bases for the assumptions in this report.

Q. All right. And was that -- again, let me object to responsiveness. Do you know if he had Mr. Beverly's affidavit, as opposed to his deposition testimony?

A. I can't speak to that specifically.

Q. Do you know if he had Mr. McCarty's affidavit, sworn affidavit?

A. I can't speak to that. I don't know that.

Q. Do you know what particular documents he had at all? Did he tell you these are the documents I'm relying upon?

A. We discussed in general Mr. Beverly's testimony.

Q. So I guess from that you assume that he had a deposition testimony of Mr. Beverly?

A. Either that or the affidavit. I can't -- as I sit here, I can't be certain.

Q. All right. Anything other than

that, the testimony of Mr. Beverly?

A. Not that I know of. I can't speak to that.

Q. Next we have marked your -- what you identified as your CV, your current CV, as Exhibit 2; is that correct?

A. Yes.

Q. Is that document current and up to date?

A. Yes.

Q. As of when?

A. As of sometime this month.

Q. All right. Then for Exhibit 3, we put into the blue folder -- these are documents that we've marked as a group of studies which we have referred to as documents and studies related to your Shanghai studies, correct?

A. Yes.

Q. You don't mind if we take these or have the court reporter make copies of all Exhibits 1, 2 and 3; is that correct?

A. No.

Q. You mentioned that you were familiar with John Spencer's reputation and credentials, correct?

A. Yes.

Q. Have you in the past received quantitative exposure estimates from John Spencer?

A. Yes.

Q. Have you always found those to be

reliable?

A. Yes.

Q. Have you always found those to be reliable and sufficient -- and with sufficient enough data so that you could make an exposure assessment yourself?

A. I don't know whether I can -- I don't know quite how to answer that question.

MR. SCOTT: I object to form of the question.

A. An exposure assessment is always based upon information which may be anecdotal, it may be quantitative, it may be based on modeling, and it may be based on actual measurements. There may be more modeling in one case than another. I've always found that John Spencer's analyses are reliable in that they state the assumptions and they provide justification for the approach that was taken.

I can't say that every time I've evaluated an exposure assessment, whether it's from John Spencer or anyone else, that there's been a great deal of data sufficient to do a quantitative assessment.

Q. (BY MR. BROWN) That's what I'm

getting at. There have been times when John Spencer has provided you with quantitative exposure assessments of individuals involved in lawsuits where you were representing -- or you were testifying on behalf of defendants that you could not in good conscience rely upon what he did, correct?

A. No, I can't say that. I think that, again, the assumptions that go into an exposure assessment may be limited and the assumptions may limit the value of an exposure assessment. That does not necessarily reflect poorly on the assessor or the methodology.

Q. Well, my question is, there have been times, sir, that John Spencer has given you a quantitative exposure assessment which you did not rely upon, correct?

A. I think that's highly likely.

Q. In fact, you did it when you

testified in the James Cowey case back in November of 2003; do you recall that?

A. No, I don't.

Q. All right. Let me show you the testimony I'm referring to. Page 79 in the Cowey deposition. Mr. Lubel asked you, "But as

you sit here today as an expert witness for United States Steel Corporation, you can't tell us under oath whether or not the product that Mr. Spencer tested was in fact the product that had same or substantially similar ingredients to that which Mr. Cowey used; is that correct?" And you said, "I cannot say that, no."

MR. SCOTT: Object to the form of the question.

Q. (BY MR. BROWN) Did I read that correctly?

MR. SCOTT: Object to the form of the question.

A. You read that correctly, but that does not speak to the adequacy or the quality of Mr. Spencer's assessment.

Q. (BY MR. BROWN) Well, you recall now, after having read this, that Mr. Spencer did provide an assessment to you in that case,

correct?

MR. SCOTT: Object to form of the question.

A. Yes.

Q. (BY MR. BROWN) All right. Over here he asked you, "Did you earlier today in your

testimony make a statement that you didn't feel as though you were in a position to make a meaningful evaluation of Mr. Cowey's benzene exposure based upon the information that had been provided to you?"

Your answer was, "That's correct."

The next question is, "Do you stick by the statement that I'm now asking you in the question?"

You said, "Yes, that's correct."

Did I read that correctly?

A. Yes, you did.

MR. SCOTT: Object to the form of the question.

Q. (BY MR. BROWN) In that case, even though Mr. Spencer had provided you with exposure assessment, you did not feel like you had sufficient information to use one yourself. You used what he provided, correct?

A. Based upon that testimony, I believe that that's a fair description of the testimony. By the same token, as I said before, the nature of the data on which an exposure assessment is based is critical, and in that particular case apparently I wasn't prepared to reach a

conclusion as to what exposure was.

Q. All right. But in this case based on a 30-minute conversation with his assistant, you feel like you are; is that correct?

A. I am prepared to render an opinion with respect to the likelihood or probability that Mr. McCarty's disease was associated with his exposure as outlined in the testimony I reviewed and based upon the quantitative assessment that I have. That's -- that is my testimony.

Q. Let me ask it this way: Could you render an opinion based on quantitative exposure evidence and information without the 30-minute conversation that you had with Mark Plisko yesterday?

MR. SCOTT: Object to form of the question.

A. Based on the information and the

material that I've been provided, no.

Q. (BY MR. BROWN) So you have to rely upon Mr. Plisko's information in your 30-minute conversation yesterday to formulate your opinions about what Mr. Beverly's exposure would have been, correct?

MR. SCOTT: Object to the form of the question.

A. As we sit here today, yes.

Q. (BY MR. BROWN) And I believe I may have misspoke and said Mr. Beverly's exposure would have been. Let me reask the question. You have to rely upon Mr. Plisko's 30-minute conversation with you to determine and assess what Mr. McCarty's exposure would have been; is that correct?

MR. SCOTT: Object to form of the question.

A. Based upon the material I've reviewed in this case to date, that is correct.

Q. (BY MR. BROWN) Do you expect to be receiving any further or additional materials in the case?

A. I don't know.

Q. You have -- you don't have those

expectations as you sit here; is that correct?

A. No.

Q. I'm correct about that?

A. As we sit here, no.

Q. No, you do not have those expectations?

A. No.

Q. Okay. Have you prepared a written report in this case?

A. No.

Q. Not even a draft?

A. No.

Q. Have you been asked to?

A. No.

Q. Do you typically prepare a written report in a case like this?

A. If I'm asked. If not, no.

Q. Have you reviewed any written reports or opinions or documents from any of the other defendants who have been hired by Mr. Scott -- strike that. Have you reviewed any written reports or opinions or other documents from any of the other experts that have been hired by Mr. Scott in this case to testify on behalf of the defendant?

A. No.

Q. All right. You haven't reviewed anything in writing from Mr. Spencer, correct?

A. No.

Q. I'm correct about that?

A. Yes, you are correct.

Q. You haven't reviewed anything in writing from Dennis Paustenbach; is that correct?

A. That's correct.

Q. You haven't reviewed anything in writing from Mr. Raabe; is that correct?

A. That's correct.

Q. You haven't reviewed anything in writing from Mr. Nadelson; is that correct?

A. That's correct.

Q. Have you spoken to Mr. Spencer about this case at all?

A. No, I have not.

Q. Have you spoken to Mr. -- or Dr. Paustenbach about this case at all?

A. No, I have not.

Q. How about Mr. Raabe?

A. No, I have not.

MR. SCOTT: It's Dr. Raabe.

Q. (BY MR. BROWN) Have you spoken to Dr. Raabe about this case at all?

A. No.

Q. Have you spoken to Dr. Nadelson about this case at all?

A. No, I have not.

Q. Would it be fair to say the only person that you have spoken to other than Mr. Scott about the facts of this case would be Mark Plisko; is that correct?

A. That's correct.

Q. You've only had the one conversation with him yesterday morning?

A. Yes.

Q. Is it your understanding that Mark Plisko or Mr. Spencer has done any type of model of what Mr. McCarty's exposure would have been while he worked at Van Waters & Rogers?

A. That's basically what I provided in my notes.

Q. All right. So am I correct to infer that you have the understanding that they have prepared a model of what Mr. McCarty's exposure would have been?

A. Yes.

Q. Do you know what type of model that they prepared?

A. Yes. It's an inverted cone model predicated on a model for drum loading that is published by the American College of Government and Industrial Hygienists. I have a reference

to it in my notes.

Q. Prior to this case, had you ever seen any model regarding estimated exposure for drum-filling operations?

A. No.

Q. Do you have any idea about the facts or criteria by which the model for the drum-filling operations is controlled or governed?

A. Yes.

Q. All right. And where do they come from?

A. They come from my discussions with Mr. Plisko and the notes that I took.

Q. All right. What is your understanding of the parameters of the drum-filling operations that were involved in the model that Mr. Plisko did?

A. Well, as he described them to me,

first of all, they were based upon the frequency and rate of drum-filling that Mr. McCarty may have been involved in, was described very briefly in Mr. Beverly's testimony.

Q. All right.

A. And that was the basis for -- the

initial basis for the calculations that were performed.

Q. All right. What were the parameters in terms of the duration of the drum-filling activities in the model that you described?

A. The duration of the activities -- you mean as related to the individual exposures?

Q. No, sir. How long did the model, as you understood from Mr. Plisko, determine or use as the time that Mr. Oliver McCarty would have been doing the drumming activity on the days that he would be filling up the 55-gallon drums with benzene?

A. Specifically 240 minutes, 40 drums. It's basically a four-hour continuous-exposure assumption.

Q. All right. Did you see in the testimony that they may have drummed for as long as five hours each day?

A. There were -- as I recall, the range was described was either three to five or four to five. This particular model assumes a four-hour exposure.

Q. All right. Do you have any understanding why they chose four hours instead

of five?

A. This particular -- this particular model assumes no breaks, constant activity over a four-hour period. That seemed to me reasonable based upon my review of testimony. Other than that, I can't tell you precisely why any particular value was chosen other than trying to model the testimony that was provided.

Q. Well, let me object to responsiveness. I take you don't know why Plisko chose five -- or four hours rather than five hours, correct?

A. That's correct. I'm relying on the exposure assessment that was provided by Environmental Profiles and that Mr. Plisko described to me, and that's the basis for my opinion with respect to quantitative exposure.

Q. All right. Let me object to responsiveness for anything after "that's

correct." The exposure for four hours would result in a lower part per million than exposure for five hours at the same intensity, correct?

A. It would. It would result in a relatively minor difference.

Q. You say minor. About 20 percent

difference in the amount of time, correct --

A. 20 percent --

Q. -- or more?

A. Yes, I would consider that minor.

Q. All right. Do you know anything about the materials that were used in terms of the model?

A. The model assumes exposure to neat benzene.

Q. Pure benzene?

A. Pure benzene.

Q. Do you know anything about the regularity or the frequency that the drumming activity model assumes?

A. Constant. Over a four-hour period constant.

Q. I'm not talking about over the time from beginning of start-up to the first drum to the last drum. I'm talking about from the time

he drums on one day until the next batch of benzene comes in and they drum a different day?

MR. SCOTT: Object to form.

Q. (BY MR. BROWN) What does the model assume on that, as you understand it?

MR. SCOTT: Object to form of the

question.

A. The model assumes -- for overall cumulative exposure the model assumes a one-time, one-day-a-month frequency.

Q. (BY MR. BROWN) All right. And you were familiar with the testimony by Mr. Beverly that they would have done that as many as three times per month, correct?

MR. SCOTT: Object to form of the question.

A. As I -- to the best of my recollection, Mr. Beverly stated a range. One time he said one; one time he said maybe three.

Q. (BY MR. BROWN) Well, let me object to responsiveness. Do you know Mr. Beverly has testified that they would have been drumming benzene, pure benzene, from a truck in 55-gallon drums for as many as -- for as often as three times per month, correct?

MR. SCOTT: Object to form of the question.

A. I believe I said that, yes.

Q. (BY MR. BROWN) And so do you have an understanding of why it is that Mr. Plisko chose a one-time-a-month number as opposed to a

three-times-a-month number?

A. The one-time-a-month assumption does not change the nature of the quantitative estimate and can be adjusted up or down depending upon what assumptions you wish to make.

Q. Let me object to responsiveness. My question is, do you know why he chose only once per month as opposed to three times per month?

MR. SCOTT: Object to form of the question.

A. No.

Q. (BY MR. BROWN) In your conversation yesterday did you ask Mr. Plisko to fax you any of the information that he was telling you or informing you of over the telephone?

A. No.

Q. With regard to the model from drum-loading activities published by the

American Conference of Governmental Industrial Hygienists that you say you have, where did you acquire that?

A. I did not say I had that.

Q. I'm sorry.

A. I said that was the basis for this

particular assessment as it was related to me by Mr. Plisko.

Q. Okay. You were told that he used an ACGIH drum-modeling model?

A. I asked him to provide me for the basis, the publication that formed the basis for the mathematical modeling that he used, and he told me that this is the model that he used.

Q. And I assume that your notes there that are contained in Exhibit 1 after the exposure information tab are complete; is that correct?

A. Yes.

Q. There is nothing else that you all of talked about in your conversation relating to Mr. McCarty's exposure; is that correct?

A. That's correct.

Q. And there is nothing else that you need from Mr. Plisko or Mr. Spencer in order to

formulate your opinions in this case about Mr. McCarty's exposure; is that correct?

A. There is nothing else I need to render an opinion based upon this particular exposure assessment.

Q. All right. Well, I mean, and that's

what you've done, correct?

A. That's what I've done.

Q. What I'm asking you is, with regard to the exposure assessment that you have -- that you have now come to this deposition and intend to give opinions about, do you need anything else from Mr. Plisko or Mr. Spencer?

A. I think that I should restate that I am not offering an exposure assessment. I am relying upon the exposure assessment that was provided to me. And based upon the information that I have from that telephone conversation and my notes, I'm prepared to render an opinion based on that exposure assessment.

Q. Fair enough. What I'm saying is, do you have all the information that you need and all that information, is that contained in your notes in your notebook?

A. Yes.

Q. You indicated that you reviewed the medical records of Mr. McCarty, and I would assume that you would have reviewed his records from M.D. Anderson; is that correct?

A. M.D. Anderson and prior to that as well, I believe.

Q. The St. Elizabeth Hospital records and Dr. Schachner, Texas Oncology?

A. Yes.

Q. I believe you have got that up in the forward part of your notebook under Texas oncology?

A. Um-hum.

Q. Any other medical records that you reviewed on Mr. McCarty?

A. I've reviewed all the records that were provided to me. These are the ones that were salient with respect to my opinions in this case. He -- there are a lot of medical records.

Q. Have you reviewed Mr. McCarty's sworn affidavit?

A. I don't recall. Actually, I don't believe so.

Q. If he had given an affidavit --

A. No, I have -- I do and I have, yes.

I just don't recall.

Q. What do you recall from his affidavit, as you sit here, in terms of being significant to your opinions?

A. My opinions with respect to exposure, I've relied upon Mr. Beverly. My

opinions with respect to his diagnosis and his disease, I'm relying upon the medical records. So I have not relied on Mr. McCarty's affidavit with respect to either of the exposure assessment or the diagnosis.

Q. Why not? I mean, it's the man's sworn testimony. Don't you typically rely upon somebody's sworn testimony about what they say their exposure was?

MR. SCOTT: Object to the form of the question.

A. I don't believe that Mr. McCarty's testimony provided me with a basis for rendering an exposure assessment.

Q. (BY MR. BROWN) Why is that, based on what his affidavit said?

A. As I recall, to the best of my recollection, the only description I have that relates to his specific exposure at Univar

relating to drum-filling is contained in the affidavit and the testimony of Mr. Beverly.

Q. Are there any other documents that you reviewed or relied upon in forming your opinions in this case that are not contained in Exhibit 1, which is your case notebook file?

A. Other than the literature in general related to myelodysplastic syndrome and acute myeloid leukemia, I don't believe so.

Q. All right. And you told us with regard to the studies that you felt were substantial and significant to your opinions, you've included those in Exhibit 1; is that correct?

A. Yes.

Q. Let's mark the subpoena as Exhibit 4, please.

(Irons Deposition Exhibit 4 was marked.)

Q. I noticed you had a copy of Exhibit 4 in your notebook here?

A. Yes.

Q. When is the first time you saw Exhibit 4?

A. I believe it was the 25th of this

month.

Q. You brought with you a copy of your resume or CV, correct?

A. Yes.

Q. Do you have a current or present job description?

A. No.

Q. You don't have one either for the University of Colorado or for your work over in Shanghai?

A. No.

Q. I'm correct about that?

A. Yes.

Q. Have you brought with you all the documents that have been supplied to you or reviewed by you in preparation for your deposition?

A. Yes.

Q. All right. The next set of requests, 4 through 13, deal with documents relating to your study in Shanghai. Did you bring with you any -- anything other than the studies that relate to your Shanghai work?

A. Other than a description of the laboratory, no, I have not.

Q. Are you able to -- how do you communicate with the others in Shanghai when you are here in the states? Do you do that by email?

A. Others? I'm not sure what you are referring to.

Q. Are there others?

A. Who?

Q. Do you have doctors and epidemiologists and other people involved in the Shanghai study that are in China?

A. At any given time, I communicate with my laboratory, with pathologists, and hematologists in Shanghai. I communicate with others who are involved in -- on the research side of the project, both in the United States and China. I communicate through quantitative database interaction and also via email.

Q. How do you keep up with and store your correspondence or communications and email?

A. I review emails like anybody else. I don't store them. The data that's collected as part of this study is contained in a database that provides both support for the clinical operations and data that is used in the various

research projects.

Q. All right. What about other correspondence? Do you have correspondence that you do keep?

A. No.

Q. Next item is No. 5: Invoices,

receipts, bills, accounts receivable, and other documents reflecting any payment or consideration given to you by the University of Colorado relating to your work at Shanghai. Did you bring any such documents?

A. No.

Q. Does the University of Colorado, are they the ones who pay you for your work in Shanghai or somebody else?

A. I'm an employee of the University of Colorado. The University of Colorado provides me with a salary. The study in Shanghai is basically conducted through international treaty and contract involving the University of Colorado, the State of Colorado, Fudan University, the Shanghai Public Health Bureau, University of Cincinnati, the Chinese Ministry of Health and Exxon-Mobil Biomedical Sciences.

Q. So do you have invoices, receipts,

and checks that you get from those entities relating to payments made to you?

A. No, no payments are made directly to me other than the salary that I receive from the University of Colorado.

Q. Well, so for your work in Shanghai,

the University of Colorado pays you, correct?

A. The University of Colorado pays my salary. Part of that involves the studies that I'm doing in Shanghai.

Q. When you started these studies in Shanghai in 2000, was there an increase in your salary for the work you are doing in Shanghai?

A. No.

Q. With regard to Item No. 6, you can see that there, did you bring any documents that are responsive to that?

A. No.

Q. Do you have any in your possession?

A. I have received no scientific article -- no, I've not received any of those types of materials from any of the sponsoring corporations or any other company.

Q. Next item is any PowerPoint presentations relating to benzene studies being

conducted in Shanghai, China. Do you have any of those?

A. I have some PowerPoint files that contain data and/or information that I provided or presented at various meetings and scientific meetings. I did not bring those today.

Q. Why is that?

A. I informed Mr. Scott when I received the subpoena that a great deal of the information that you requested is either -- I'm prohibited by law from providing it to you, either Chinese or U.S., and that some of the information that you've asked for I need to ascertain from the participating organizations to get permission to provide them. And at this particular point, I didn't have any particular presentations that I felt that I could provide to you without going through that process.

Q. Others besides you have made PowerPoint presentations concerning efforts to gain additional members of the petroleum industry to participate in the funding of your Shanghai studies, correct?

MR. SCOTT: Object to form of the question.

A. I can't speak to that in an informed way one way or the other.

Q. (BY MR. BROWN) You have no knowledge of that?

A. No.

Q. With regard to No. 8, do you have

any documents responsive to No. 8?

A. It's essentially the same response that I gave before. The memoranda and correspondence that I have relating to the individual sponsoring companies concerns solely the work product progress and budgetary items related to the project, and at this point I don't feel that I can provide those to you within the limitations that I have both legally and contractually.

Q. Can you check with the member companies and find out if there would be any problems with us having any of that correspondence?

MR. SCOTT: Object to the form of the question.

A. The only correspondence I have relates to progress on the budget. And the budget with respect to the study, like any other

research study, is confidential, and this would require both the approval of the University of Colorado, Fudan University, and the various -- not sponsoring -- the various participating organizations that I mentioned.

Q. (BY MR. BROWN) The only way that we

would be able to obtain copies of that would be have a court order to get it; is that right?

MR. SCOTT: Object to the form of the question.

A. I'm not an attorney. I can't tell you that.

Q. (BY MR. BROWN) You are not going to turn it over to me just for the asking. You are telling me that, correct?

A. Yes.

Q. So if it gets turned over to me, a court is going to have to tell you to do; is that right?

MR. SCOTT: Object to the form of the question.

A. I presume so.

Q. (BY MR. BROWN) With regard to any of the other enumerated items there through 14, do you have any of those items in your possession

that you brought with you today?

A. No.

Q. Do you have any other items in your possession that you -- would be responsive to any of those specific enumerated requests and, if so, would you be able to provide me with a

copy of them?

A. I'm not sure I understand the question.

Q. Well, I mean, I guess we could go through it one by one and say -- you know, ask the question, but if you could go down the list and say, yes, I have documents responsive to this question but I can't give them to you because of X reason or I will give them to you -- could you do that for me, please?

A. I think we would have to go through them one by one. Some of these I'm prohibited by U.S. law. I can't give them to you. No. 10, I'm prohibited by the U.S. Common Rules. CFR 41, whatever it is, relating to independence of clinical trials and confidentiality of patient and clinical information. Information such as that the sponsors in the studies don't have, and it isn't public, and therefore I can't as a

principal investigator on this study provide many of these things based upon those regulations and governing procedures.

Q. All right. What about the rest of them?

A. 12 we've discussed. 10 we've

discussed.

Q. Well, 12, I don't know if we discussed any expenses that you've incurred and if there is any evidence or documents that show that relating to your work at Shanghai.

A. This would fall under the same restrictions that -- with the budgetary. I mean, this is all part and parcel of the budgetary issues relating to the study.

Q. Have there been any payments like in No. 13 from any of the oil companies funding the study made directly to you?

A. No.

Q. All right. He needs to change his tape so we need to go off the record.

THE VIDEOGRAPHER: The time is approximately 11:51 a.m. and we are now off the record.

(A break was taken.)

THE VIDEOGRAPHER: The time is approximately 11:55 a.m. and we are back on the record.

Q. (BY MR. BROWN) Sir, we were about finished talking about the subpoena duces tecum. I think we were to the last page. Is there Item

14 on there or is 13 the last one?

A. I think 13 is the last one.

Q. We are finished. With regard to the notes of your conversation with Mr. Plisko yesterday and his exposure assessment model, would you agree if the information that he has provided you in his model was incorrect in any way that you would have an inaccurate exposure assessment?

A. To the degree that those particular assumptions influence the model, that's correct. Some of them would influence the model in a great -- in a very significant way; others would influence the model in a minor way. That's the nature of an exposure assessment.

Q. All right. Every one that you've written down in your notes you felt like was important, correct?

A. I felt that these were in fact the

assumptions that were the basis for the calculations that he did. Some are more critical than others.

Q. Which ones are more critical than others, in your opinion?

A. Airspeed is critical.

Q. Airspeed of what?

A. Air. The speed of air in the area in which the drumming is being performed and conducted is a critical factor. The --

Q. Why was there any reference to airspeed at all?

A. Because the model assumes a concentration associated with an inverted cone around the drum and in the area of the operator.

Q. All right.

A. That concentration and the equilibration within that cone is dependent upon airspeed.

Q. What was the airspeed that was assumed?

A. One mile per hour.

Q. All right. Is that typical of a vapor recovery system or exhaust system in place?

A. This is -- my understanding is that this does not involve a recovery system. This is open or, slash, loading. Now, that's my assumption based upon the conversation that I had, that in fact this particular model does not -- is not associated with a vapor recovery

system.

Q. Is it your assumption that this model that you have is not associated with any engineering controls regarding ventilation?

A. I believe so, yes.

Q. That's very important to know that?

A. That's my assumption.

Q. If that assumption is incorrect, then your exposure assessment and the things that you've relied upon would be flawed, correct?

MR. SCOTT: Object to form.

A. I have not made a exposure assessment. This particular assessment would vary depending upon the assumptions that go into it.

Q. (BY MR. BROWN) Your opinions based upon this exposure assessment would be flawed if the exposure assessment is wrong, correct?

A. If in fact the conditions are different, then I would have to review whatever other exposure assessment is performed and understand it before I would be willing to render an opinion on that particular quantitative exposure assessment.

Q. You would want to make sure that what Mr. Plisko was telling you were the conditions better have been the conditions that Mr. McCarty was exposed to before you make opinions about whether or not his exposure could have caused his illness, correct? That's only fair, isn't it?

MR. SCOTT: Object to form of the question.

A. As an expert, all I can do is render an opinion on the information I have been provided and that I rely upon. My opinion relates -- my opinion with respect to quantitative exposure is based on this assessment. If in fact this assessment is modified, changed, or improved or invalidated, that would change my opinion. With respect to the exposure assessment, I would have to render a opinion based on that exposure assessment.

Q. (BY MR. BROWN) That's the old rule garbage in, garbage out, correct?

A. I don't know what you are referring to.

Q. Well, your opinions in this case that you are going to be providing are only as

good as the information that you have to base them on, correct?

A. That's the nature of any expert opinion.

Q. Okay. And you, as you are sitting here, you are not an expert on exposure assessments, are you?

A. Not as an industrial hygienist. As a toxicologist I am.

Q. Well, you are not an expert and you are not going to be providing opinions as to whether or not the exposure assessment that has been provided to you is done in compliance with all industrial hygiene standards and in compliance with good practices, correct?

A. I can't speak to the specific industrial hygiene standards that relate to the assessment. I am assuming that in fact the model that was used in fact is consistent with

the mathematical modeling that is published by the ACGIH. I can only render an opinion based upon the expertise of the industrial hygienists and engineers that provided this to me.

Q. Okay. That's what I'm getting at. I don't think we are disagreeing. You are not

the guy to come in and say this model is appropriate and is a reasonable relationship or reasonable estimate of what was going on; you are relying upon Mr. Spencer's group or Mr. Plisko to tell you that, correct?

A. I'm relying upon them to tell me about the model they used and I am assuming that the conditions and assumptions that are part of the model in fact are accurate insofar as we can determine.

Q. All right. If we want to know -- without an assumption, if we want to know for sure, we need to ask Mr. Plisko or Mr. Spencer, correct?

A. With respect to this model, correct.

Q. One thing you told me is that you thought that there was an assumption that Mr. McCarty was filling drums with benzene on a one-time-a-month basis, correct?

A. That is what this model that I have in front of me assumes.

Q. All right. I think it says down here six times a year. Do you see that, six days a year?

A. That's cumulative, yes.

Q. That's equivalent to two times, once every two months, correct?

A. No. That's six days a year at four hours a day. Four hours a day is roughly half a day. So over a year period it would amount to six days a year. 240 minutes represents basically half a day. So this assumes a six-day-a-year total. You can modify the quantitative assessment based upon changes in assumption with respect to the times per month or with respect to the number of days, depending upon whatever information that you have.

Q. When you typically rely upon exposure assessments in performing opinions about causation of benzene-related illnesses, are they typically in the format that you have right here, based on a 30-minute telephone conversation with somebody you don't know?

MR. SCOTT: Object to form of the

question.

A. I rely on exposure assessments that are provided to me that range from very detailed reports to very sparse information, depending upon what the nature of the information is and what I've been asked to do. I am not providing

the exposure assessment here. I'm relying on this quantitative exposure assessment.

Q. (BY MR. BROWN) All right. We were discussing the things that you thought were most critical on the assumptions that you were making or on the model and the notes that you took from the model that you -- was discussed with you. What other besides airspeed was significant or would be of the most critical factors on that model?

A. The airspeed is the most critical. It changes -- it changes the quantitative assessment in a linear way, which means if you fluctuate the airspeed, you change the nature of the assessment --

Q. All right.

A. -- number.

Q. What besides airspeed then is critical?

A. I think that's the most critical. The rest of them are part of the equation. If you take any one of them out, you don't have an equation. But in terms of sensitivity with respect to how it changes the nature of the exposure assessment, the airspeed is the

critical factor.

Q. In the line of ranking, the assumptions that are in your notes there, from most critical to least critical, airspeed would be at the top, then; is that correct?

A. Airspeed would be at the top.

Q. What would be next?

A. Number of air changes.

Q. What is that?

A. That's the number of times per minute that the volume of air changes. And those are -- those are related, but the airspeed is probably the driving factor.

Q. All right. The number of times that the air changes, what is the assumption in the notes that you have there?

A. It's 15 cubic meters per minute.

Q. What's the next most critical factor in your opinion?

A. The fact that this is neat benzene.

Q. Do you have any reason to doubt that what was being drummed into the 55-gallon drums was pure benzene?

A. I haven't seen anything in the information that was provided to me that would

corroborate or document that in fact it was benzene. I am simply assuming for purposes of this analysis that it is benzene. I have no basis in terms of what I've read to rely or to conclude that in fact benzene, neat benzene was being drummed.

Q. Well, you have read the deposition of Mr. Beverly, and I know you told us you didn't read the deposition or affidavit of Mr. McCarty, but you know --

A. I did read Mr. McCarty's affidavit.

Q. Oh, you did.

MR. SCOTT: Object to the form of the question.

A. I don't recall his affidavit in terms of substance.

Q. (BY MR. BROWN) Okay. Did you see where Mr. Beverly said it was benzene that they were drumming?

A. I did.

Q. Did you see Mr. Harold Wellen's testimony where he said, "Yes, in fact we did drum benzene at that facility during the 1960s"?

MR. SCOTT: Object to form of the question.

A. I recall seeing references to the possibility of drumming at that facility. I don't recall that it was during the same period that we -- that's under -- that's at issue in this case.

Q. (BY MR. BROWN) What period do you recall benzene having been drummed from Harold Wellen's testimony?

A. I don't recall specifically Mr. Wellen's. I reviewed a great deal of testimony relating to this particular site and the information that was provided to me in the exhibits that Frank Parker provided, and in there, there were frequent references to the fact that benzene was not drummed at this facility, and I recall there was one reference that suggested perhaps there was -- that there's a possibility of benzene being drummed in the late 1960s.

Q. Let me object to responsiveness.

With regard to the deposition of Harold Wellen -- well, let me ask: Did you see the notes, the invoice documents from ARCO which said benzene was delivered to the Van Waters & Rogers Beaumont facility in tank trucks to be

drummed out of the tank trucks?

A. If -- I don't recall specifically those documents. If you would like to discuss them, I would like to pull them out and refer to them.

Q. I'm just asking if you recall them.

A. I don't recall them specifically.

Q. With regard to the next most critical factor in the assumptions that were made in the exposure estimate, what is next in line after pure benzene?

A. Probably the rate of drumming.

Q. The fill rate?

A. Yes. The number of drums per minute is probably next, although at this point we begin to get into areas that I don't think -- I mean, this assumes an inverted cone model, which is what the ACGIH assessment strategy for estimating exposure from drumming uses. If you

accept that model based on the ACGIH, then that's what it is, an inverted cone. So there are aspects of this model that if you eliminate those assumptions, you don't have a model, but I wouldn't say they are critical with respect to taking the individual conditions and numbers

that are used in this equation and calculating an exposure estimate from it.

Q. Are there assumptions in that, your notes, dealing with the temperature that it is alleged to have been drummed in?

A. There -- is it Mr. Plisko? I'm not sure. Mr. Plisko described to me the condition standard temperature and pressure. He related to me the temperature that was used in the model. I did not write that down because it was not -- in terms of my use of this model, I wasn't prepared to evaluate the role of temperature in the actual equation. But I believe that it was on the order of 70, or something like that, Fahrenheit.

Q. 70 degrees Fahrenheit?

A. I believe, but I'm not sure.

Q. It would be a huge difference in the amount of benzene vapor getting into the air if

it was being drummed in 90-degree temperature as opposed to 70-degree temperature, correct?

A. That would make some difference. You would have to ask Dr. Spencer or Mr. Plisko as to the specific impact that would have on the calculation.

Q. All right. You have not done that as part of your reliance upon his work that you've got there, correct?

A. No.

Q. The size of the bung holes that are in the drum, would that make a difference in the amount of benzene vapor escaping into the air?

A. I couldn't tell you that. Obviously if you are talking about an open container or you are talking about a bung hole, it would make a difference. How that impacts on the model, I can't tell you.

Q. Just some more background information. Prior to your deposition today, did you have a chance to meet with Mr. Scott?

A. I've discussed this case with Mr. Scott, yes.

Q. Did you discuss this case with Mr. Scott today?

A. Briefly, yes.

Q. Well, for how long?

A. Probably half an hour related to this case.

Q. All right. You met with him this morning, what time?

A. 9 o'clock.

Q. Did you meet with him yesterday?

A. Yes.

Q. What time did you all meet yesterday?

A. We met yesterday afternoon from approximately 3 o'clock till about 6 or 8, somewhere in there.

Q. All right. Any other meetings in preparation for your deposition besides today and yesterday?

A. Specifically relating to deposition, no. I believe I've consulted by telephone with Mr. Scott on matters related to this case maybe a total of an hour and a half, maybe two hours over the last several months.

Q. Are you being compensated for your time here today, sir?

A. Yes.

Q. What is your rate of compensation for your time in testifying?

A. My rate for consulting in all matters is $500 an hour.

Q. Do you know how many total hours you've spent in this case so far?

A. All told, maybe 30. I'm not sure.

Q. All right. So we are talking about $15,000 is a ballpark estimate of what you've been paid in this case so far for your time?

A. I haven't billed yet in this case, so I haven't been paid.

Q. All right. With regard to the 30 hours that you spent, you will bill at $500 an hour regardless of what you were doing during the hour in working on this case --

A. Correct.

Q. -- whether it's deposition, prep time, travel time, or anything else; is that correct?

A. That's correct.

Q. As of right now if you had to bill Mr. Scott, you would bill him about $15,000; is that correct?

A. I believe so.

Q. Is your educational background accurately stated in your CV, sir?

A. Yes.

Q. Is the work history accurately stated in your CV?

A. Yes.

Q. Do you have any licenses or certifications in any fields?

A. I am board-certified in toxicology. I have an inactive license as a clinical laboratory scientist in the state of California. I have an active license to operate a laboratory and render laboratory diagnoses in the People's Republic of China. I have a clinic laboratory license in Shanghai.

Q. You've told us you are not an epidemiologist and a medical doctor, correct?

A. That's correct.

Q. You are not a certified safety professional; is that right?

A. I'm a toxicologist.

Q. All right. You are not a certified safety professional, correct?

A. That's a jargon I'm not familiar with. I'm not an industrial hygienist.

Q. You are not an industrial hygienist, and there's a -- are you aware of a certification in the field of safety that people can get and they are entitled certified safety professionals?

A. Well, I don't have a certification.

Q. You are not an engineer; is that correct?

A. That's correct.

Q. Do you have any legal training as a lawyer?

A. No.

Q. Do you consider yourself to be an OSHA compliance expert?

A. No.

Q. Do you consider yourself to be an expert in human factors engineering or warnings expert?

A. No.

Q. Have you ever had your testimony or opinions stricken, disqualified, or limited by any court in any case where you've been an expert witness?

A. I know of one occasion where I believe a judge in West Virginia ordered me to

render an opinion I did not hold. Ordered me not to -- sorry -- ordered me not to render an opinion that I did not hold.

Q. So a judge has ordered you or prohibited you in the past from rendering an opinion in a case where you were testifying as

an expert; is that correct?

MR. SCOTT: Object to the form of the question.

A. I believe what I've said -- what I said is that a judge has ordered me not to render an opinion which I also did not have. So I did not make an opinion that a judge ordered me not to give.

Q. (BY MR. BROWN) What was the order? Do you remember what the judge ordered you not to give an opinion on?

A. Not as I sit here. I can't -- I can't state it precisely. It's fairly obtuse.

Q. Well, just in general. I'm not asking for an exact.

A. I believe it related to offering an opinion associated with what -- with a particular type of cytogenetic abnormality associated with leukemia.

Q. The cytogenetic abnormality, was it a loss or deletion of certain chromosomes?

A. Yes.

Q. Did it deal with a loss or deletion of chromosome 5 or 7?

A. One or both. I can't recall.

Q. Do you recall if he was saying you cannot give an opinion on what that means in terms of etiology of disease or causation?

A. No. It was far more restricted than that. It was far -- it wasn't that general at all.

Q. Do you have a copy of that order?

A. No.

Q. Do you remember the lawyer who had hired you in the case where you were limited like that?

A. Joseph Hollingsworth.

Q. When did that occur?

A. A long time ago. I don't remember precisely.

Q. In your entire career, have you ever testified as an expert on behalf of a cancer victim who has brought suit against a company claiming a chemical exposure caused his or her

disease?

MR. SCOTT: Object to form of the question.

A. No.

Q. (BY MR. BROWN) Have you ever testified as an expert on behalf of any

plaintiff in a personal injury case?

A. I have not given any testimony on behalf of a plaintiff. Excuse me. I have to qualify that. I have not given any testimony on behalf of an individual. I have given testimony on behalf of a plaintiff.

Q. Let me object to responsiveness. Have you ever given any testimony on behalf of a plaintiff who was an individual involved in a personal injury case?

A. No.

Q. When you have testified in the past in cases where someone had brought suit claiming exposure to chemicals had caused them to acquire an illness, it has always been for the defendant, correct?

A. I'm sorry; could you repeat the question.

Q. Yes, sir. With regard to the

testimony in the past, you always testified on behalf of defendants, correct?

A. In personal injury cases, that's correct.

Q. And most of those times in a personal injury case, if not all, have been

testifying for defendants who are oil or chemical companies, correct?

A. Not all but certainly most.

Q. When have you testified in a case involving a plaintiff claiming a personal injury as a result of exposure where you were not testifying for an oil or chemical company?

A. Although I can't think of a specific case, I'm fairly certain I've testified in cases or consulted in cases in which the defendant was a premises or a factory or a company that was not an oil or petroleum company or a chemical company.

Q. Have you testified for pharmaceutical companies?

A. I have certainly consulted for pharmaceutical companies. I'm trying to remember whether I've testified. I don't specifically recall having testified in a

personal injury case on behalf of a pharmaceutical company. I have consulted with pharmaceutical companies on many issues.

Q. The publications that you have, those are accurately set forth in your CV; is that correct?

A. That's correct.

Q. Have you ever submitted an article for publication that was denied?

A. Oh, yes.

Q. Have you ever submitted an article for publication on benzene that was denied?

A. Oh, yeah.

Q. And were there efforts by you to go back and change it, the article, so that it could be published?

A. I'm not sure how to respond to that. I'm not sure exactly what you are asking. Could you restate the question.

Q. Yes, sir. I mean, you've -- you said you've submitted articles relating to benzene that have been denied, and I was wondering, when that occurred, did you go back and try to, with regard to the part that was in dispute, reconsider it and so that it could be

published?

MR. SCOTT: Object to form of the question.

A. I don't think that's an accurate representation of the peer review process, so it's difficult for me to answer that in a

responsible way.

Q. (BY MR. BROWN) Can you answer it one way or the other?

A. Yes, I can answer it. It will take a while. In the peer review process, a scientist writes a manuscript or a paper that is based upon a study, based upon data and interpretation of that data, design, other issues related to research or findings, observations, and submits them to a journal for consideration for publication. Part of that involves editorial determination on the part of the journal whether the article is topical or the material is of sufficient interest to the readership to warrant publication.

If in fact the article is deemed to have sufficient interest for the readership, then it will be submitted -- should be submitted for peer review by independent reviewers, who

will review the article for scientific merit.

In general if it is deemed to have sufficient scientific merit, there will then be some recommendations for modifying the paper or changing it, which is called revision. For the majority of articles that I've submitted for

publication, they have involved some revision one way or the other. That's the nature of the process. Some on rare occasions are accepted without revision.

In at this particular point in time, competition in the scientific and medical literature is such that it's very frequent that you will submit a paper for publication and it will never get to review because the editor decides it's not of sufficient topical interest to warrant publication. I have that happen all the time. As a matter of fact, that's a frequent problem relating to benzene because the general interest in the scientific and medical communities for benzene is far less than it used to be. And therefore often a journal will decide that papers relating to benzene per se are not of sufficient interest to warrant publication.

Q. How many articles have you submitted where -- on benzene where they were denied not because of lack of interest but because of something that you stated in the article that they either asked for revisions or for some other reasons that they didn't want to publish?

A. I couldn't tell you. I don't recall having any specific articles that I've submitted that have not at one point or another been published. If not in the first journal, then the second or the third. That's the nature of the process. Some undergo extensive revision, some don't.

Q. As of last year, 2005, what percentage of your income was derived from testifying as an expert on behalf of defendant oil companies?

A. I can't recall exactly, but I would say less than 20 percent. Again, that, I can't tell you as I sit here exactly what it is.

Q. Could it have been as high as 50 percent?

A. No.

Q. Has it ever in any year been as high as 50 percent, the amount of income you've

derived testifying as an expert in litigation?

A. Yes, probably.

Q. How long ago was that?

A. Five, six, seven years ago.

Somewhere in that, on the order.

Q. Slowing down a little, I guess?

MR. SCOTT: Object to form of the question.

A. I'm busy.

Q. (BY MR. BROWN) Okay. I mean, is that a fair statement that you are slowing down in terms of the amount of work you are doing as a defendant expert on behalf of oil or chemical companies involved in litigation?

MR. SCOTT: Object to form of the question.

A. That's certainly been the case for the last few years.

Q. (BY MR. BROWN) Can you give us an idea of how much money you earned last year in testifying for oil and chemical companies?

A. No, as a matter of fact I can't.

Q. Don't have any idea?

A. No. It's 20 percent or less of my income, as I recall.

Q. How many active cases do you have where you are retained as an expert for a defendant oil or chemical company?

MR. SCOTT: Object to form of the question. When you say active, Darren -- I'm not trying to interrupt your question; I'm just

trying to understand the question.

Q. (BY MR. BROWN) Active files.

MR. SCOTT: Object to form of the question.

A. I don't think I can tell you, simply because I don't know what's active and what isn't. I don't know -- I would say at the present time, a few.

Q. (BY MR. BROWN) Well, let me ask you this. When a case is resolved, do you usually keep everything about the case and keep it in storage, or do you get rid of it?

A. I get rid of it.

Q. All right. So when you have a case, you usually have a notebook like this on a case?

A. Um-hum.

Q. Is that right?

A. Yes, if I'm preparing a report or preparing for a deposition or trial, yes.

Q. All right. When you open a file or are retained in a case, do you keep a file, contract in the file, and some kind of evidence that you've opened the case so you can have that for your records?

A. I don't -- I don't sign retention

letters. I don't -- I mean, I don't sign contracts. So I have no contracts and/or --

Q. How is it that you keep up with the cases that you've been asked to open?

A. If I have -- if I have file information relating to a case, whether it's testimony or medical records, I will keep notes that are related to that.

Q. Correspondence where you've been retained or asked to help?

A. Yes.

Q. How many of those letters, correspondence, letters, that you have that are active and haven't been thrown away, in your filing cabinets?

A. Probably between -- probably less than a dozen. Maybe six. Probably more than six. I can't tell you exactly.

Q. Between six and a dozen; is that

right?

A. I would say yes. That I've been contacted at one level or another.

Q. And that are currently still active; is that right?

A. That I can't tell you.

MR. SCOTT: Object to form of the question.

Q. (BY MR. BROWN) Well, they haven't been thrown away, is what I'm asking?

A. I haven't thrown them away. That doesn't mean the cases are still active. I don't think I'm necessarily kept that current with respect to what's going on in terms of litigation.

Q. Would it be fair to say that you have earned a good living working for oil companies testifying as expert when you've been hired by Mr. Scott or Mr. Dillard or somebody else who is representing them in lawsuits?

MR. SCOTT: Object to form of the question.

A. I'm sorry. If I had to rely on my income from testifying, I think I would be in pretty poor shape right now.

Q. (BY MR. BROWN) So the answer to my question is, no, you don't think you've earned a good living doing this?

A. I'm certainly not earning a living from testifying.

Q. Let's talk about hazards of benzene.

You would agree, sir, that benzene is a poison, correct?

A. Yes, almost all -- all chemicals are poisons, and benzene is a poison.

Q. When you say almost all chemicals are poison, does that include water?

A. Yes, water is a poison, and if you drink enough fast enough, it will kill you.

Q. Do you consider what degree of toxicity or poisonous nature that benzene is on any stretch of the imagination equivalent to water?

MR. SCOTT: Object to form of the question.

A. No, but all chemicals are poisons. So if you ask me if benzene is a poison, as a toxicologist my answer is yes, and all chemicals are poisons. Benzene is more toxic than water. It is more potent than water.

Q. (BY MR. BROWN) Benzene is an extremely insidious and toxic poison, correct?

A. I wouldn't agree to that characterization.

Q. Would you agree it's an insidious poison?

A. No.

Q. So you would dispute the National Safety Council in their 1950 document where they said benzene is one of the most insidious poisons ever to find wide industrial use?

A. Insidious is not a toxicologic term. It's not a technical term. It's an adjective. I wouldn't consider any poison necessarily to be insidious. Benzene causes chronic toxicity. It causes acute toxicity. I don't think insidious is a technically useful term in describing a poison.

Q. Let me object to responsiveness. My question was, would you agree with the National Safety Council in its 1950 document where they describe benzene as one of the most insidious poisons ever to find wide industrial use?

MR. SCOTT: Object to form of the question.

A. I wouldn't consider that kind of a statement to be a type of statement I would rely upon in rendering an opinion as a toxicologist.

Q. (BY MR. BROWN) My question was, would you agree or disagree with it?

A. I disagree with the statement as a

toxicologist.

Q. You agree that benzene is a cancer-causing substance, correct?

A. I do.

Q. That it has been determined to be a cancer-causing substance in humans by nearly every governmental agency or entity who studies cancer causation, correct?

A. The science relating to the toxicity of benzene demonstrates that chronic exposure can lead to the development of acute myeloid leukemia. Any government agency that relies on or reviews the world literature should reach the same conclusion.

Q. Acute myeloid leukemia is cancer of the blood, correct?

A. It is one cancer of the blood.

Q. It is the type of cancer that Mr. McCarty had, correct?

A. It is part of the spectrum of bone marrow disease that Mr. McCarty had.

Q. The other part was meylodysplastic syndrome that transformed to acute myelogenous leukemia, correct?

A. Apparently that's the case, yes.

Certainly the laboratory records demonstrate that he had refractory anemia with excess blasts. Whether or not in fact that transformed into what is technically AML is not significant with respect to the nature or severity of the disease that he had.

Q. Let me object to the responsiveness. IARC, what is IARC?

A. International Agency for Research on Cancer.

Q. They have determined that benzene is a confirmed carcinogen, correct?

A. That's correct.

Q. National Toxicology Program, what is that?

A. That's a research arm of the National Institute of Environmental Health Sciences based on -- I believe it's still part of the National Institute of Environmental

Sciences, but I'm not sure administratively whether it is.

Q. That's an organization that has determined that benzene is a known cancer-causing agent in man, correct?

A. I believe the NTP has based their

criteria on animal studies, to the best of my recollection they have in fact determined that it does produce cancers in animals.

Q. All right. Is it your testimony that the National Toxicology Program does not consider benzene to be carcinogenic in man?

A. I don't recall whether the NTP rates carcinogens, human carcinogens, specifically on human data.

Q. So you don't know; is that fair?

MR. SCOTT: Object to the form of the question.

A. I'm just answering -- I'm trying to answer responsively your specific question. The NTP does animal studies, and I believe that they have classified benzene according to those studies. Part of that classification scheme, as I recall, does involve probable human carcinogen.

Q. (BY MR. BROWN) What is the National Toxicology Program's assessment of benzene and whether or not it is confirmed or a probable carcinogen for a possible carcinogen?

A. It's confirmed.

Q. How about OSHA, do they recognize

benzene as a cancer-causing substance in man?

A. Yeah, I believe so, yes.

Q. How about NIOSH?

A. I believe so.

Q. What about the Environmental Protection Agency?

A. Yes.

Q. You yourself recognize benzene as a cancer-causing substance, correct?

A. That's correct.

Q. And you would agree that it is generally accepted among members of the medical and scientific field who study cancer that benzene can cause cancer, correct?

A. Yes.

Q. You recognize that exposure to benzene can cause acute myelogenous leukemia, correct?

A. Acute myeloid leukemia, yes.

Q. Do you also recognize that it can cause acute myelogenous leukemia?

A. That term is not the currently accepted terminology for the diagnosis of the disease, which is why I differed. You can call it myelogenous, but the current WHO standard

classification refers to it specifically as acute myeloid. It's a trivial point, but it's a specific term.

Q. Is it a point that makes any difference in your opinions in this case?

A. No.

Q. What types of AML do you believe are sufficiently linked to exposure to benzene?

A. Based on the current classification scheme, it's difficult to ascertain specifically which -- within the current classification, which particular subtypes have or which particular subtypes haven't been demonstrated to be caused by benzene but I would say in general it's impossible to exclude the possibility of any particular subtype. Some have far more evidence associated with them than others.

Q. All right. As a toxicologist, as a defendant's expert in this case, are there any

types of AML or variants of AML which you can rule out as being associated with benzene?

MR. SCOTT: Object to the form of the question.

A. I think there are specific types for which there has not been -- one can't say that

it's more probable than not that benzene is associated with their development, but I also can't say that benzene is not associated with their development.

Q. (BY MR. BROWN) Just for the record, and respectfully, I need to object to responsiveness, my question is simply is there any type of AML or its variants which you as a toxicologist can rule out as not being associated with benzene?

A. I can't prove a negative. I can't rule out anything. I can, however -- I do have opinions with respect to the level of evidence associated with specific subtypes.

Q. You recognize that exposure to benzene can cause meylodysplastic syndrome, correct?

A. Yes.

Q. You would agree with me that

epidemiological evidence is strong that exposure to sufficient levels of benzene can cause acute myelogenous or acute myeloid leukemia in man, correct?

A. Yes.

Q. What are some of the other diseases

or conditions of the blood and blood-forming elements that you believe can be caused by exposure to sufficient levels of benzene?

A. Specific diseases involve myelodysplastic syndrome, some of its subtypes, acute myeloid leukemia. Chronic exposure to benzene can cause specific abnormalities of the blood that may or may not be considered diseases but certainly are conditions. Pancytopenia, cytopenias, to some extent anemias. Did I mention individual cytopenias? Thrombocytopenia.

Q. Leukopenia?

A. That's synonymous with cytopenia, yes.

Q. Okay. Aplastic anemia?

A. That's been a prevailing opinion for many years. The number of actual cases for which there is pathologic confirmation is very

small. At this point in time I'm not -- I'm not, as I sit here, able to tell you whether or not aplastic anemia and myelodysplastic syndrome can be differentiated in terms of causation by benzene.

Q. I don't know if I -- let me object

to responsiveness because I don't know if I understand what you said. My question is, do you have an opinion that aplastic anemia is associated with exposure to benzene?

A. The literature definitely -- the previous historical literature provides an indication that exposure to benzene is associated with development of aplastic anemia. I personally -- I can't render an opinion with respect to reasonable scientific certainty, but I personally have some question as to whether those cases of aplastic anemia were not in fact myelodysplastic syndrome.

Q. Let me ask it this way. Would you agree that it's generally accepted among members of the medical and scientific fields in medicine that benzene is associated with causing aplastic anemia?

A. Yes.

Q. And you just have an opinion that it might not be because somebody got the diagnosis wrong and diagnosed aplastic anemia instead of meylodysplastic syndrome; is that correct?

A. Many times.

Q. Meylodysplastic syndromes are

related to benzene; you've told us that, correct?

A. That's correct. Let me clarify this. I can -- I'm offering -- I'm giving you an opinion with respect to reasonable scientific probability, but I also have opinions that I cannot -- or I have information and/or a perception with respect to the actual evidence that does not rise to that level of certainty and that I can't offer as an expert opinion.

I can and I have said that the literature indicates, the literature supports the general scientific consensus that chronic exposure to benzene can cause aplastic anemia. At this point in time, as a scientist, I have some questions whether the diagnoses and the criteria that were used in the past to make those -- to render that diagnosis were in fact adequate.

Q. Let me object to everything after "that's correct." Do you have an opinion whether or not benzene can cause chronic lymphocytic leukemia?

A. I don't believe that there is sufficient evidence in the scientific and

medical literature to reach that conclusion.

Q. My question is, Do you have an opinion that benzene can or cannot cause chronic lymphocytic leukemia?

MR. SCOTT: Object to the form of the question.

A. I believe I've answered that. I don't believe that to a reasonable scientific and medical probability there's evidence to indicate that chronic exposure to benzene can lead to the development of chronic lymphocytic leukemia or small lymphocytic leukemia.

Q. (BY MR. BROWN) So the answer --

A. Small lymphocytic lymphoma, excuse me.

Q. So the jury and I can understand exactly what is, would it be -- what you are telling me is it's your opinion that benzene is not sufficiently associated with causing CLL,

correct?

A. There is not sufficient evidence to conclude that benzene causes CLL.

Q. You are aware of studies that find excess CLLs among workers exposed to benzene where those studies find a greater than twofold

increased risk at a confidence interval above 95 percent, correct?

A. Not based upon -- certainly not -- not studies that I would rely on with respect to the information, the data, or the relationship with benzene specifically.

Q. All right. You are aware that the Australian Healthwatch study comes to that conclusion, correct?

A. No, I do not.

Q. You are not aware of that?

A. No. As a matter of fact, I brought the latest publication relating to the Australian Healthwatch study that documents or at least brings up to date their current findings, and it did does not comport with that.

Q. All right. So the excess CLLs in the original Healthwatch study in Australia you are saying have vanished?

MR. SCOTT: Object to the form of the question.

A. I'm saying that that study does not in its totality support a conclusion that benzene exposure is associated with the development of CLL.

Q. (BY MR. BROWN) The study as originally published, do you agree that it showed excess CLLs?

A. No, I cannot speak to that as I sit here. My general understanding with respect to the -- the Australian Healthwatch study is an ongoing, progressive study. It has undergone several revisions and modifications over time with multiple different publications that have come out and multiple changes in the conclusions of the authors. The most recent conclusions of the authors is there is insufficient evidence to support a relationship in the study with development of CLL, and in fact there's some --

Q. All right. Let me object to the nonresponsive portions of the answer. Do you have an opinion that -- whether benzene can cause CML?

A. I believe, again, that the

scientific and medical literature does not support a causal relationship between chronic exposure to benzene and the development of CML.

Q. Are you aware of any studies that associate benzene with CML where the risks, relative risk or observed risk, were greater

than twofold at a 95 percent confidence interval?

A. I have not reviewed the literature on CML in many years, so I don't remember the specific studies that provide information related to CML, but I don't believe there are any that meet those criteria and certainly with respect to reliable diagnosis of CML.

Q. Let me object to nonresponsive portions of the answer. Are you aware of any -- well, do you have an opinion as to whether acute lymphocytic leukemia can be caused by exposure to benzene?

A. There are no quantitative epidemiology studies that provide a reliable or significant data set to indicate that chronic exposure to benzene results in the development of ALL.

Q. Let me see if I understand what you

are saying. You are testifying that there are no studies that show an increased risk of ALL among benzene-exposed populations that exceed twofold increased risk at a confidence interval or 95 percent or greater; is that correct?

A. I believe so.

Q. How about non-Hodgkin's lymphoma, do you have an opinion whether benzene can cause non-Hodgkin's lymphoma?

A. There have been studies that have suggested that, but there are no adequate epidemiology studies with respect to data set to reach a conclusion with respect to the relationship between benzene and non-Hodgkin's lymphoma. There are certainly no studies that provide a reliable quantitative indication of a quantitative increased risk associated with benzene exposure in non -- in Hodgkin lymphoma.

Q. Let me object to responsiveness. Would your opinion be, as you sit here today, that benzene does not cause non-Hodgkin's lymphoma?

A. Are we talking Hodgkin lymphoma or non-Hodgkin lymphoma?

Q. My question is non-Hodgkin's

lymphoma.

A. For non-Hodgkin lymphoma, there are -- there's no indication that I believe provides a clear-cut -- there are studies that question there may be a relationship. There are studies that have found no relationship. I

don't believe that as a whole, taken as a whole, the epidemiology literature supports the conclusion that chronic exposure to benzene is associated with a significant or dose-dependent increase in NHL.

Q. All right. Are you saying that in terms of reasonable scientific certainty?

A. Yes.

Q. All right. So you are not just saying in reasonable medical probability; you are saying in terms of reasonable scientific certainty there is no studies?

A. I'm not sure how you distinguish or discriminate between reasonable scientific certainty and reasonable medical probability. Seems to me that those are basically -- if there are legal differences, I don't know what they are.

Q. All right.

A. I think to a reasonable scientific probability, that has not been demonstrated to, certainly within the general scientific community.

Q. Were you aware of the Yin study? Correct?

A. Oh, yes.

Q. I know you have some criticisms of that study, but that study does find that there is an increased risk of non-Hodgkin's lymphoma among benzene-exposed populations, correct?

A. It finds -- that study reported some associations that are not related to dose -- were not dose-dependent and raise very serious questions with respect to whether in fact there is a quantitative association. And the authors of the Yin study and the NCI studies that followed up on that I think reached similar conclusions. It was a provocative finding and suggested a possibility but certainly didn't establish a causal relationship.

Q. Well, are you saying that the study is unreliable?

A. I'm saying that the study has to be taken in the context of the overall literature

and that there are aspects of the study that render an interpretation -- an interpretation that benzene causes NHL. The study is not adequate to say that.

Q. My question was, Are you saying the study is unreliable?

MR. SCOTT: Object to the form of the question.

A. I'm saying there are aspects in the nature of that study that do not allow me as a scientist to reach a conclusion that it demonstrates an increased risk associated with benzene exposure.

Q. (BY MR. BROWN) You would agree that it's generally accepted among scientists and health professionals in your field that sufficient exposure to benzene can cause AML, the type that Mr. McCarty had, correct?

A. It can cause AML. There are aspects of his disease that I don't think necessarily allow me to reach a conclusion that benzene is associated with the development of the specific type of disease that Mr. McCarty had.

Q. Well, let me object to responsiveness. My question is, You

recognize -- or it is generally accepted among scientists and health professionals in your field that sufficient exposure to benzene can cause AML, correct?

A. Yeah, that's correct.

Q. All right. You recognize that

Mr. McCarty was diagnosed by his treating doctor, Dr. -- the cancer specialist, Dr. Jay Schachner, and his doctors at M.D. Anderson with AML, correct?

A. Refractory anemia with excess blasts transforming into AML.

Q. You agree that refractory anemia with excess blasts has been associated with causing benzene in the medical and scientific literature, correct?

A. Some forms of refractory anemia, yes. Others, no.

Q. Do you have any information with regard to the type of refractory anemia that Mr. McCarty had?

A. There's reference in certainly refractory anemia with excess blasts. There's also information in his medical records to suggest that in fact he may have had refractory

anemia with ring sideroblasts, and he certain -- and the records certainly demonstrate that he had prominent ring sideroblasts in his bone marrow.

Q. Let me object to responsiveness. Sir, do you dispute the diagnosis of M.D.

Anderson that found that Mr. McCarty had acute myelogenous leukemia?

A. No, I don't dispute it. It's not clear to me what subtype. It is clear to me that he had refractory anemia with excess blasts that transformed -- probably transformed into AML. That is not -- I don't believe that that distinction is important with respect to the issue of evaluating the relationship with exposure to benzene. Benzene can cause -- with sufficient exposure I believe benzene can cause refractory anemia with excess blasts, and it can cause AML.

Q. Let's go ahead and let him change his tape.

THE VIDEOGRAPHER: The time is approximately 12:54 p.m. and we are now off the record.

(Discussion off the record.)

(Irons Deposition Exhibits 5 and 6 were marked.)

THE VIDEOGRAPHER: The time is approximately 12:56 p.m. and we are back on the record.

Q. (BY MR. BROWN) Let me show you what

I've marked as Exhibit No. 5 to your deposition. Do you recognize Casarett and Doull's Toxicology as a reliable, learned treatise in the field of toxicology?

A. It's a textbook that's respected in the field of toxicology.

Q. Do you utilize that textbook in your practice?

A. I haven't looked at it in quite some time, but I have used it in the past.

Q. All right. This is the sixth edition, and it's -- I want to refer you over to page 402 where I've got the highlighted portion. Is yours highlighted there, sir?

A. There's highlighting, yes.

Q. It says, "Curiously, AML is the dominant leukemia associated with drug or chemical exposure, followed by MDS," myelodysplastic syndrome, and they quote Irons,

1997; is that correct?

A. That's correct.

Q. Is that you?

A. Yes.

Q. And you agree with that statement?

A. Yes.

Q. It says, "The evidence that this represents a continuum of one toxic response is compelling," and again they quote you, Irons, 1997, correct?

A. That's correct.

Q. You agree with that statement?

A. Yes.

Q. Had you reviewed this portion of this textbook prior to today either in terms of peer review or for accuracy?

A. Not in particular, no. I don't think I've ever seen this particular paragraph.

Q. The next statement, it says, "This has also been linked to cytogenic abnormalities, particularly the loss of all or part of chromosomes 5 and 7; is that a correct statement, sir?

A. That's definitely an accurate description of the literature, and I have had

said that before several times.

Q. You still agree with that, correct?

A. I believe that the published literature suggests a high association of secondary leukemia with 5 and 7.

Q. "Remarkably, the frequency of these

deletions in patients who develop myelodysplastic syndrome and/or AML after treatment with acylating or other antineoplastic agents ranges from 67 to 95 percent, depending on the study." Did you know that to be a true statement?

A. I believe that that's very similar to what I've said in the past, and that refers specifically to acylating chemotherapeutic agents.

Q. All right. The next sentence says, "Some of these same changes have been observed in AML patients occupationally exposed to benzene." Do you see that?

A. That's correct.

Q. Do you agree with that statement?

A. Yes, I do.

Q. Next statement says, down there at the highlighted portion, "The relative low

frequency of deletions in chromosomes 5 and 7 in de novo as compared with secondary AML suggest that these --

(The reporter interrupted for clarification.)

Q. Let me just restart the whole

sentence over. It says, "The relatively low frequency of the deletions in chromosomes 5 and 7 in de novo as compared with secondary AML suggests that these cytogenic markers can be useful in discriminating between toxic exposures and other etiologies of this leukemia," and they quote you; is that correct?

A. That's correct.

Q. Do you still agree with that sentence?

A. I think it can be useful. I don't think it's diagnostic. There are other potential causes and etiologies for AML associated with 5 and 7. Certainly the literature currently supports that in fact 5 and 7 can be found with increased frequency in benzene-associated leukemia.

Q. And what you mean here in "useful in discriminating between toxic exposures and other

etiologies of leukemia" means that it could be useful in determining whether or not a toxic substance like benzene is the cause of the AML as opposed to some other cause, correct?

A. It is one of the factors that can be used in ascertaining potential cause.

Q. Do you agree with what they say you said here, sir?

A. Yes.

Q. You don't want to change that at all; is that correct?

A. That's what I just -- I just said.

Q. Are there any medical tests or diagnostic procedures that can help determine whether an AML is caused by exposure to toxic substances such as benzene?

A. Specific procedures, no. The sum total of the characteristics and presentation can be useful in ascertaining the likelihood of one etiology over another.

Q. All right. And one of those specific tests would be a chromosomal analysis, correct?

A. It's one of the tests. It's one of those -- it's one of the procedures that can be

useful in discriminating between one etiology and another.

Q. All right. You would agree that if a person had sufficient exposure to benzene and develops AML and his chromosome analysis indicates a deletion of chromosome 5 or 7 then

such would provide strong evidence that the AML was benzene-related, correct?

A. It would be more probable than not, in my opinion.

Q. Would you even go so far as to say it would be strong evidence that it would be --

A. It would be more probable than not. It would be more likely than not.

Q. All right. Let me ask you this: Have you testified before that if somebody had sufficient exposure to benzene and developed AML and then his chromosomal analysis showed a deletion of chromosome 5 or 7, that that would be strong evidence that the AML was benzene-related?

A. Those would be sufficient criteria for me to reach a conclusion that it is more likely than not that that AML was associated with exposure to benzene.

Q. Do you remember testifying in a case called Betsy Lakie versus Smith Kline Beecham back in April 12, 1996, sir?

A. I don't remember testifying in that case. I think I gave a deposition in that case. I don't recall testifying.

Q. Testifying in a deposition, took place in Denver, Colorado?

A. I believe so.

Q. If you'll read along with me. It says here, "If you saw an individual with one of these deletions you've just described with AML and there was evidence of benzene exposure, would your opinion, given the simple parameters -- given those simple parameters, be more probably than not that it was caused by the benzene exposure?"

I think your answer, if I read it correctly, is, "If there was evidence of a significant chronic benzene exposure, and you have AML that with respect to its presentation and criteria fits the category of a secondary AML, the presence of a clonal lesion of 5q or 5- would provide fairly strong evidence that there was a potential relationship." Did I read that

correctly?

MR. SCOTT: Object to the form of the question.

A. Yes, you read this correctly.

Q. (BY MR. BROWN) Do you agree with your statement there that you made in that

deposition back in 1996?

A. Yes, and that's what I've just said.

Q. Let me show you what we've marked as Exhibit 6 to your deposition. I will represent to you that those are certain medical records of Oliver McCarty that I pulled. I know I've seen some of those in your deposition or your case notebook here. Do you recognize the document that we've marked as part of Exhibit 6 to your deposition from Mr. McCarty's medical records?

A. Yes.

Q. First page of that is Dr. Jay Schachner's records. Dr. Jay Schachner is a cancer specialist, oncologist. You are aware of that, correct?

A. I believe so, yes.

Q. He states on March 26th of '03 -- he describes the problems or his diagnoses of Mr. McCarty as, No. 1, meylodysplastic syndrome.

You've told us that could be related to benzene, correct?

A. Yes.

Q. The next one was preleukemia associated with pancytopenia. You told us pancytopenia is another condition of the blood

that can be related to benzene, correct?

A. Yes.

Q. If you turn to the next page, it's the April 3rd entry for Oliver McCarty's clinical note of Dr. Jay Schachner. It states, "Problems: Refractory anemia with excess blasts and transformation to AML." Do you see that?

A. Yes.

Q. He's saying that his myelodysplastic syndrome has transformed into AML, correct?

A. He says it is transforming into AML. That is -- that is technically a diagnosis of RAEB-2, refractory anemia with excess blasts 2, which is associated with a numerical increase in the blast cell count in excess of 9 percent.

Q. Well, you said --

A. So that's a --

Q. Okay. I don't mean to interrupt you. I'm sorry.

A. However, that's inconsistent with what he describes, which has been one of -- which I don't think is significant with respect to ultimately diagnosis of his disease, but it's -- the description is inconsistent with the laboratory results.

Q. Well, let me ask you, do you dispute Dr. Jay Schachner's diagnosis, the treating physician for Mr. McCarty, who is a cancer specialist? Do you dispute his diagnosis of refractory anemia with excess blasts and transformation to AML?

MR. SCOTT: Object to form of the question.

A. No. I believe -- I don't dispute his diagnosis. I believe there are inconsistencies between his description and the laboratory, is all.

Q. (BY MR. BROWN) Let me object to the nonresponsive portions of the answer after "no." Next document is a page No. 49. It is the Chromosome Analysis, Bone Marrow report. Do you see that?

A. Yes.

Q. And the findings indicate that there

is deletion of chromosome 5, correct?

A. Yes.

Q. And also in the arm, 5q 31 and q 35, correct?

A. Yes. That's the specific region that's deleted. It's usually an interstitial

deletion.

Q. That is consistent with the publication and the theories being put forth in the toxicology book that we saw as Exhibit 5 and your prior testimony that this is evidence that strong -- or fairly strong evidence that his acute myelogenous leukemia is related to exposure to benzene, correct?

A. This particular finding is consistent with an AML associated with chronic exposure to benzene. There are other findings that are associated with his leukemia that are inconsistent. So this is one finding that is consistent. There are others that are not.

Q. Let me object to the nonresponsive portions of the answer. What diagnosis and what condition did Mr. McCarty have, in your opinion, and that you say as someone who has never seen Mr. McCarty or treated him, that is inconsistent

with exposure to benzene?

MR. SCOTT: Object to the form of the question.

Q. (BY MR. BROWN) As you say.

MR. SCOTT: Same objection.

A. Refractory anemia that's associated

with ring sideroblasts has to my knowledge not been associated with exposure to benzene, and in fact that is described in his records. So within the context of -- or within the overall data that we have available, there are features of his disease that are consistent or would be consistent with a benzene-induced leukemia, if in fact he had sufficient exposure to benzene, and there are others that are not consistent or have not been reported associated with benzene.

Q. (BY MR. BROWN) Well, you know he ends up having AML with a loss of chromosome 5, correct?

A. I believe -- I don't -- I believe that he developed refractory anemia with excess blasts and that refractory anemia with excess blasts are RAEB-2. The distinction between RAEB-2 and acute myelogenous, myeloid leukemia is moot. These are basically the same disease

process. So the point at which he develops one and no longer has the other is not clinically or scientifically important with respect to the severity of his disease, his prognosis, or potential causation.

I believe that there are some --

there are some discrepancies between the report and the medical record that make it difficult for me to differentiate on the basis of the reports precisely what disease he had at what time. I don't think these are significant with respect to my conclusions, but there are some discrepancies.

Q. All right. In your opinion, would you conclude that if Mr. McCarty had benzene exposures like his coworkers and that he has testified or sworn that he had due to the type of disease, leukemia he had, AML and the deletion of the chromosome 5, that his exposures would have been at least a contributing factor to his disease?

MR. SCOTT: Object to the form of the question.

A. My opinion is based upon the -- both an analysis of the potential exposure

Mr. McCarty had and the presentation of his disease. If in fact he had sufficient exposure to reach a conclusion of an association based on the literature, I would be quite surprised at the presence -- in fact I would find it remarkable that in fact his pathology report

includes significant percent of ring sideroblast. That is a finding that has not been reported in the literature, to my knowledge, associated with an AML or an MDS associated with benzene exposure. So my opinion with respect to the likelihood that Mr. McCarty's disease is associated with exposure to benzene is based on the basis of the quantitative exposure assessment and my review of his overall pathology report.

Q. (BY MR. BROWN) Let me object to responsiveness. Can you point to me one diagnostic study or exam that says that his diagnosis, his medical diagnosis by any of his doctors, was refractory anemia with ring sideroblasts?

A. Whether or not it was refractory anemia with ring sideroblasts is dependent on a specific numerical count, which is not provided.

What there is, is a reference to the presence of multiple ring sideroblasts. So I am saying that based upon the assessment -- my assessment of these reports, I see features of his disease that would be consistent with exposure to benzene, given evidence of sufficient exposure,

and there are features that are not consistent, and this is one of them. The presence of a chromosome aberration involving 5q 31 clonal aberration would be consistent with an AML associated with exposure to benzene. The presence of ring sideroblasts is not.

Q. Let me object to responsiveness. My question to you, sir, is, can you show me and a the jury, pull out one medical record of all these significant records that you've already pulled and went through that says Mr. McCarty had a diagnosis of refractory anemia with ring sideroblasts?

A. No, I can't. Refractory anemia with ring sideroblasts would in -- if it progressed in course, develop into refractory anemia with excess blasts. There is no way for me to conclude, in the absence of a quantitative numerical count of ring sideroblasts, to

conclude that in fact that was his disease. But the presence of ring sideroblasts I do find is a significant factor.

Q. Let me object to the responsiveness as to everything after, "No, I cannot." The Bone Marrow Morphology, which is the next

document, what was the diagnosis on that, sir?

A. Myelodysplastic syndromes -- okay.

Class C. "Evolving acute myeloid anemia cannot be categorically excluded."

Q. That's the comment. What's the diagnosis right above it?

A. "Finding consistent with refractory anemia with excess blasts (17%) (RAEB-2 by WHO scheme)."

Q. Do you have any reason to dispute that diagnosis?

A. No.

Q. The next document is a document from M.D. Anderson that's dated April 17, 2003, with Dr. Charles A. Koller being the physician. Do you know him?

A. No.

Q. Do you know anything about the reputation for M.D. Anderson?

A. Certainly.

Q. What is it?

A. M.D. Anderson is a prominent, world-class cancer center.

Q. In your opinion, are there any cancer centers that are more prominent or have

better reputations in the United States than M.D. Anderson?

A. I think there are plenty of competent, reliable cancer centers with very good and excellent reputations. M.D. Anderson is in the top tier of cancer centers in the United States.

Q. Dr. Koller's impression and diagnosis of Mr. McCarty when he went to see him, right there, No. 1, acute myelogenous leukemia. Do you see that?

A. I'm sorry; I'm trying to follow with you.

Q. It says "impression" right down here.

A. Are we on the same page?

Q. Should be page 73. Let me see if I can find that for you.

A. My glasses need replacement.

Q. Dr. Koller's impression was that Mr. McCarty had acute myelogenous leukemia. Do you see that?

A. Um-hum.

Q. And in fact he told him that he had acute myelogenous leukemia. Do you see that?

A. Um-hum.

Q. Do you have any reason to dispute Dr. Koller at M.D. Anderson's diagnosis that Mr. McCarty had acute myelogenous leukemia?

A. No. If you notice the date on this, it's 4/17. The initial date is earlier in the month. There is no inconsistency between a diagnosis of RAEB-2 at the beginning of the month, earlier in the month, and a diagnosis of acute myelogenous leukemia later in the month. It's simply a function of the numerical blast count. I don't see the blast count here, but I'm fully prepared to accept that Dr. Koller's impression of his disease is accurate.

Q. Let me object to responsiveness as to everything after his answer "no." I've included into Exhibit 6 the Koller diagnosis, medical. The next page is from M.D. Anderson records, page 92. This is the physician,

Dr. Alessandra Ferrajoli. Do you see that?

A. I'm sorry. I'm not following.

Q. Right there.

A. Okay.

Q. Do you know Dr. Ferrajoli?

A. Not personally, no.

Q. It says reason for admission, do you see up at the top, treatment of AML, recent diagnosis of AML?

A. Um-hum.

Q. Do you see that, sir?

A. Yes.

Q. Do you have any reason to dispute Dr. Ferrajoli's diagnosis of AML in Mr. McCarty?

A. No.

Q. All right. If you go to the next page, the discharge diagnosis was refractory acute myelogenous leukemia. Do you see that?

A. Um-hum.

Q. Is that a yes?

A. Yes.

Q. If you will turn to the next page, I think it's page 102 of Dr. Schachner's records, it's the death summary. I noticed you had a copy of the death summary in your records?

A. Yes.

Q. Indicates that Mr. McCarty unfortunately deteriorated and expired with his family at his bedside on July 25th of '03. Do you see that?

A. Yes.

Q. And the final discharge diagnosis was acute myelogenous leukemia; do you see that?

A. Yes.

Q. And then the last record is the death certificate for Mr. McCarty, who at age 70 dies of acute myelogenous leukemia as recorded by Dr. Schachner on his death certificate. Do you see that?

A. Yes.

Q. Do you have any reason to dispute Mr. McCarty's diagnosis of AML?

A. No.

Q. Do you have any reason to dispute that Mr. McCarty's AML caused his death?

A. No.

Q. You are not a medical doctor. You've told us that, correct?

A. That's correct.

Q. You are not -- by law you cannot

practice medicine, correct?

A. That's correct.

Q. You have no medical license, correct?

A. That's correct.

Q. You've never attended medical school

and went through the rigorous training and courses that medical students who get their M.D. go through, correct?

A. I've attended medical school, but I did not obtain an M.D.

Q. You didn't take all the courses that the M.D.s have to take to get there or you would have gotten your M.D., correct?

A. That's correct.

Q. And because you don't have a medical license, you cannot render medical pathological diagnoses to a patient, correct?

A. That's correct. That's correct.

Q. And you have no reason, as you've told us, to dispute the diagnosis of M.D. Anderson or Dr. Jay Schachner with regard to the disease Mr. Schachner (sic) suffered and died from, correct?

A. I have no reason to dispute the

diagnosis rendered by M.D. Anderson.

Q. So I take that you would dispute the diagnosis rendered by Dr. Jay Schachner for some reason; is that what you are saying?

A. I simply am saying that there are descriptions in his report that don't comport

with the pathology report, but I believe that his conclusion with respect to refractory anemia with excess blasts transforming into AML was correct.

Q. All right. So any differences you have in what his records would have said from one day to the next as he had his hands on and treating Mr. McCarty you believe are insignificant; is that right?

A. With respect to the diagnosis of his disease, I believe they are insignificant.

Q. All right. Is there any significance in the differences and the records that you say exist in your opinions?

A. With respect to Mr. McCarty, no. They would be very significant in other cases. But in this particular case, based on all the information I have, no.

Q. And so we know that Mr. McCarty had

a disease process, meylodysplastic syndrome that transformed into AML, and he has a loss or deletion of chromosome 5. If he was exposed to benzene like his coworkers said he was, would it be your opinion that that exposure to benzene was a contributing factor to his development of

AML and his death?

A. I can't say that based on the information that I reviewed in this case. There are other issues that are important in reaching the conclusion, and those are not -- those are not enough to reach that conclusion.

Q. Can you rule out benzene as a cause of Mr. McCarty's AML and death?

A. No, I cannot.

Q. I don't have any further questions at this time. I will have some after we take our lunch break, but why don't we take our lunch break now.

MR. SCOTT: I was going to say if you are done, I can get done in 20 minutes, but if not, let's come back.

THE VIDEOGRAPHER: The time is 1:23 p.m. and we are now off the record.

(A lunch break was taken.)

THE VIDEOGRAPHER: The time is approximately 2:30 p.m. and we are back on the record.

Q. (BY MR. BROWN) All right. Sir, we are back on the record after lunch. Can you show me the reference in the medical records to

the ring sideroblasts that you were referring to earlier?

A. Yes.

Q. All right. This is from the bone marrow morphology that's dated, report date of March 8, 2003. All right. Is there any other reference to ring sideroblasts in your medical records that you reviewed or that you found and thought were significant?

A. I believe that's it.

Q. If we could go back and talk about that. That's also one of the records that we put in Exhibit 6, I believe. We'll look at yours. Page 044 in Exhibit 1 of your notebook.

A. 043, I believe.

Q. Okay. That's what I thought. Page 43 on my records in Exhibit 6, and it's also page 43 in your records, and you've also written some handwritten notes out beside?

A. Yes.

Q. What do those notes say?

A. It says 15 percent, question mark, RARS, R-A-R-S, arrow, RAEB.

Q. Where do you get the 15 percent refractory anemia with ring sideroblasts and the

RAEB?

A. RAEB is what he's diagnosed as.

Q. All right.

A. I'm asking the question, what is the percentage of ring sideroblasts, because it doesn't say. 15 percent is the arbitrary number that defines refractory anemia with ring sideroblasts.

Q. And people who diagnose -- pathologists and people who read these bone marrow morphologies, they realize that, don't they?

A. I don't know. I would assume they do, but most reports that I've seen or would generate would specifically list the number of ring sideroblasts if in fact you noted there were multiple ring sideroblasts.

Q. Well, anyway, this is the hemo -- hemic (phonetic) pathologist, what is that?

A. Hematopathologist is a pathologist who specialized in the diagnosis of hematopoietic diseases.

Q. Are you a hematopoologist (phonetic) -- let me see if I can back up and say it. Are you a hematopathologist?

A. No.

Q. And we've got Dr. Joon S. Lee, M.D., who is reading the bone marrow morphology. How do they do that? Do they put it under a microscope and read a slide?

A. They put in under a microscope and use an iron stain, and they would look at the smear and look at it in a nonquantitative way, and then they would also typically enumerate the number of ring sideroblasts.

Q. In any event, even though the iron stain reflects ring sideroblasts, the diagnosis that they come up with is refractory anemia with excess blasts, 17 percent, correct?

A. Yes. Those are not inconsistent.

Q. Well, what you are saying is that you have a problem; you don't know if there is a ring -- refractory anemia with ring sideroblasts, and that because of the ring

sideroblasts, that prevents you from saying that Mr. McCarty's AML with a loss of his chromosome 5 was a benzene-related AML; is that correct?

A. No, I don't think that's a correct characterization of my testimony. My opinion is that the presence of ring sideroblasts is not a

finding that's reported in previous described cases of benzene-associated leukemia or myelodysplastic syndrome in the literature. A diagnosis of refractory anemia with ring sideroblasts requires that there be 15 percent ring sideroblasts, which is a significant number, a large number. If there are less, it's still a remarkable finding to me that it is present. So in the absence of any other information, my opinion, based on this, is this is not -- this -- this specific finding is not consistent with literature associated with hematopoietic disease associated with benzene.

Q. When you say this specific finding, you are talking about under iron stain where it says ring sideroblasts, correct?

A. Yes.

Q. That's the specific finding you are talking about?

A. Yes.

Q. And even though they found it -- there is a diagnosis for refractory anemia with ring sideroblasts, correct?

A. Yes, it requires 15 percent of ring sideroblasts.

Q. And this pathologist who read this says -- they don't put in the diagnosis refractory anemia with ring sideroblasts; they put in refractory anemia with excess blasts, correct?

A. Yes, and there's a perfectly logical reason for that, and that's because RAEB-2 supersedes refractory anemia. So the diagnosis of RAEB-2 based on 17 percent blasts obviates the initial diagnosis.

Q. Do you dispute the diagnosis that Dr. Joon Lee came up with there?

A. No, I don't.

Q. Let me just ask it this way. If it weren't for this one mention of sideroblasts in this one record, would you have any hesitancy in saying that Mr. McCarty's benzene -- I mean, acute myelogenous leukemia with his loss of chromosome 5 is related to benzene exposure?

A. Yes. There is no quantitative evidence that I've seen in the record to indicate that he had substantial benzene exposure, and the latency between his last opportunity for exposure and the presentation of his disease is 35 years, which is way beyond the

latency that is reported in the quantitative epidemiology literature.

Q. Let me object to the nonresponsiveness of the answer. If it -- the loss of chromosome 5 and the fact that he had AML, that is indicative to you as strong evidence that it was a toxic exposure that caused his leukemia, correct?

MR. SCOTT: Object to the form of the question.

A. It is not -- it is not in and of itself diagnostic of a secondary leukemia. There are many acute myelogenous leukemias that present and are characterized as de novo that present with minus 5. There is an increased frequency of minus 5 and/or minus 7 in AMLs that develop secondary, clearly associated with exposure to alkylating chemotherapy agents and most probably associated with benzene.

That in and of itself does not provide a diagnosis -- evidence all by itself that an individual's AML developed as a consequence of benzene exposure. It's one of the factors that has to be taken into account, along with quantitative exposure and/or exposure

assessment, issues such as latency, and the relationship and temporal relationship between the presentation of disease and the cessation of exposure, as well as other characteristics in the presentation and diagnosis of the disease.

Q. (BY MR. BROWN) Let me object again to the nonresponsive portion of the answer. My question was simply, that is strong evidence that the AML is toxic-induced AML and it's not just -- in terms of probability you even report yourself that it's between 76 and somewhere 90 percent of the time that when there is an AML that has those chromosomal damage, the loss or deletion of 5 or 7, that you can trace it back to a toxic substance, correct?

MR. SCOTT: Object to form of the question.

A. No, and that's not my testimony. The fact of the matter is approximately

12 percent -- 12 to 15 percent of AMLs presented in this country that are described as de novo present with clonal aberrations involving 5 and/or 7. If you have an AML that presents secondary to previous chemotherapy for other neoplastic diseases, the frequency of

aberrations involving 5 or 7 goes much higher and is present at levels between two-thirds and 90-some-odd percent. That's the frequency of this lesion in cases where you've got documented exposure to alkylating agents at concentrations that produce very frank bone marrow damage and go on to develop MDS or AML. That's separate and distinct from the incidence of this chromosome aberration in the general population and in AMLs that are de novo.

Q. (BY MR. BROWN) You say here even in your studies, in your prior depositions and in the toxicology treatise that we've got here marked as Exhibit No. 5 to your deposition, that it -- not only does it occur with people who have had chemotherapy with alkylating agents, but it's -- these same changes have been observed in AML with patients occupationally exposed to benzene. That's a true statement;

You've told me that, correct?

A. That's correct.

Q. Is there any incidence or number difference in the frequency that you see people with occupational benzene exposures developing AMLs with loss of 5 or 7 as opposed to people

developing AMLs secondary to chemotherapy with a loss of 5 or 7?

A. To the extent that we have literature describing cytogenetic aberrations associated with exposure to benzene, and this goes back to case-controlled studies that have been published in the 1980s and 1990s, the frequency of aberrations is less in those associated with benzene than in the chemotherapeutic literature, but you have to -- you have to keep in mind that the incidence -- individuals who are treated with chemotherapy, the exposures are clearly demonstrable. They are quantitative because we know what the dose is the individuals have received. We can define precisely what the natural history of that disease is. And the frequency of aberrations involving 5 or 7 in those individuals is extremely high, and studies have repeatedly

demonstrated that to be the case.

That doesn't mean that everyone who present with an AML has had previous chemotherapy or that they have been exposed to benzene that's associated with the development of their disease. There is a difference between

the frequency, the relative frequency of these aberrations in patients who have received chemotherapy and patients who have not. And that's the significant difference.

This aberration is more frequently found in individuals who develop AML secondary to exposure to chemotherapeutic agents, and the evidence associated with benzene also suggests that that relationship to some extent holds for those individuals as well. But there are many people who present with AML that have involvement in chromosomes 5 and/or 7 for which there is no demonstrable evidence that they had any significant exposure to benzene or chemotherapeutic agents.

Q. Let me object to responsiveness. That was my question is, what is the difference in the number of percentage of people? You said it's less. Less by how much?

A. We -- I can't put a quantitative number on it because we have much more compelling quantitative data on patients receiving chemotherapy than we do patients who have been previously chronically exposed to high concentrations of benzene.

Q. If you can't put a quantitative number on it, then you really can't say that it's less, can you?

A. I think it's more probable than not that AMLs associated or that have 5 and/or 7 are among those leukemias that benzene -- chronic exposure to benzene is associated with. I can't tell you what number less than those that are associated with alkylating chemotherapy is. There is no basis for basically rendering an opinion on a negative.

Q. You can't tell me the number, correct, for benzene --

A. I can't -- I can't -- I can't render an opinion on a negative finding in general. That's not something scientists can do.

Q. Well, it's not a negative finding. You know, here you are rendering it for people with chemotherapeutic agents who develop AML and

the loss of 5 or 7. What I'm saying is you can't tell me what the number is below that for benzene because you don't know, and are there any studies that support your opinion that it's less?

A. Yes.

MR. SCOTT: Object to the form of the question.

A. Yes, certainly.

Q. (BY MR. BROWN) What studies?

A. The same studies that support my opinion that in fact 5 and 7 are increased in frequency in cases of AML associated with benzene exposure. I have repeatedly testified to that, to those studies. I published on those studies. It is my opinion here today, and it was my opinion ten years ago, that the frequency of clonal aberrations involving 5 or 7 in AMLs associated with previous benzene exposure is more probably than not increased relative to the frequency in the general population. I can't say that it's the same frequency as associated with chemotherapeutics. We don't know that.

Q. That's what -- that's all I'm asking you is, I thought you told me earlier you

thought it was less, but you can't put a quantitative number on it?

A. I'm saying exactly the same thing. I cannot provide you with a quantitative estimate of what that frequency is based upon the world literature.

Q. All right. And that's my next question. Is there any literature that would support your opinion that it's less in terms of quantitative -- a quantitative number?

A. The actual frequency in studies where in fact the AML has been associated with previous benzene exposure do not rise to the same number that the chemotherapeutic -- alkylating chemotherapeutic literature does. That's a fact.

Q. Is that just what your assumption is, or do you have hard scientific data or studies that back you up when you say that?

A. I have the existing case-control quantitative epidemiologic literature associated with --

(The reporter interrupted.)

A. Associated with benzene.

MR. SCOTT: Cytogenetic aberrations

and benzene. That is a good point about slowing down if we can. Some of this is a little dense.

Q. (BY MR. BROWN) Well, you are saying you've got those studies, and those studies show that when there is exposure to benzene, it causes a loss of the chromosome 5 or 7, that it

would be with less regularity or frequency than chemotherapy-induced damage to 5 or 7. What in those studies is the quantitative number that they use related to benzene?

A. You know, as I sit here right now, I can't give you a number because I would not -- I don't believe that those studies allow me to provide or to render an opinion on the precise number. I've testified and I've repeatedly said in the literature that the studies that exist suggest there is an increased frequency of those aberrations in AML associated with benzene. I cannot tell you exactly what the number is.

It's been my opinion and it's certainly been my hypothesis for many years that the alkylating chemotherapeutic literature informs us with respect to plausible mechanisms that may be involved in the development of AML from benzene, and it would be consistent that

you would see an increase. I've said that many times. That's still my opinion today.

Q. Okay. Let me object to responsiveness. You say you can't say an exact number with regard to benzene-induced damage to 5 or 7, but is there a study that has a number

in it like these studies with chemotherapeutic agents, you know, saying that you see that with 75 to 90 percent of the cases?

A. No.

Q. Okay.

A. There are studies that show an increased frequency. The problem with the existing studies is where you've got very reliable quantitative cytogenetic analysis of clonal lesions, you have far less reliable exposure data related to the specificity of the exposure to benzene. In the studies where you've got much higher -- where you have a much more reliable quantitative estimate of benzene exposure, you have far less reliable, if totally unreliable, cytogenetic data.

Q. You have not reviewed any of the slides or the pathology for Mr. McCarty, correct?

A. No, I have not.

Q. Have you even requested it?

A. No.

MR. SCOTT: Darren, let me say, it's not particularly important here, but we've been trying to get pathology from M.D. Anderson for

some time, and it took us a period of time to get an agreement with you all about what we could ask for and how. And at one point Anderson told us they found some blocks, and now they can't seem to find them, but we have been continuing our efforts to get that material.

MR. BROWN: Object to sidebar.

MR. BARNWELL: We have had an agreement in place for several months that would allow you guys chain of custody.

MR. SCOTT: Absolutely, and I think there were some posttreatment slides that we got that were forwarded to Dr. Nadelson.

MR. BROWN: Same objection.

MR. SCOTT: I don't mean it for the record.

Q. (BY MR. BROWN) Well, I mean, because it's not insignificant. The significance is you haven't even asked for them because you didn't

plan on using those, correct?

A. No, that's not true. Actually I have been informed by Mr. Scott that they didn't have them available, which is why I haven't asked for them. At this particular point in time I would be happy to look at the smears if

in fact they were presented to me. I think that it would be informative for me to look at them, but I don't think that at this point it's going -- that those slides are likely to change my opinion based upon the reports and the information that I have.

Q. But bottom line is you never asked for them and you've been working on this case since February, correct?

A. I have received the material in this case over the course of the last several months. I agreed to render an opinion on this case in February. I have not been working consistently on this case since February. I've been working on this case for approximately the last month.

Q. Let me object to responsiveness. You've been hired in this case since February, and even up to today you've never asked for the slides that you are referring to correct?

A. That's correct.

Q. Is it your opinion, sir, that in order for there to be an AML that would be benzene-related that there would always be a loss or deletion of chromosome 5 or 7?

A. No.

Q. That doesn't have to be necessary; you don't have to see a loss or deletion in 5 or 7 in order for somebody to say this benzene -- this AML is benzene-related, correct?

A. That's correct. That's never been my opinion.

Q. Likewise a person doesn't have to have a deletion of both 5 and 7 before one can opine that his AML is benzene-related, correct?

A. I don't think we have enough information to conclude that. I don't think we have enough information to render a -- I don't have enough information to render an informed opinion on that.

Q. Well, you have testified in the past that if you have a loss of 5 or if you have a loss of 7, that could be a benzene-related leukemia, correct?

A. Based upon the available data that

we have, the literature that's available, that's correct.

Q. All right. Are there any other chromosomes whose loss or deletion has been associated with a toxic exposure?

A. There are other -- there are other

abnormalities that are found with equal frequency in both de novo and in cases for which there's documented exposure. There are also recurring abnormalities that have been variously hypothesized to be associated with exposure to alkylating agents. There are specific translocations that have been associated with exposure to a specific class of alkylating -- actually, classic chemotherapeutic agents known as topoisomerase inhibitors.

Q. Well, can you tell me in terms of loss or deletion of other chromosomes besides 5 and 7, if any, that would be associated with potential benzene-induced AML?

A. I wouldn't be at all surprised to find a case of documented benzene exposure in the development of an AML that involves trisomy 8, but trisomy 8 occurs with equal or greater frequency in spontaneous AMLs as well, so it

doesn't provide any kind of distinguishing feature in evaluating the disease and potential causation by benzene.

Q. How about chromosome 11?

A. I don't believe that the data on chromosome 11 is sufficient to reach a

conclusion associated with exposure to benzene.

Q. Have you seen that hypothesized?

A. I've seen -- I've seen that hypothesized. I've seen many hypotheses with respect to cytogenetic aberrations in benzene, including my own, but I don't believe that the quantitative literature supports that conclusion.

Q. How about the loss of the Y chromosome?

A. No. Minus Y is extremely nonspecific. It can be associated with a clonal lesion. It can be associated with age. It's often found in AMLs associated with elderly individuals, and the question is always what happened first: the development of AML or the loss of -- incidental loss of chromosome Y. It can be indicative of a clonal lesion in general, which would be any neoplastic condition, or it

can simply be a function of age and not related to disease.

Q. You talked a little bit earlier -- talked a lot earlier about the quantitative assessment was provided to you by Mr. Plisko or Mark Plisko. Are you familiar with the

scientific methodology that was used by Mr. Plisko in his alleged exposure assessment that he provided to you?

A. In terms of the specific mathematical model that he used, no.

THE VIDEOGRAPHER: Time is approximately 2:55 p.m. and we are now off the record.

The time is approximately 2:57 p.m. and we are back on the record.

Q. (BY MR. BROWN) Since you haven't been a doctor -- or since you are not a medical doctor, you've never had to tell a patient that he has cancer or leukemia, correct?

A. That's not true. I have.

Q. You've been the one to tell a person he has cancer or leukemia even though you were not his doctor?

A. Yes.

Q. Difficult thing to do, isn't it?

A. Yes.

Q. Had to look into the eyes of another human being and give him the bad news that they had cancer and that if it's a cancer like leukemia, they are not going to be around long,

correct?

MR. SCOTT: Object to the form of the question.

A. It's very possible.

Q. (BY MR. BROWN) Do you know what a person goes through when they have a terrible disease like acute myelogenous leukemia?

A. Sir, my laboratory in Shanghai routinely diagnoses leukemias and lymphomas on a daily basis and provides reports and diagnoses that clarify and/or confirm these diseases in individuals routinely. I'm aware and I understand the nature and the severity of and the seriousness of these diseases.

Q. Well, let me object to responsiveness. I understand that your lab does diagnoses, clinic -- I mean research diagnoses of individuals, but you have never had to treat anybody with cancer?

A. No, sir.

Q. Have you ever had a family member who has had cancer, died of cancer?

A. Yes.

Q. And you know exactly what it is I'm talking about, how difficult of a process it can

be for a family to have to sit through the months of pain and grief that they -- are associated with somebody who is dying of a cancer like leukemia, correct?

A. Absolutely.

MR. SCOTT: Object to the form of the question.

Q. (BY MR. BROWN) And dying of leukemia is not an easy or preferred way to go, is it?

A. No. It wouldn't be my preferred way.

Q. Often AML people just bleed uncontrollably, correct?

A. That's one of the adverse consequences that often occurs in AMLs.

Q. They are often delirious and have pain, correct?

A. That's correct.

Q. And they often end up dying of

sepsis, correct?

A. Sepsis is another adverse complication associated with AML.

Q. What is sepsis?

A. Widespread infection, systemic infection.

Q. And systemic infection of all of their organs, correct?

A. Often, yes.

Q. It's painful, isn't it?

A. Yes, it can be.

Q. You saw his death certificate where the immediate cause of death after suffering with AML -- Mr. McCarty finally succumbed to that disease and the sepsis that resulted from it, correct?

A. Sepsis -- I believe sepsis was the precipitating cause of death.

Q. You've been consulting as an expert for oil companies and chemical companies in litigation involving benzene for, I think we established earlier, about 16 years or so, correct?

A. Yes.

Q. Over those years you've been

involved with benzene issues, legislation, studies, and that type of thing even longer than 16 years, correct?

A. I've been studying benzene for the better part of 30 years.

Q. Then you are, I would assume, aware

that benzene was reported to cause fatal diseases of the blood and blood-forming organs as far back as the 1800s, correct?

A. There were reports of bone marrow failure and toxicity associated with benzene back at least as far as the late 1800s.

Q. I assume that you be aware that benzene was reported being associated with and causing leukemia as far back as 1928, correct?

A. There were hypotheses and opinions, observations made by individuals as far back as the -- certainly the late 1920s, early 1930s.

Q. All right. And to the extent that those hypotheses were that benzene can cause leukemia, they turned out to be correct, correct?

A. To the -- yes, with the caveat that the -- what was considered and/or understood to be leukemia and/or bone marrow failure at that

time was not what we now consider to be those diseases. There's been a lot of changes over the last 100 years, the last 80 years with respect to our knowledge of both the disease and benzene.

Q. I mean, if in 1928 it's being

reported that benzene causes leukemia, then those who have access to that report would know that benzene can cause fatal diseases, correct?

MR. SCOTT: Object to form of the question.

A. Isolated reports don't provide us -- isolated individual case reports don't provide the scientific community with a -- with proof that in fact there is a causal association between any given etiology and a given disease. In the case of the reports that are usually -- the cases that were reported in France in 1920 and 1929, there is really actually no clear-cut evidence that those individuals were actually exposed solely to benzene. In fact, there's evidence to suggest they were exposed to alkylating radiation as well as benzene and other toxic chemicals.

So the fact that we have a given

case report or group of case reports does not give us a quantitative evidence that there is a causal relationship between a given etiology and a given disease. That requires quantitative epidemiology.

Q. (BY MR. BROWN) I object to

responsiveness. You are aware that by 1948 the American Petroleum Institute was reporting that benzene could cause fatal illnesses and was even reporting at that time that there were reasonably well documented instances of the development of leukemia from chronic exposure to benzene --

MR. SCOTT: Object to form of the question.

Q. (BY MR. BROWN) -- correct?

A. My recollection of the reports that I've seen and the statements that I've seen by American Petroleum Institute or their representatives raise concern over the possibility and the potential of benzene to cause blood diseases.

Q. Well, I guess let me object again to the responsiveness. My question was, Were you aware that in 1948 API, American Petroleum

Institute, in its toxicological review for benzene was reporting at that time that there were reasonably well documented instances of the development of leukemia associated with exposure -- chronic exposure to benzene?

A. I have -- yes, that's basically what

I've said.

Q. And what is the American Petroleum Institute?

A. American Petroleum Institute is a trade organization that membership includes petroleum companies.

Q. All right. Companies like Mobil and Exxon and Shell and Gulf, Chevron -- all those companies were members of the American Petroleum Institute back in the 1940s; were you aware of that?

A. I believe so.

Q. I'm showing you the API toxicological review for benzene, 1948. You've seen that document before, haven't you, sir?

A. Yes, I have.

Q. Numerous times; would that be correct?

A. Yes.

Q. Did you know Philip Drinker?

A. No, not personally.

Q. You know him to be a reputable professor of the school of medicine at Harvard University back at that time that that report was written, correct?

A. I believe so, yes.

Q. And if a doctor with the reputation of Philip Drinker is reporting in 1948 that there are reasonably well documented instances of the development of leukemia from chronic exposure to benzene, he's not just guessing that there might be a connection; he is making a statement that there is some legitimate concern among the members of that profession and there should be of the American Petroleum Institute that benzene can cause leukemia; isn't that right?

MR. SCOTT: Object to form of the question.

A. I can't testify to that. I think it's obvious from Professor Drinker's statement that he thought there was legitimate concern. I can't say that on the basis of the opinion or statement of a single professor that in fact

that's the case. That's what in fact quantitative epidemiology and biomedical sciences is all about, proving a relationship that is hypothesized through observation. Professor Drinker made some very salient and significant observations and expressed his

concern about exposure to benzene, which was extremely well-founded. That doesn't on the basis of his opinion make it so.

Q. (BY MR. BROWN) Let me object to responsiveness. The fact where he reports there are reasonably well-documented instances of development of benzene -- development of leukemia from chronic exposure to benzene, that would have been information that should have put the companies on notice that benzene could result in a cancerous disease like leukemia?

MR. SCOTT: Object to form of the question.

Q. (BY MR. BROWN) Don't you agree, sir?

MR. SCOTT: Object to form of the question.

A. I think that that's information that should have and did raise concern on the part of the scientific and medical communities and the

industry alike in terms of understanding and studying the potential toxicity of benzene.

Q. (BY MR. BROWN) All right.

Additionally, he reports in this API document that due to individual susceptibilities, inasmuch as the body develops no tolerance to

benzene and as there is wide variation in individual susceptibility, it is generally considered that the only absolute safe concentration from benzene is zero. Do you agree with that statement, sir?

A. That's what he said. In doing so, he also -- he goes on to say that a limit of 50 ppm or less is strongly recommended. So based upon our ability and our understanding of -- our ability to measure benzene, I think Dr. Drinker was roughly equating zero or nothing, which is really toxicologically not a cogent concept, to levels at or below 50 parts per million.

Q. Let me object to the responsiveness. My question was, Do you agree that because there's variation in individual susceptibility to exposures to chemicals such as benzene, that the only absolutely safe level of exposure for everybody would be zero?

A. No, I think that's -- that's not been demonstrated in the scientific literature and in fact is an impossible concept.

Q. Well, have you testified before that there is -- well, first of all, do you agree that there is no safe level of exposure to

benzene?

A. I don't believe that the scientific and medical literature supports the concept of no threshold or that there is no safe level or level below which there are no demonstrable health effects.

Q. Have those opinions that you are expressing right now, have you gained those in the last three years?

A. No. I've had them for 20 years.

Q. Can you point to a study that says that there is a safe level of exposure to benzene?

A. As I said earlier in this deposition, scientists can't prove a negative. We have no scientific basis to conclude that zero is the only safe level. It's a theoretical concept, and I don't think that it is supported by the scientific and medical literature.

Q. Is there a level in your mind for benzene exposure, a threshold level at which -- or below which no one in this world will develop a benzene-related illness?

A. I can't -- I can't make a reasonable scientific -- render an opinion to that

question. I don't believe that there is any data that allows us to draw a conclusion one way or the other below those levels of exposure that have been demonstrated in quantitative studies to be associated with an increased risk.

Q. So in your opinion, as I understand what you're saying, there is no threshold level below which exposure is safe for every individual; is that a fair statement?

MR. SCOTT: Object to the form of the question.

A. Could you repeat or rephrase that question.

Q. (BY MR. BROWN) Yes, sir. There is no -- in your opinion there is no threshold level below which exposure to benzene is safe for all people?

A. No, that's not my opinion. My opinion is that the scientific and medical

literature does not indicate that there is no safe level of benzene exposure. The scientific and medical literature -- scientists can't prove a negative. What we can do is ascertain in studies at what levels or what conditions of exposure to a chemical such as benzene are

associated with a significant increase in the incidence of a disease and therefore an increased risk. That's all we can do.

Q. Let me object to responsiveness. My question is, Do you have a level that you have in your mind would be your opinion that would -- if somebody did not exceed that level, that it would be safe for all people?

MR. SCOTT: Object to the form of the question.

A. I believe that reliable quantitative epidemiologic studies don't provide an indication of increased risk of acute myeloid leukemia associated with cumulative exposures certainly below 45 parts per million years and on the order of 60 parts per million years. That is the state of the art today.

Q. (BY MR. BROWN) Let me object to responsiveness. Sir, is it your testimony that

no person in this world will develop benzene-related leukemias if they do not exceed the 45 parts per million years?

MR. SCOTT: Object to the form of the question.

A. I'm sorry. There are too many

negatives in that sentence.

Q. (BY MR. BROWN) Is it your testimony that no person in this world will develop a benzene-related leukemia so long as they do not exceed the 45 parts per million years quantitation that you just described?

MR. SCOTT: Object to the form of the question.

A. No, I can't say that and no responsible scientist would say that.

(The reporter interrupted for clarification.)

Q. (BY MR. BROWN) That's because every person reacts differently to chemicals; that's the whole concept of individual susceptibility, isn't it?

MR. SCOTT: Object to the form of the question.

A. You now have asked me two questions.

I believe the first question -- well, actually could you restate that.

Q. (BY MR. BROWN) Yes, sir. Are you familiar with the concept of individual susceptibility?

A. Oh, yeah.

Q. What is that concept?

A. Individual susceptibility is that individual variations in individuals, even genetic or environmental, can influence or alter their susceptibility to development of diseases following exposure to individual agents. It is a very well described and understood concept.

Q. It's been described and understood in the medical literature for years, correct?

A. To the extent that we understand it exists, yes. With respect to specific genetic susceptibility for individual diseases, that's really an area of ongoing research, intense research.

Q. Have you ever seen the 1943 Soley document report on benzene to Shell before?

A. I don't know whether I have or not.

Q. Did you -- have you ever heard of Dr. Mayo Soley?

A. No, I haven't. Not that I recall.

Q. Have you ever heard of the University of California Medical School Pharmacological Laboratory?

A. I've heard -- I'm not sure which branch of the University of California you are

talking about.

Q. Well, it's 1943, so I don't know how many different branches they would have had back at that time. You can see the data at the bottom.

A. San Francisco. I'm not familiar with this specific laboratory.

Q. I want to read to you what his report to Shell was on the chronic health hazards of benzene. First of all, you can see here where he mentions chronic poison and some of the diseases that result from exposure to benzene include leukemia, correct?

MR. SCOTT: Object to the form of the question.

A. Yes. To the -- whatever that means.

Q. (BY MR. BROWN) He is saying leukemia, right?

MR. SCOTT: Object to the form of

the question.

A. The definition and diagnosis of leukemia has changed dramatically since 1943. It's even spelled here l-u-c as opposed to l-u-k. I see that on the page. I don't know what Dr. Soley is specifically referring to.

Q. (BY MR. BROWN) Are you saying that the way he spelled it, l-u-c-e-e-m-i-a, that that's not leukemia?

MR. SCOTT: Object to the form of the question.

A. I'm not saying that. I'm saying I can't assume what precise disease he is talking about there.

Q. (BY MR. BROWN) You've never seen the English variation of the spelling of leukemia?

A. That is not current English spelling for leukemia and hasn't been certainly during my career.

Q. This is a 1943 document that is mentioning leukemia, and you are not disputing that, are you?

MR. SCOTT: Object to the form of the question.

A. No.

Q. (BY MR. BROWN) Okay. What I wanted to show you is where he says, "While prolonged exposure to any concentration of benzene is dangerous, there is marked variation in susceptibility of individuals so that some, for unknown reasons, are particularly resistant

while others are quite susceptible."

MR. SCOTT: Object.

Q. (BY MR. BROWN) That concept, that's the same concept of individual susceptibility that you are referring to, correct?

MR. SCOTT: Object to the form of the question.

A. Yes, I believe I've already said that.

Q. (BY MR. BROWN) For that reason you couldn't tell us, nor any other scientist could tell us, how much benzene it takes for any particular individual to be exposed to before that they might develop a disease related to that exposure, correct?

MR. SCOTT: Object to form of the question.

A. I don't believe that is the reason we can't prove a negative. I believe that

individual susceptibility is -- is an operating influence in all the studies that have examined quantitative exposure to benzene and demonstrated a relationship between cumulative exposure to benzene at certain levels and an increased risk of AML. Those are the

individuals who are susceptible to development of the disease. Individuals who are -- many individuals exposed at comparable levels do not develop the disease. So we can assume that the frequency of AML and the risk of AML associated with quantitative exposure to a given level includes those individuals by definition who have increased susceptibility. So I believe that individual susceptibility is a major factor in determining risk of developing a disease such as AML associated with benzene, and they are included in the quantitative studies that we currently have that provide us with information as to that quantitative risk.

Q. (BY MR. BROWN) All right. Let me object to responsiveness. Can you tell me, sir, and the jury at what level it took for Mr. McCarty -- or would have taken for Mr. McCarty individually in terms of exposure to

benzene for him to develop leukemia?

MR. SCOTT: Object to form of the question.

A. All I can tell you is what I've already testified to, which is the quantitative epidemiology literature does not support an

increased risk of the development of AML below certainly 45 ppm-years, and most studies on the order of 60 ppm-years. Those are the lowest levels of cumulative exposure that to date have been associated with significant increased risk of AML following benzene exposure.

Q. (BY MR. BROWN) Object to nonresponsive. My question is, sir, can you tell me and the jury at what level it was required for Mr. McCarty to be exposed to benzene and him develop leukemia?

MR. SCOTT: Object to form of the question.

A. I believe I've just given you the best answer I can to that question.

Q. (BY MR. BROWN) Well, have you ever seen Mr. McCarty's name in any epidemiological study?

A. No.

Q. Have you ever seen any other individual's name in any epidemiological study?

A. I would hope not.

Q. All these epidemiological studies do is study population. They cannot tell us at what level any specific individual would develop

a disease process; isn't that true?

MR. SCOTT: Object to form of the question.

A. The quantitative study of populations in epidemiology and quantitative controlled clinical studies is the best and only tool we have with which to ascertain the potential risk of developing a disease associated with a given agent. That is the limits of scientific and medical state of the art that we work within, and as a scientist that is the basis and can be the only basis for my opinion on the quantitative relationship between benzene and the development of acute myeloid leukemia.

Q. (BY MR. BROWN) I understand that you are limited in what you can use to formulate your opinions as a toxicologist, but the bottom line answer to my question is, you can't tell a

jury how much benzene exposure it took for Mr. McCarty to develop his leukemia, can you?

MR. SCOTT: Object to form of the question.

A. I have seen no data that would allow me to reach a conclusion that in fact

Mr. McCarty's acute myeloid leukemia was associated with exposure to benzene.

Q. (BY MR. BROWN) I mean, it would be possible that an individual could be exposed to high levels of benzene and not develop a disease, correct?

A. Yes, definitely.

Q. And it's also possible that individuals can be exposed to extremely low levels of benzene and develop a disease, correct?

A. Well, we have quantitative data to support the former. We cannot determine the latter based upon scientific quantitative data. We can't prove a negative.

Q. Okay. Can't prove it, in other words?

A. Can't prove a negative. That's not something that a scientist can do.

Q. All right. Were you familiar with Dr. Hine who consulted with Shell concerning toxicology of chemicals?

A. I don't know who you are talking about.

Q. All right. Was it -- in your

experience in studying benzene over these last 20 to 30 years, have you come to understand that chemical companies and industry often hire consultants to help them assess hazards of the chemicals that they use and make and require their employees to work around?

A. Sometimes, yes.

Q. And with regard to -- tell me some of the companies who you are familiar with that going on, that type of thing happening in particular with regard to benzene.

A. Well, certainly most of the companies -- certainly any of the companies that I've consulted for in the past have been interested in precisely that, which is understanding the nature of my opinions or my -- or to ask my expertise in evaluating potential hazards associated with in the case of -- in this specific case, benzene.

Q. And that type of activity went on and was used by companies in the 1940s, 1950s, correct?

A. I've seen reports by individuals who presumably were hired as consultants by companies in the '40s and '50s.

Q. Here is another document I'm going to refer you to. This is a report to Shell Development Company regarding certain problems of environmental cancer in the petroleum industry. Do you see that?

A. Yes, it's by Charles Hine. Now I know who Dr. Hine was.

Q. How did you know Dr. Hine?

A. Dr. Hine was a professor of toxicology and pharmacology at the University of -- if this is the same Dr. Charles Hine that I knew, at the University of California, San Francisco. A grand old man. Many, many, many years ago. We are talking -- I knew him briefly 30 years ago. He was retiring at that point.

Q. Did you know him to be a consultant of Shell?

A. No.

Q. Did you know him to be a reputable

and competent toxicologist?

A. Yes. I think he was highly respected. Certainly for his day he was highly respected.

Q. Let me show you what he has to say about benzene. He says, "In only a relatively

few instances can the origin of an environmental cancer be traced to a contact with a well-defined chemical agent possessing established carcinogenic qualities. Among such compounds include arsenic," and the second one is on this list is benzol, correct?

MR. SCOTT: Object to the form of the question.

A. What he says is benzol; that's correct.

Q. (BY MR. BROWN) That's benzene, isn't it, sir?

A. It can be. I would presume he is talking about benzene, but he also could be talking about gasoline, but I think he is talking about benzene.

Q. Are you saying back in 1950 that this reputable toxicologist would have been saying it was a well-established carcinogen?

Gasoline was a well-established carcinogen?

A. No. That's why I'm saying -- I'm saying the use of -- specific use of the term benzol most likely relates, pertains to benzene, but I can't -- I can't get inside his mind, but that's what I assume he is saying.

Q. As you read that document, this Dr. Hine who you knew as a competent toxicologist, well respected, is saying in 1950 that benzene has well-established carcinogenic characteristics, correct?

MR. SCOTT: Object to the form of the question.

A. That's what -- that apparently is what he is saying, yes.

Q. (BY MR. BROWN) Did you know Allen Dooley from Texaco?

A. No.

Q. Did you know that even in 1954 he was reporting that benzene had a destructive effect on the bone marrow and could cause latent injuries and was even citing to the 1948 API as something that companies should use to help them assess the risks associated with benzene exposure?

MR. SCOTT: Object to form of the question.

A. As I said, I don't know Dr. Dooley. I don't know of him. However, the statement that in fact benzene causes bone marrow damage was generally understood much earlier than even

that.

Q. (BY MR. BROWN) Bone marrow damage, including aplastic anemia, which is a fatal blood disease, correct?

A. It can be, yes.

Q. Were you aware that in Texas it has been the law since 1958 that employers monitor their work environment so that they can be assured that they don't overexpose employees to benzene?

MR. SCOTT: Object to form of the question.

A. Well, I'm not specifically aware of Texas law in particular dating back to the 1950s, but I'm generally aware that in fact that's an obligation of employers.

Q. (BY MR. BROWN) Were you aware that as early as 1960 the Walsh-Healey Act required employers or companies who sold goods over

$10,000 to the government, the United States government, that they had to monitor and protect their workers from exposure to chemicals including benzene?

MR. SCOTT: Object to the form of the question.

A. I have absolutely no argument with the notion, but I'm not familiar with the Walsh-Healey Act.

Q. (BY MR. BROWN) Never heard of that or had any questions asked to you about that in all of your 40 or so benzene depositions?

MR. SCOTT: Object to form of the question.

A. I probably have, but I don't recognize it as the Walsh-Healey Act, so I'm not sure what you are talking about specifically, but I have no argument with the concept.

Q. (BY MR. BROWN) Would you agree, sir, that by 1960 there was no secret in the chemical industry that benzene was known to cause deadly blood diseases?

MR. SCOTT: Object to form of the question.

A. Benzene was associated with chronic

bone marrow toxicity far earlier than that. At that particular point in history, there were no quantitative epidemiology studies that demonstrated a clear-cut relationship between benzene and leukemia. Acute myelogenous leukemia in particular. At this -- during that

decade we became aware not only -- and when I say "we" I'm talking with the scientific community as a whole because I was -- I was a youngster at that point. But in the 1960s -- over the 1960s is when there was first evidence in the medical community that bone marrow toxicity and bone marrow damage per se could lead to the development of AML, and there was also emerging evidence associating benzene in particular with the development of AML. So those began to occur over the decade of the '60s.

Q. (BY MR. BROWN) Let me object to responsiveness. My question was more simple than that. Isn't it true that by the 1960s, it was well known among the chemical industry that benzene could cause deadly diseases?

MR. SCOTT: Object to form of the question.

A. With respect to bone marrow toxicity, it certainly should have been, yes.

Q. (BY MR. BROWN) There would have been no reason for a company who was selling and handling chemicals and requiring its employees to work around chemicals, for them not to know

and have knowledge that benzene could cause deadly diseases, correct?

MR. SCOTT: Object to form of the question.

A. As a matter of principle, in general, I think that employers have an obligation, whether or not and independent of specific laws, to provide information relating to the potential hazards and toxicity of the substances that they are exposed to.

Q. (BY MR. BROWN) All right. I would agree with that, too, but my question was more along the lines of your previous answer that by the 1960s members of the chemical industry should know about the hazards of benzene and that it could cause deadly diseases for those who were exposed to it?

MR. SCOTT: Object to form of the question.

A. By the 1960s and certainly during the 1960s, the evidence in the scientific and medical literature associated with the development of bone marrow toxicity in general and benzene was generally recognized.

Q. (BY MR. BROWN) It was widespread by

the '60s, correct?

A. With respect to bone marrow toxicity, yes.

Q. And with respect to deaths resulting from bone marrow toxicity, correct?

A. From severe chronic exposure, that's true.

MR. SCOTT: Darren, if you get to a spot someplace, I would like to take a break, just whenever you get there, a rest room break.

MR. BROWN: Okay. If you need to take a rest room break, we can take one right now.

MR. SCOTT: If this is good for you to stop, that's fine.

MR. BROWN: I ain't going to keep a man from going to the rest room.

MR. SCOTT: I'm good for a while. Just early warning here.

MR. BROWN: I got so noticed out of your questions that I might have to go someday. So if we need to take a rest room break, let's do it.

MR. SCOTT: Thanks.

THE VIDEOGRAPHER: The time is

approximately 3:33 p.m. and we are now off the record.

(A break was taken.)

THE VIDEOGRAPHER: The time is approximately 3:38 p.m. and we are now on the record.

Q. (BY MR. BROWN) Sir, you've told us you are not an industrial hygienist, correct?

A. That's correct.

Q. Are you seen any quantitative industrial hygiene monitoring data for Mr. McCarty for the years that he worked at the Van Waters & Rogers facility in Beaumont, Texas?

A. No.

Q. Have you seen any monitoring data for Mr. McCarty at all?

A. No.

Q. Have you seen any monitoring data for any person whom you believe or whom others

have told you were engaged in representative jobs that Mr. McCarty would have been doing? You don't understand that question?

A. No, I didn't.

Q. Let me reask it. Have you seen any industrial hygiene monitoring data that you

contend is representative of Mr. McCarty's exposure while he worked at Van Waters & Rogers?

A. Specifically, no.

Q. And that would be, as you I'm sure recall from reading of the depositions that you told me that you read, because Van Waters & Rogers never did monitor workers like Mr. McCarty, correct?

A. I can't speak to that one way or the other, based upon my review of the testimony and information that I have read. My focus in reviewing the boxes of information that I have looked at in this case were based upon rendering the opinions that I have and I really did not focus on other issues.

Q. All right. Sir, I will represent to you that Mr. Wellen and others who we've deposed have told us that there was no industrial hygiene monitoring of anybody at Van Waters &

Rogers during the years that Mr. McCarty worked there for industrial hygiene monitoring in the breathing zone for benzene. All right? You can take that as a representation to me -- from me based on my review in those depositions that you have copies of, okay?

Assuming that that is true, wouldn't you agree that it would be very difficult to have actual quantitative data on the types of exposures that Mr. McCarty would have been experiencing without the industrial hygiene monitoring data?

A. To the extent that -- to the extent that -- a quantitative exposure analysis optimally requires monitoring data. In the absence of specific monitoring data, often mathematical models are used or analogies or surrogate exposures or monitoring data can be substituted. It's not as robust as direct monitoring data, but it's the state of the art with respect to industrial hygiene.

Q. And with respect to trying to assess an individual's exposure to a certain chemical, wouldn't you agree that the most accurate and the best way of doing that would be to do

industrial hygiene monitoring of that person's breathing zone?

A. The most accurate way of doing it would be to do personal monitoring of the individual, not necessarily breathing zone, but basically quantitative, repeated analysis, but

that's -- that is simply not possible to do individual monitoring on everyone that has ever worked, even in benzene manufacturing. Individual personal monitoring data is used in developing and documenting exposure estimates, but not everyone all the time can be monitored for exposure to benzene or anything else.

Q. Let me object to responsiveness. Sir, do you recognize -- I know you are not an industrial hygienist, but you say you have experience in some issues of industrial hygiene. Do you recognize that the standard for determining worker exposures to a chemical is to do industrial hygiene monitoring of that worker's breathing zone?

A. Yes, I do.

Q. All right. Is there any more accurate way of assessing a worker's exposure to chemicals than conducting the standard and

competent industrial hygiene monitoring of his breathing zone?

A. There are other techniques that can be useful in evaluating an overall quantitative exposure for an individual or group of individuals besides breathing zone measurements.

This includes exhaled breath measurements, this includes personal monitoring, this includes a variety of different quantitative techniques that can be used in a -- certainly in an environment where you are doing research or trying to assess quantitative exposure.

The techniques I'm talking about are not routinely used in monitoring individual workers in the workplace. They are the subject of studies, and they are used in research in industrial hygiene.

Q. Let me object to responsiveness. My question was, Do you know of a more accurate way of assessing a worker's exposure to a particular chemical than to do industrial hygiene monitoring of his breathing zone?

A. I think I just told you that one can obtain more accurate assessments -- the more information you have, the more data you have

always the better it is. And there are experimental procedures besides doing breathing zone measurements. Breathing zone measurements are the standard for evaluating exposure in the workplace on a routine basis.

Q. Okay. And without having any type

of monitoring for Mr. McCarty or any of his coworkers, those who try to assess or quantify his exposures has to rely upon less accurate or less reliable means of doing so, correct?

A. You can always -- I can't think of a situation where one has absolutely every piece of quantitative data that you can imagine in order to evaluate an exposure. That simply is not the case. I do believe that there are very effective and useful methodologies that involve surrogate analysis and involve use of physical monitoring. And I think mathematical models can be very useful in evaluating and extrapolating that data. I think that certainly within terms of approximating quantitative exposure, those are reasonable surrogates in many circumstances.

Q. Let me object to responsiveness. You have not done on your own any mathematical modeling or quantitative or surrogate analysis

of Mr. McCarty's exposure, correct?

A. No, I have not.

Q. All right. And you had to rely upon somebody else to do that for you, correct?

A. That's correct.

Q. You didn't have enough information

to make those decisions or design a model that you felt comfortable with on your own; is that true?

A. I did not want to make a quantitative assessment of his exposure.

Q. Okay. Another way of trying to get an estimate of what a worker's exposure would have been would be to use means other than quantitative measurements, correct?

A. Yes.

Q. And those are -- to do that they are often referred to as qualitative estimates of exposure, correct?

A. That's correct.

Q. And that's at least been in your practice acceptable, scientific means of trying to assess a worker exposure, correct?

A. I think it's less valuable than a quantitative exposure assessment. In the

absence of a quantitative exposure assessment, a qualitative exposure assessment is basically all I would have to fall back on in terms of evaluating exposure, but it's by no means the ideal.

Q. It's not the ideal, but it's

accepted and within the epidemiological field within the toxicological field for determining what potential exposures or range of exposures may have been at a particular time, correct?

A. I would disagree with that. I think that certainly in terms of epidemiology and in quantitative epidemiology, qualitative estimates are not acceptable surrogates for quantitative exposure assessments.

Q. Well, that's what I'm saying. Not all -- not all epidemiological assessments have the benefit of quantitative data, correct?

A. That's correct, but I'm saying that a quantitative epidemiology study requires a quantitative exposure assessment.

Q. All right. Well, there are many epidemiological studies that don't have quantitative data that are relied upon by epidemiologists and toxicologists in determining

disease causation, correct?

A. Not in determining quantitative disease causation. You have to have some estimate of exposure. You have to have some basis for determining that you understand the nature of the exposure that an individual or

individuals in a population have experienced.

Q. Some basis of exposure, and that can be gained through talking with a plant industrial hygienist or gathering other information that would indicate exposures at a particular level, other than by quantifying or quantitative means or industrial hygiene monitoring, correct?

A. Obviously in the absence of the ability to do any quantitative measurements, which is rare, or assessments, epidemiologists will rely on qualitative analysis. I don't believe those studies are as reliable in determining the quantitative relationship between exposure and disease. Often -- the most often scenario that I think is as reliable as most studies are involves a combination of assessing the qualitative exposure and providing some kind of quantitative estimate, modeling or

actual analysis of industrial hygiene data.

Q. Let me object to responsiveness.

One qualitative method of evaluating exposures would be to consider or look at the odor threshold of a particular chemical, correct?

A. That's not a useful quantitative

measure of assessment.

Q. All right. Are you saying it's totally useless?

A. I'm saying certainly it's not a quantitative -- it's not a quantitative estimate, and it's not a reliable estimate in determining the nature of a quantitative exposure.

Q. Let me object to responsiveness. My question is, Are you saying evaluating exposures by odor threshold of chemicals is a totally useless way of trying to come up with at least a minimum exposure to a chemical?

A. Close to useless. It is certainly not useful in a quantitative assessment of exposure.

Q. Well --

A. It's a highly unreliable parameter and is subject to a host of deficiencies.

Q. Well, you indicated earlier that you had gathered data on odor threshold of chemicals. So I assume you did that with some purpose in mind, correct?

A. Yes. I provided those, the references that I have relating to exposure,

because I believe that Mr. Parker's analysis of the potential exposure to benzene based on odor thresholds are not -- don't comport with the scientific and medical literature associated with odor thresholds of benzene, and in fact there are numerous authorities that disagree with his conclusions.

Q. Okay. Again, I need to object to responsiveness. Do you know what the American Industrial Hygiene Association is?

A. It's an association of industrial hygienists.

Q. Do you recognize the American Industrial Hygiene Association as a reliable source of industrial hygiene information?

A. Sometimes. Not necessarily just simply on the basis of the fact that it's the Industrial Hygiene Association. It depends upon what it is, who it is that is making a

statement, whether it's the association or an individual, whether it's based upon experimental direct evidence, what the source of it is, and I would take into -- I take into consideration not only its single source, but what are the available -- what are the available data sets

and authorities and sources of information that are available in the world literature.

Q. I'm just asking a simple question. Do you or do you not recognize the American Industrial Hygiene Association as a reliable source of industrial hygiene information?

MR. SCOTT: Object to form of the question.

A. Sometimes.

Q. (BY MR. BROWN) I mean, in general do you feel like that that group publishes reliable information concerning industrial hygiene and exposure information, for instance?

A. Sometimes.

Q. Can you think of anytime specifically that you say that they have been unreliable and you have not relied upon them?

A. To the extent that any summary of data doesn't comport with the preponderance of

data available in the scientific and medical literature concerning a given finding, then I would have to disagree. I can't -- I can't on the basis of defining a single authority, textbook, or organization say a priori that because there is a document that has been

published by them or that information contained in a tome that's produced by -- in a textbook or any organization a priori carries the weight of scientific proof. So I would have to evaluate every individual item or piece of evidence or conclusion on an individual basis.

Q. And so I guess what you are saying is that if the matter concerned a matter of industrial hygiene, you would include as a source to consider the opinions of the American Industrial Hygiene Association?

A. I would consider -- I would consider them as a source, and I would -- yes, I would -- I think that they are an authority that should be considered in evaluating any particular issue related to industrial hygiene, but not categorically would I conclude that any given document or statement is accurate or true.

Q. Do you know that the American

Industrial Hygiene Association has published odor threshold figures for various chemicals?

A. Yes.

Q. And do you know that they have published one for the chemical benzene?

A. I believe they published one many,

many, many years ago.

Q. Go ahead.

A. I recall seeing -- I don't remember the date, but it's very old.

Q. Well, I mean, you don't recall the date. So again, you don't know how many times they've published odor thresholds for various chemicals, I would assume, correct?

A. They published -- I know that there are compendiums of odor thresholds. There are numerous authorities that have published on odor thresholds for many -- for many compounds. I've provided several here today. There are many different groups, government agencies, as well as nongovernment groups like the ACGIH. Many different organizations have published data on the physical, chemical properties of chemicals, including odor thresholds and other properties.

Q. Other than for litigation, do you

use odor thresholds at all in your business?

A. I think they are highly unreliable.

I would not use them in a quantitative way, and only in the most general qualitative way, because most individuals' knowledge and understanding of what it is they are smelling or

what the nature of a given smell is, is highly unreliable when untrained.

Q. Let me object to responsiveness. My question is, Other than in litigation, do you use odor threshold in your business for any reason?

A. For any reason? Only as a very general guidance.

Q. For what particular application?

A. For ascertaining whether or not an individual perceives that they have been exposed to a solvent or some other compound or chemical.

Q. Are you familiar with what the American -- I mean, the American Industrial Hygiene Association says is the odor threshold for benzene?

MR. SCOTT: Object to form.

A. If you want to show me the document, I would be happy to discuss it. Specifically

the exact number you are talking about, I would have to see it.

Q. (BY MR. BROWN) All right. Well, if the American Industrial Hygiene Association has published that the odor threshold for benzene, the median value, is 61 parts per million, would

you dispute that figure?

MR. SCOTT: Object to the form of the question.

A. I think that the vast majority of studies on odor threshold of benzene and authorities who have published on odor threshold of benzene are not consistent with that level and in fact are consistent with vastly lower odor thresholds in terms of concentration.

Q. (BY MR. BROWN) Let me object to responsive. Would you dispute the American Industrial Hygiene Association's median odor threshold value of 61 parts per million for benzene?

MR. SCOTT: Object to form.

A. Based on my knowledge and the knowledge of the literature, yes.

Q. (BY MR. BROWN) All right. What studies do you have that would allow you to

dispute the American Industrial Hygiene Association odor threshold for benzene particularly when you are not even an industrial hygienist?

MR. SCOTT: Object to form of the question.

A. I only brought a few. I didn't bring everything in the literature. But I've brought references associated with the determination of odor threshold for benzene from numerous sources. This one is the National Safety Council, which reports an odor threshold of approximately 2 parts per million.

Q. (BY MR. BROWN) Is that odor identification threshold? Is that odor recognition threshold? Can you --

A. It's odor -- it's odor recognition threshold.

Q. Of 2 parts per million?

A. Yes. By recognition I mean exposure to something. Got a 1989 report from the American Industrial Hygiene Association, which I think is the same organization we've been talking about, and it gives a geometric mean for a odor threshold of 61. A geometric mean is not

a mean. It's not an arithmetic mean. It is a single point in any number of random measurements giving only weight to the individual point. It does not -- there could be -- for example, out of 100 measurements there could be 99 at 5 parts per million or less, for

example, and one at -- my arithmetic is not this good -- let's say something on the order of 90, and the geometric mean would be 61.

Q. That's an average of what you were saying, correct?

A. This is a geometric mean. That's not an arithmetic mean.

Q. The range that they report for the 61 parts per million geometric mean was what?

A. It's hard to say. They give two measurements. They don't -- it's hard for me to tell from this whether it's a range. Again --

Q. Does it say 34 to 97 on there?

A. No. It says 61 and then there's another figure on the line below that says 97. But as I said before, that's a geometric mean. It's not an arithmetic mean. I've got other citations. Here's the Texas Medical Library -- let's see. AMA, American Medical Association,

from -- this is 1959, which is extremely old.

Q. Before we go any further, I hate to stop you. We are going to run out of tape. If we could stop right now and hold your thought and we will continue on right there.

THE VIDEOGRAPHER: The time is

approximately 4:01 p.m. and we are now off the record.

The time is approximately 4:03 p.m. and we are back on the record.

A. As long ago as 1959 the American Medical Association published a report on the state of knowledge with respect to the toxicology, biochemistry, and industrial hygiene of various substances and reported an odor threshold of benzene of 1.5 ppm.

The World Health Organization on environmental health criteria dated 1993 presented the same type of data on benzene and reported a range of odor -- a range of concentrations for odor threshold of between 4.8 and 15 milligrams per cubic meter, which is one -- roughly one to four and a half parts per million benzene.

A reference from 1972 in Clinical

Toxicology, Fifth Edition, reports an odor threshold of benzene of 1.5 to 5 ppm for detection. The U.S. Department of Health and Human Services from 1997 reported a detection threshold odor, which is again 4.9 milligrams per meter cubed, which would be 1.5 parts per

million. These are just some examples.

Q. (BY MR. BROWN) Let me object to responsiveness. Sir, with regard to the study by the American Industrial Hygiene Association, first of all, do you have any reason to think that that industry is biased in any way --

MR. SCOTT: Object to form --

Q. (BY MR. BROWN) -- or that association is biased in any way?

MR. SCOTT: Object to the form of the question.

A. No. I believe that the variance between virtually all the other authorities on odor threshold and that particular report is the use of a geometric mean, which does not convey the weight or frequency of measurements made at a given level. And I believe that most of the other odor thresholds have been calculated on the basis of the quantitative weight of the

number determinations and arithmetic mean.

Q. (BY MR. BROWN) Let me object to the nonresponsive portions of the answer. With regard to the American Industrial Hygiene Association study, do you know how they came up with their numbers, and do you know if such

method was an accepted scientific method of arriving at odor threshold of chemicals?

MR. SCOTT: Object to form of the question.

A. I haven't -- I have not evaluated or studied their specific methodology. I am simply providing you with my opinion based upon the number of authorities that have provided information on an odor threshold for benzene which indicate that the odor threshold is far less than that reported by the ACGIH in that particular report.

Q. (BY MR. BROWN) All right. Object to nonresponsive. As I understand your answer, you have not evaluated the methodology used by the American Industrial Hygiene Association for arriving at odor thresholds that they did, correct?

A. Other than my analysis of the

statistical parameters that they use, which was geometric mean, which I do understand and I do understand the difference between the methodology for calculating the mean between an arithmetic and a geometric mean.

Q. What I'm asking is, did you

understand what methodology that they used. Do you know how they determined what levels, what kind of industrial hygiene monitoring, or any kind of monitoring that they used?

A. No.

Q. Do you know anything about the parameters of their studies or their report?

A. No.

MR. SCOTT: Object to the form of the question.

Q. (BY MR. BROWN) All right. With regard to any of the other studies that you've referenced here today, do you know anything about the methodologies that were used in those that have the values which you say are more in line with what the odor thresholds are?

A. No.

Q. So you couldn't vouch for those studies or say that they were accurate in any

way; you just have to read the studies just like anybody else, correct?

A. That's correct.

Q. Have you ever done any independent research on odor thresholds of chemicals, specifically with regard to benzene, yourself?

A. I'm currently directing a study that is evaluating exposure to benzene. As I said before, my reliance on odor threshold is -- I don't rely on odor threshold except the anecdotal indication that an individual may have that they perceive they have been exposed to a volatile substance.

Q. Let me object to responsiveness. My question was, Have you done any independent research on the odor thresholds of chemicals, specifically benzene?

A. No.

Q. All right. With regard to the research that you are doing in Shanghai, do you use odor thresholds in any aspect of that research?

A. No, not substantively.

Q. Do you know how much benzene exposure that it takes for an individual to

become dizzy or lightheaded?

A. Usually between 100 and 500 parts per million. Most individuals will experience some sensory -- sensorium impairment before they get to levels of 500 parts per million.

Q. All right. Well, are you saying

that if somebody is exposed at the level above 500 parts per million, that he is going to be physically impaired to the extent he can't do his job?

A. No.

Q. That's not your testimony, correct?

A. No, I'm saying they will experience some dizziness or lightheadedness.

Q. You read the affidavit of and deposition of Mr. Beverly where he indicated while they were drumming benzene, he experienced lightheadedness or dizziness, correct?

A. I vaguely recall that, yes.

Q. All right. And do you recall Mr. McCarty saying those same things in his sworn affidavit?

MR. SCOTT: Object to form of the question.

A. As I indicated to you earlier, I

don't recall specifically Mr. McCarty's comments.

Q. (BY MR. BROWN) Okay. You didn't even use Mr. McCarty's affidavit in formulating your opinions; that's correct?

A. I did not use his affidavit in

formulating my opinion with respect to exposure.

Q. Isn't it important, sir, to you as a toxicologist trying to assess exposure, if somebody said I drummed benzene and I drummed it where the concentrations were such that it made me lightheaded or dizzy, that would be an important fact in your evaluation?

A. If in fact there was evidence to support or corroborate that in fact that individual was exposed to benzene, it would be of some value to me. But I don't believe that individual anecdotal reports with respect to perception are useful in determining the nature of the specific substance that an individual is exposed to.

Q. Are you saying to the jury you don't think Mr. McCarty was exposed to benzene?

A. I am saying that there is no -- I've seen no specific evidence to -- that I can rely

upon to determine that in fact Mr. McCarty was exposed to benzene, and I don't believe that his anecdotal testimony relating to what he smelled is useful in answering that question.

Q. Sir, the man said he was exposed to benzene. He filled it up in 55-gallon drums.

His co-worker said he did the same thing. Are you saying he's lying?

MR. SCOTT: Object to the form of the question.

A. I am not saying and I do not believe that Mr. McCarty is lying. I don't believe that his testimony provides me with sufficient information to determine that in fact he was exposed to benzene.

Q. (BY MR. BROWN) But he says he was, so that's not good enough for you, correct?

MR. SCOTT: Object to form of the question.

A. That's not -- that is not information as a toxicologist I can use in determining or assessing his potential quantitative exposure to benzene.

Q. (BY MR. BROWN) All right. What would you have to have in addition to his word

and his co-worker's word that he was exposed to benzene and that they filled up 55-gallon drums of benzene often during the month for four to five hours at a time without any protection -- what in addition to that would you need to say you had enough information to perform a

toxicological evaluation that he was exposed to benzene?

MR. SCOTT: Object to the form of the question.

A. Well, I don't recall in fact that I read testimony in my review of the material provided to me that provides independent corroboration that in fact, with the exception of Mr. Beverly and Mr. McCarty, that they in fact -- were in fact drumming benzene. And furthermore, I think that in order to ascertain that that in fact was the substance -- solvent that they were involved in drumming, that I need additional information to corroborate that that was in fact the solvent.

Q. (BY MR. BROWN) That's what I'm asking. What additional information do you need other than the workers' own good word, hey, it was benzene we were putting in these drums --

and we got tickets from 1969 that says they were doing that; somehow for some reason we can't find them going back any further -- but what do you need in addition to the man's word it was benzene we were putting in those drums before you feel confident enough to say that he had

some exposure to benzene?

MR. SCOTT: Object to form of the question. We are just arguing now. And Mr. McCarty wasn't there at that point that you got tickets, so --

Q. (BY MR. BROWN) What additional information do you need other than their word that they got exposed to benzene?

MR. SCOTT: Same objection.

A. As a toxicologist, I would need some independent corroboration that in fact benzene was present on the facility, that they were in fact drumming benzene, and in fact they were working with -- in that environment at a time when benzene was present at the facility.

Q. (BY MR. BROWN) Did you see Mr. Wellen's testimony where he said, yes, we did drum benzene at the Beaumont facility in the late '60s?

MR. SCOTT: Object to form of the question.

A. I -- as I -- as I testified earlier today, I recall testimony that suggested the possibility or that referred to the possibility that in fact there was benzene at that facility

in the late '60s. That does not comport with my understanding of the period of time that's at issue with respect to Mr. McCarty's exposure.

Q. Bottom line is you don't know if Mr. McCarty was exposed to benzene or not, correct?

A. That's correct.

Q. And you wouldn't take that job away, that fact-finding job away from the jury, if they wanted to believe Mr. McCarty and his co-worker that they were exposed to benzene while they worked at Van Waters & Rogers, would you?

MR. SCOTT: Object to form of the question.

A. As a toxicologist, I cannot render an opinion with respect to potential for Mr. McCarty's exposure to benzene without some objective evidence that in fact he was exposed.

I am simply telling you that based upon my opinion, I've seen nothing that would lead me to conclude that in fact he was exposed to benzene.

Q. (BY MR. BROWN) And to come to that opinion you have to ignore his coworker's testimony and his sworn affidavit that he was,

correct?

MR. SCOTT: Object to form of the question.

A. I don't believe that's the case at all.

Q. (BY MR. BROWN) Well, that's what you are doing, isn't it, sir?

MR. SCOTT: Object to form of the question.

A. I reviewed a great deal of material including testimony that did not indicate that benzene was drummed at that facility during the time that Mr. McCarty was there.

Q. (BY MR. BROWN) Are you ignoring their testimony or are you not ignoring it?

MR. SCOTT: Object to form of the question.

A. I am not using anecdotal testimony as a basis for determining what in fact

Mr. McCarty's exposure to benzene was.

Q. (BY MR. BROWN) His statements about what he was exposed to are of no use to you and you can ignore those in terms of how you come up with your opinions, correct?

MR. SCOTT: Object to form of the

question.

A. As an expert in rendering opinion with respect to benzene exposure and the relationship between benzene exposure and the development of Mr. McCarty's disease, I cannot rely on anecdotal information in reaching -- solely anecdotal information in reaching that conclusion.

Q. (BY MR. BROWN) Do you have any studies, sir, that you think fairly and accurately represent what Mr. McCarty's exposure to benzene was while he was drumming that chemical into 55-gallon drums for four to five hours at a time?

MR. SCOTT: Object to form of the question.

A. No.

Q. (BY MR. BROWN) As a toxicologist and a health professional, what would you think

about a work practice of a company that allowed employees to drum pure benzene for four to five hours at a time into 55-gallon drums without respiratory protection or any PPE or any warning of benzene hazards?

MR. SCOTT: Object to form of the

question.

A. Based upon our knowledge of the hazards of benzene and the existing state of the art with respect to regulation of benzene, I believe that that would be both illegal and inappropriate today.

Q. (BY MR. BROWN) That would be utterly ridiculous today, wouldn't it, sir?

MR. SCOTT: Object to form of the question.

A. It is -- with respect to the state of the art today, that would not be acceptable.

Q. (BY MR. BROWN) All right. And even back in the 1960s when companies, as you said, should have known that benzene caused deadly diseases, that information was available to them, that was utterly ridiculous back at that time too, wasn't it, sir?

MR. SCOTT: Object to form of the

question.

A. I still, as I sit here today, don't believe, and I don't have any information to conclude, that the conditions associated with the -- certainly the described -- the conditions described in Mr. Beverly or Mr. McCarty's

testimony -- Mr. Beverly's as I remember -- that provides me with a -- even a qualitative indication that the levels of exposure that Mr. McCarty was potentially exposed to, if you assume all of the conditions in Mr. Beverly's affidavit, necessarily is associated with an exposure that would be consistent with that which has been previously demonstrated to result in a quantitative increase in the risk of AML. That's one of the reasons why I think it's important, when possible, to rely on a quantitative exposure estimate.

Q. (BY MR. BROWN) I think I'm supposed to object to nonresponsive here. My question was, It would have been utterly ridiculous in the 1960s for a company to allow its employees to drum pure benzene into 55-gallon drums for four to five hours at a time without respirators, PPE, or any warning of benzene

hazards whatsoever due to the fact that in the '60s it was widely known that benzene could cause deadly illnesses, correct?

MR. SCOTT: Object to form of the question.

A. Based on my knowledge, that's not

the case. That is not necessarily consistent with state of the art during the '60s.

Q. (BY MR. BROWN) Assume with me that that is what was happening. I know you don't necessarily believe Mr. McCarty and his co-worker, Mr. Beverly, but assume with me that the jury does. Would you at least be able to tell the jury that would have been a silly, utterly ridiculous practice for a company to allows employees to drum benzene, pure benzene into 55-gallon grams without any protection whatsoever and without any warning that benzene could hurt?

MR. SCOTT: Object to the form of the question. It's repetitive and harassing.

A. I don't believe that the state of the art at the time that that procedure or those practices would be inconsistent with state of knowledge at that particular time.

Q. (BY MR. BROWN) And what basis do you have to say that the state of the art was such that at that time that would have been an acceptable practice?

MR. SCOTT: Object to the form of the question.

A. The general scientific and medical communities did not reach the conclusion that quantitative -- quantitative exposure, repeated exposure to benzene was associated with the development of AML until the mid to late 1970s. During the 1960s, the medical and scientific communities were first becoming aware that exposures to benzene at or below 100 -- 50 to 100 parts per million could be associated with the development of toxicity.

This is an evolving field, and the level of scientific knowledge and understanding has developed markedly over the last 80 years, and at that particular point in time, although I don't think that those practices would be considered appropriate today, I think that that is consistent with my understanding of state-of-the-art during the '60s.

Q. (BY MR. BROWN) Let me object to

responsiveness. Sir, did you read any of the depositions from Shell that -- from their representatives that said had they known Van Waters & Rogers was doing this back in the '60s, they wouldn't even have sold the benzene to them at all?

MR. SCOTT: Object to the form of the question.

A. I don't recall.

Q. (BY MR. BROWN) Did you read the testimony of the Shell representative that said, If they would have told us that, we wouldn't have sold it to them at all, and we asked them what they were using it for and if -- the only way they could have gotten it and used it like they did in drumming it from trucks would have been to lie to us to get it?

MR. SCOTT: Object to the form of the question.

A. Sir, I don't understand the question.

Q. (BY MR. BROWN) Yes, let me reask it. Did you read any of the testimony from the Shell representative in this case who said when they sold benzene to companies like Van Waters &

Rogers, they would ask what the company was going to do with it, and the only way that Van Waters & Rogers could have gotten the benzene from Shell and put it into drums out of a truck would have been to lie to Shell so that Shell would sell it to them?

MR. SCOTT: Object to form of the question.

A. I don't recall that.

Q. (BY MR. BROWN) As you sit here, can you tell us what the exposure would have been for Mr. McCarty as he filled up 55-gallon drums of benzene with both bungs out of the barrel for four to five hours in hot summer months out there on the docks of the Van Waters & Rogers facility in Beaumont, Texas?

MR. SCOTT: Object to form of the question.

A. I base my opinions and my understanding of Mr. McCarty's exposure on the quantitative assessment that I've received that we've discussed today, and I have no opinion as to what his exposure specifically would have been under the conditions you described.

Q. (BY MR. BROWN) You are not able to

tell us a number, correct?

A. No.

Q. Sir, do you know of any reliable peer-reviewed information that says intermittent exposures to even low levels of benzene can be more toxic to the blood-forming elements than

continuous exposures to even higher levels?

A. I have hypothesized in the past that intermittent exposure is probably from a pathogenic standpoint the most significant exposure with respect to the pathogenesis of specific toxicity of the bone marrow, but intermittent exposure in that context is in fact the exposure that I believe is associated with the typical workplace environment that is included and subsumed in the quantitative exposure analysis.

So I think the typical industrial exposure environment is that of a succession of intermittent exposures far more frequently than some constant average, theoretical, low-level or high-level exposure. The level of exposure in most workplace situations, whether it's high or low, tends to be sporadic and intermittent.

Q. All right. With -- you have stated

to others before that you think that intermittent exposures could be more damaging to the bone marrow, correct?

A. Mechanistically, yes, but epidemiology -- quantitative epidemiology studies don't support that as a metric as being

more sensitive than cumulative exposure.

Q. So are you saying you are wrong about intermittent exposures being more dangerous than continuous exposures?

A. No, I think we are looking at two different things. I'm looking at the potential molecular mechanisms of bone marrow toxicity and what's the nature of the exposure environment. And the epidemiology studies are looking at the sum total human experience of exposure. And based upon the technologies and the methodologies that are used in assessing exposure, intermittent exposure metrics have proved to be less sensitive than cumulative exposure methods.

Q. You are aware that the concept that the body with only --

(The reporter interrupted.)

MR. BROWN: Take a break if you need

to.

THE VIDEOGRAPHER: Time is approximately 4:26 p.m., and we are now off the record.

(A break was taken.)

THE VIDEOGRAPHER: The time is

approximately 4:30 p.m. and we are back on the record.

Q. (BY MR. BROWN) Sir, we were talking about the -- your opinions as to relative danger of intermittent exposures to levels of benzene being more toxic to the bone marrow than higher levels of continuous exposure. Do you recall our questioning about that?

A. Yes.

Q. In 1983 do you recall verifying to Dr. Peter Infante that idea that you had and also placing that information into the OSHA benzene document?

A. No, I did not. I didn't do that. I gave a -- and I did not talk to Dr. Infante personally. I gave a presentation at a meeting in Washington, D.C., based on my experimental data in animals suggesting that intermittent exposure might produce greater toxicity than

continuous in the context of benzene metabolites introduced directly into the bone marrow in in vitro and vivos situations. That information was not provided by me to OSHA. I would not have thought that it would have been significant to the issues that OSHA was reviewing with

respect to benzene emissions from the lake and hydride plants at that time.

Q. So you would deny that you made those statements, that those statements were transcribed, and it was the New York benzene meeting that I'm referring to, and that those -- that presentation that you gave at the meeting was transcribed and the transcription of that meeting was put into the OSHA benzene document?

A. I'm not sure what you just said. I don't know what the question is.

Q. Well, we were talking about your idea about the intermittent exposures being more toxic to the bone marrow than continuous exposures to benzene even at higher levels, and that concept being put into a transcribed document and placed into the OSHA benzene document. You are saying that did not happen, correct?

MR. SCOTT: Object to form of the question.

A. No, I'm not saying that. What is the question you are asking me?

Q. (BY MR. BROWN) That is the question I'm asking.

MR. SCOTT: Object to form.

A. I'm sorry. I did not detect a question.

Q. (BY MR. BROWN) All right. You are saying that you never told Peter Infante that there was -- you had the opinions that intermittent exposure to levels of benzene are more toxic to the bone marrow than continuous exposures at even higher levels?

A. No, I'm not saying that. That's not what I was saying.

Q. You are not saying that?

A. No, I'm not saying that.

Q. So you wouldn't deny Peter Infante if he said you told him that, correct?

A. I did not tell him individually specifically that. I presented that at a meeting once that Dr. Infante attended. I presented research data from experimental

20 animals. That does not specifically reach the
21 same conclusion that you did but I think
22 supports another -- something else that I've
23 already responded to you in one of my answers.
24 What I'm saying is I did not specifically talk
25 to Dr. Infante by himself concerning

intermittent exposures, and I did not submit that presentation of mine to OSHA.

Q. During the years of 1977 through 1988, you worked for the Chemical Industry Institute of Technology; is that correct?

A. Chemical Institute of Toxicology.

Q. Okay. I'm sorry. During the years of 1977 through 1988, you worked for the chemical institute -- Chemical Industry Institute of Toxicology; is that correct?

A. That's correct.

Q. Who funded the work being done by the Chemical Institute -- Industry Institute of Toxicology?

A. CIIT received funding primarily from chemical companies.

Q. What were some of those companies?

A. Chevron, Texaco, Shell, Mobil, Exxon, to name a few.

Q. DuPont?

A. I believe so, yes.

Q. Dow?

A. Yes.

Q. W.R. Grace?

A. Yes.

Q. Conoco?

A. I think so, but I can't recall specifically.

Q. Phillips?

A. Again, I think so, but I can't recall.

Q. Do you recall testifying that those two entities did fund the CIIT before?

A. Well, I don't recall specifically they did, but I don't find it at all inconsistent. I just don't remember that far back.

Q. Wouldn't deny they were contributors or sponsors, correct?

A. No.

Q. Monsanto?

A. Yes.

Q. Any others?

A. As I sit here, I don't recall. It's

been a long time.

Q. While you were there, what was your job?

A. I was a pathologist, scientist, and senior scientist.

Q. Did any of your job duties include

peer review?

A. Peer review -- I've been involved in peer review for my entire career. So while I was at CIIT I was involved in peer review.

Q. Did you know a health scientist or health professional by the name of Kligerman?

A. Yes.

Q. How did you know him?

A. He was another scientist at CIIT.

Q. What was his name, first name?

A. Andrew.

Q. Were you working at CIIT when Kligerman had trouble getting his findings published that benzene was causing chromosomal abnormalities at concentrations as low as 1 part per million?

A. I worked at CIIT during -- certainly during the period when Dr. Kligerman was doing studies on chromosomal aberrations on benzene.

I don't recall specifically what you are describing.

Q. Well, do you recall him having trouble getting his chromosomal findings on -- chromosomal abnormalities associated with benzene exposures, getting that work published?

A. I don't recall what specific difficulties he had with respect to peer review of his publications.

Q. Do you recall that he had opined and found through his work that benzene was causing chromosomal abnormalities in concentrations as low as 1 part per million?

A. I recall that he had data that he produced that suggested or that he thought represented demonstration of an association, and I don't even remember the end point, between exposures to benzene at low concentrations and chromosome aberrations. Not aberrations. Other end points. I don't remember specifically the concentration or the end points. I remember there was controversy over the quantitative nature of the results.

Q. Did you peer review that work by Dr. Kligerman?

A. I think every scientist probably did at CIIT. That would have been common and typical.

Q. Did you agree with his work or his findings?

A. I don't recall the specific issues.

I remember -- I remember that I often had and offered opinions that criticized or critiqued studies of my colleagues, and my studies were also criticized and critiqued by my colleagues.

Q. Do you know who, if anybody, was behind any effort to discourage or prevent the publication of Dr. Kligerman's work concerning chromosomal abnormalities?

MR. SCOTT: Object to the form of the question.

A. I don't know what you are talking about. I don't know what you are referring to.

Q. (BY MR. BROWN) Well, did he get his work published?

A. I believe that -- I believe Kligerman's studies were published. I don't recall specifically where or specifically when, but it's my recollection that his studies were published. I can't tell you specifically one

way or way or the other, but it's certainly my -- it's my recollection that they were.

Q. A finding that benzene was causing chromosomal abnormalities in concentrations as low as 1 part per million would have been a finding that none of the companies who were

funding the Chemical Institute -- Industry Institute of Toxicology would have wanted, correct?

MR. SCOTT: Object to form of the question.

A. I think if in fact Dr. Kligerman's studies provided quantitative, definitive evidence of that, it would have been important. I vaguely recall that there were some issues related to the nature of the studies, not the findings themselves. But I don't remember the details.

Q. (BY MR. BROWN) Well, my question was that people who were funding your company where Dr. Kligerman was working and you were working would not have really liked to have seen a finding that benzene could cause chromosomal damage in concentrations as low as 1 part per million back at that time, would they?

MR. SCOTT: Object to the form of the question.

A. I can't speak to the individual companies or representatives, but whether in fact -- whether or not an individual company would have liked those results or not, if in

fact CIIT scientists did studies that produced reliable peer-reviewed data that that was in fact the case, it would have been published by the institute. We -- certainly in my case I -- it was -- actually, all of us, it was made demonstrably clear to us many times that our work and our results were independent of the sponsoring companies and our publications were independent of the sponsoring companies.

Q. (BY MR. BROWN) So for the life of you, you couldn't tell the jury whether or not you think those companies would have liked to have seen a finding like that?

MR. SCOTT: Object to form of the question.

A. I'm saying it wouldn't have made any difference with respect to the publication of his data.

Q. (BY MR. BROWN) But my question was,

would the companies who were funding you have liked to have seen that result, and you are telling me you don't know, correct?

MR. SCOTT: Object to the form of the question. It's harassing.

A. As an expert I can't sit here today

and tell you what a company thinks or what the companies would have thought.

Q. (BY MR. BROWN) Do you remember telling Dr. Peter Infante about the Kligerman findings that we've been discussing?

A. No.

Q. Are you familiar with the Lan study?

A. Yes.

Q. The study showing blood abnormalities from benzene exposures down to .2 parts per million?

A. I wouldn't necessarily characterize those as abnormalities. The Lan study provides a cross-sectional analysis of isolated blood parameters, none of which are clinically significant.

Q. What are your disputes or concern with the Lan study?

A. The independent measurements that

they make, there's -- the individual differences that they report are less than the typical diurnal variation in an individual. They aren't -- the differences aren't associated with any clinically recognized hematologic abnormality.

Q. Sir, you said you were asked in this case to form opinions as to the cause of Mr. McCarty's myelodysplastic syndrome and acute myelogenous leukemia. Have you done that?

A. Yes.

Q. What is your opinion as to what caused Mr. McCarty's myelodysplastic syndrome and acute myelogenous leukemia?

A. It's my opinion that the evidence with respect to exposure and the development of his disease, that there is no basis for me to conclude to a reasonable scientific medical probability that in fact his disease was associated with exposure to benzene.

Q. That's based on the evidence that you've seen and how you have interpret it, correct?

A. That's correct.

Q. Do you have any other opinions about

the causation issue with regard to Mr. McCarty's disease and what it would have been related to?

A. No, other than the fact that at Mr. McCarty's age, the prevalence of myelodysplastic syndrome is relatively high, but I have no basis or opinions with respect to

alternative causes.

Q. All right. Let me ask you this question: Are you saying Mr. McCarty's age of 70 years old is what caused him to have myelodysplastic syndrome and acute myelogenous leukemia?

A. I'm saying that myelodysplastic syndrome in particular is far more prevalent in people in the fifth, sixth, and seventh decades of life than it is earlier in life. So individuals in that particular age group have a higher overall incidence of myelodysplastic syndrome.

Q. But at the same time, Mr. McCarty's age didn't cause his disease, correct?

MR. SCOTT: Object to the form of the question.

A. I didn't say that.

Q. (BY MR. BROWN) He was -- it's your

understanding from the literature you've reviewed that he was at greater risk for developing myelodysplastic syndrome or acute myelogenous leukemia because of his age, correct?

A. Yes, defining risk as an

epidemiologic -- as an epidemiologic concept, yes.

Q. And that's because the damage that occurs from the toxic exposures that cause myelodysplastic syndrome and acute myelogenous leukemia take a while to take place, correct?

A. Not necessarily. I don't think you can -- I don't think that that's a cause and effect relationship that's been established in the scientific or medical literature, and I don't think that you can necessarily assume that that is the mechanism associated with the development of MDS in the majority of individuals.

Q. Well, because of latency issues, persons will tend to be older in the development of their disease than diseases that don't have latency issues, correct?

A. Latency requires or assumes that

there is a point in time an etiologic agent that is associated that can be defined, it's associated with the development of the disease. The increased incidence of MDS in aging individuals does not inform with respect to latency at all. It doesn't tell us anything

about latency.

Q. Well, you would agree that latency would have an effect on somebody's age at the time of diagnosis, correct?

MR. SCOTT: Object to form of the question.

A. No.

Q. (BY MR. BROWN) Well, if the guy -- well --

A. Latency -- I'm sorry.

Q. If a guy -- what is -- let me just ask you, what is your opinion as to the latency period for -- from first exposure to benzene to clinical diagnosis of benzene-related leukemia?

A. Quantitative epidemiology studies indicate a very clear period of latency with a median of around 9 1/2 to 10 1/2 years, and some follow-up studies of epidemiologic studies suggest it may be as high as 15 to 20 years. So

I would say the range is basically probably from on the order of five years as a reasonable probability early and 20 years as an outside probability, based on the epidemiological evidence.

Q. Is that median?

A. No, those are ranges.

Q. All right. Well, you are aware of the studies in the literature that show ranges of latency for leukemia after exposure to a toxic substance like benzene being -- ranging in period of time from one to 40 years, correct?

A. Certainly not for acute myelogenous -- no, not for acute myelogenous leukemia, no. Sorry.

Q. You've never seen the Shell study that found a latency period of 47 years for an acute myelogenous leukemia?

A. I don't think that an individual case -- first of all, I'm not aware specifically what you are referring -- I don't know what you are referring to as the Shell study, but independent of that, a specific case --

Q. The Shell look-back study.

A. I have not -- that doesn't mean

anything to me.

Q. The study of its refinery and chemical plant down in Deer Park, Texas.

A. I can't -- I don't recall a specific individual, but you have to remember that quantitative epidemiology defines individuals

that either comprise a cohort or a defined set of study subjects, and that in any study you are going to have individuals who present with disease that some of which are likely to be associated with the exposure and some of which are going to happen independent of exposure. If you assume -- so in the design of a study, that's always going to be the case. Some individuals in the study are likely to have an etiology that's associated with -- have a defined etiology, and others are going to develop the disease independent of that etiology.

In the assessment of latency, no one individual constitutes proof of a period of latency. You have to look at the entire cohort or the entire set in order to ascertain whether or not there's a reasonable probability of developing the disease in a given period of time

based on the entire study. Cohort, not individuals.

Q. Latency is described in terms of averages, correct, or means?

A. Or ranges, based on scientific probability.

Q. All right. And is it your testimony that a person who has exposure to benzene, first exposure to benzene, and presents with a clinical diagnosis of leukemia 21 years later, that there is no possibility that that diagnosis of leukemia is related to exposures to benzene?

A. I wouldn't say that. What I'm saying is the probability that a latency exceeding 20 years is likely to be associated in terms of probability with benzene exposure, that that -- that basically it's unlikely. It's less likely that an individual with a latency of 20 years or greater, that that individual's disease is associated with benzene exposure. I don't think the literature supports it.

Q. What you are saying is based upon the median latencies that are in the quantitative epidemiology studies that you have reviewed, correct?

A. It's based on the range of latencies reported and documented in the quantitative epidemiologic literature.

Q. You agree that qualified, competent scientists have different opinions than you on the average and mean latencies of development of

leukemia from exposure to benzene, correct?

A. I have no idea in general what you are referring to. If you've got a specific document, I would be happy to look at it.

Q. Well, let's mark this as the next exhibit.

(Irons Deposition Exhibit 7 was marked.)

Q. Do you know Dr. Peter Infante?

A. I'm familiar with him, yes.

Q. Do you consider him to be a competent epidemiologist?

A. I think that he's -- Dr. Infante performed an epidemiology study that -- approximately 30 years ago that represents one of the milestones in the quantitative epidemiologic assessment of benzene chemogenesis. Since then I'm not aware that he has done any primary research in the field.

Q. Do you know of his reputation of being a competent epidemiologist or do you say he's not?

A. I can't speak to his competency as an epidemiologist.

Q. Have you ever seen a study called

Benzene and Leukemia -- or a publication called "Benzene and Leukemia: Cell Types, Latency, and Amount of Exposure Associated With Leukemia"? Have you ever seen the study before or the publication?

A. This -- I wouldn't refer to this as a study. This is a -- this is basically a review and an editorial. It's a review article in which Dr. Infante states his opinions and his review of the literature concerning benzene and leukemia.

Q. All right. He states there in that first highlighted paragraph -- would you read that for me, please?

A. Which?

Q. The first highlighted paragraph.

A. "The latency period for benzene leukemia can range from less than one year to more than 40 years after initial exposure with

the median latency for various studies ranging from 9 to 35 years."

Q. Do you agree with that statement or not?

A. No, I disagree with it.

Q. All right. So you recognize that

good scientists can agree to disagree, correct?

MR. SCOTT: Object to the form of the question.

A. I disagree with Dr. Infante's opinion as stated here.

Q. (BY MR. BROWN) What he is saying is he has looked at the literature and this is what he sees in the literature, and you are disagreeing with his statement there?

A. That's correct.

Q. Can you read the second highlighted portion, please.

A. "Quantitative risk assessment based on data from three major benzene cohort studies where relatively good information on dose was provided to estimate cohort exposures indicated a risk of ten excess leukemia deaths per thousand workers exposed over an occupational lifetime, 45 years, to an average concentration

of one ppm benzene."

Q. Do you agree or disagree with that statement?

A. Well, as I testified to earlier today, I believe that the minimum -- that quantitative epidemiology provides in the -- for

a minimum of a cumulative exposure estimate of 45 ppm-years associated with significant excess of AML. That is exactly what Dr. Infante is saying.

Q. So, I mean, you are saying Dr. Infante's opinion is you have to have 45 ppm-years to get leukemia; is that right?

A. I'm saying that Dr. Infante says here that there is a risk of 10 excess leukemia deaths per thousand workers exposed over an occupational lifetime of 45 years to an average concentration of 1 ppm benzene. His cumulative quantitative metric here is 45 ppm-years.

I don't agree that there is any evidence exposure to 1 part per million benzene at any length of time is associated with causation of AML. But there is evidence in the epidemiologic literature in at least one study that suggests that 45 ppm-years would be the

lowest cumulative exposure that's associated with significant excess.

Q. Well, you know OSHA disagrees with you on that; you know that OSHA's mathematical modeling of excess leukemias are existing even at below concentrations of 1 part per million,

correct?

A. I can't speak to what OSHA or what an organization like OSHA has for an opinion. I am talking about the quantitative epidemiologic literature.

Q. All right. It says right here, page 117, referring to Table 3, that 34 percent of those who died from leukemia were exposed to average levels of benzene ranging between .5 and 5 parts per million in this particular study. Do you see that?

MR. SCOTT: Object to form of the question.

A. That's what -- that's what this says.

Q. (BY MR. BROWN) All right. Do you agree or disagree with that statement?

A. I have no idea what he is referring to. I don't know what he means by average.

Average is not a very useful concept in terms of quantitative exposure. An individual that's exposed to an average of 5 parts per million benzene could be exposed to 250 ppm benzene sometimes and virtually infinitesimal amounts at other times. Average means nothing.

Q. What are the risk factors of myelodysplastic syndrome and AML?

A. Epidemiology -- in terms of the epidemiology, age. For certain types of MDS, previous exposure to alkylating chemotherapeutic agents. I believe that prolonged chronic exposure to benzene is associated with the development of myelodysplastic syndrome. In fact I published to that effect. There are studies that speculate that there may be infectious or viral etiologies.

I think there's some anecdotal evidence that in fact previous infection with certain parasitic agents may be associated with it but I don't think there is quantitative evidence in the literature to support that as of yet. As I sit here, I think those are the major factors.

Q. What about radiation?

A. Yes, radiation can cause myelodysplastic syndrome. I don't think it's a frequent cause of the disease. But it can, yes.

Q. With regard to the risk factors that you've just told me about, have you ruled out radiation as a cause of Mr. McCarty's

myelodysplastic syndrome and AML?

A. I have no -- I have no information that would allow me to conclude that radiation played a role. I have no information to exclude radiation.

Q. You have no information whatsoever that Mr. McCarty had any exposure to radiation, correct?

A. That's correct.

Q. You don't have any opinion that Mr. McCarty's exposure to radiation, if any, was a cause or contributing factor of his AML, correct?

A. That's correct.

Q. Do you have any evidence that he was exposed to any chemotherapeutic drugs or alkylating agents prior to developing myelodysplastic syndrome or AML?

A. No.

Q. So you don't have an opinion that any alkylating agent or chemotherapeutic drug is a cause of his myelodysplastic syndrome or AML, correct?

A. Not to my knowledge.

Q. With regard to infectious

etiologies, it's my understanding from what you've just told me, you don't think that the science is sufficiently adequate to say that any of those are a particular cause of myelodysplastic syndrome or AML, correct?

A. At this time, yes.

Q. Do you have any information that Mr. McCarty had any type of infection or any other condition that you believe could have caused his myelodysplastic syndrome or AML?

A. Since in fact I don't think the scientific literature supports that as a conclusion for causation, I have -- I don't believe that there is any evidence that Mr. McCarty's MDS is associated with infection.

Q. Other than his age, do you think -- do you have an opinion that any other risk factor or potential risk factor played a role in his development of his myelodysplastic syndrome

or AML?

A. I'm not aware of it.

Q. And you -- that is what you've been asked to try to find out, correct?

A. I've been asked to render an opinion specifically related to benzene, not to

determine a priori what the cause of his disease is.

Q. Well, did you consider any potential risk factors he had, in your opinion?

A. Yes, I considered his age, and in my review of his records, I didn't find anything that would suggest that -- I've already testified to the fact that anything to suggest that radiation played a role in the development of his disease.

Q. He smoked cigars but in the distant past. Do you have any opinion that cigarette -- cigar smoke played any role in the development of his condition?

A. No, I don't. There is evidence in the medical literature to suggest an increased risk of AML associated with cigarette smoking. There is no evidence that I'm aware of that says that there is an association specifically with

MDS or specifically with cigar smoking.

Q. Let me object to responsiveness then. Do you have an opinion in this case that Mr. McCarty's cigar smoke in the distant past played any role or contributing factor in the development of his myelodysplastic syndrome or

AML?

A. I'll say it once again. I do not believe the scientific or medical literature provides specific evidence to suggest or to support a conclusion that cigar smoking is associated with the development of MDS or AML.

Q. In cigarette smoke -- the studies that you just referred to as being suggestive of a contributing factor of AML, what is there in cigarette smoke that you believe allows one to say that cigarette smoke causes acute myelogenous leukemia?

A. There have been many hypotheses. It has been hypothesized that benzene is a causative agent. However, there are many other agents in cigarette smoke that are potentially associated with bone marrow toxicity. As a matter of fact, there are many compounds in cigarette smoke at much higher concentrations

than benzene that are actually metabolites of benzene in individuals following benzene exposure. So I don't think it's at all clear what the causative agent is in cigarette smoke. There are several substances in cigarette smoke that are potentially toxic to bone marrow.

Q. Do you have any idea what kind of concentrations of benzene one would inhale from cigarette smoke?

A. It's really -- there have been speculations and estimates of that. I don't think they are credible or cogent because I don't think that the actual concentration in cigarette smoke is a potential factor in the development of the disease. It's the overall dose and the concentration of delivery which is different than other potential sources of exposure. So I am not -- I really can't provide you with an expert opinion on what the concentration would be delivered to the lung.

Q. Do you think benzene in cigarette smoke causes or contribute to AML?

A. I don't think that is the factor that -- my personal opinion -- and there is no quantitative evidence supporting the specific

agents or agent -- agent or agents in cigarette smoke that are associated with pathogenesis of AML. So this is basically my opinion as a scientist, but not -- I can't render an informed opinion as to what the -- there's no epidemiology that will allow me to reach a

conclusion of what is the causal agent.

What I'm saying is there are several agents in cigarette smoke that could potentially alter regulation of bone marrow hematopoiesis or that might be potentially toxic, and we simply don't have a basis for understanding the pathogenesis of that mechanism.

Q. All right. Sir, have you relied upon any study, publication, information or whatsoever from Mr. Paustenbach -- or Dr. Paustenbach in forming your opinions in this case?

A. Specifically no. Dr. Paustenbach has published quantitative estimates of benzene exposure and risk associated with AML. I believe they are consistent with the studies that I've provided here today, but specifically I did not rely on any of his publications, but I think that I could just as easily have produced

a risk analysis that has been published by Dr. Paustenbach that would be consistent with my opinion.

Q. Let me object, responsiveness. My question is, for the purpose of our record, it's my understanding you have not relied upon any

information, study, or publication from Dr. Paustenbach in any way in forming your opinion, even though you would assume some might be consistent with some of these other studies; am I correct about that?

A. No, sir. That's not my testimony and that's not what I'm saying. As I said earlier, I've relied on the world literature in general and several articles specifically in rendering my opinion. I brought some that are representative of what I consider to be the state of the art in quantitative epidemiology associated with benzene and the development of AML.

I did not bring everything that I've relied upon or that I would consider the basis for my opinion, and that includes certainly at least one publication by Dr. Paustenbach which I didn't provide here today.

Q. So you've got one publication from Dr. Paustenbach that you have relied upon in performing your -- in making your opinions in this case?

A. You know, I just said I've relied on the world literature, which includes

publications by Dr. Paustenbach. As I sit here, I can recall one particular assessment that is consistent with my opinion and with the papers that I provided here today. I did not produce it, but in general I'm relying on the literature that's out there, not just specifically and exclusively those that I provided.

Q. Well, I mean, help me out here, sir. Are you relying upon Mr. Paustenbach -- or Dr. Paustenbach at all in forming your opinions in this case?

MR. SCOTT: Object to --

Q. (BY MR. BROWN) And if you are, show me the study that you are relying upon.

MR. SCOTT: Object to form.

A. I'm relying on the world literature related to the epidemiology of benzene, which includes publications by Dr. Paustenbach.

Q. (BY MR. BROWN) All right. What I

would ask you to do, sir, you say you've got other publications and studies and information, the world literature that you are relying upon. I'm asking you -- I know you have produced some of it -- to produce to Mr. Scott for me as part of this expert deposition all of the literature

you are relying upon, whatever it is, whatever is the world literature that you are going to be saying that this is a study that I've relied upon, so I'll know what it is today or sometime before trial of this case. Don't you think that's fair that I know what you relied upon?

MR. SCOTT: Object to the form of the question. World literature would fill up rooms, not boxes, and doesn't make sense.

MR. BROWN: Bob, I mean, here's the deal. I get to find out what he has relied upon in forming his opinion.

MR. SCOTT: I don't disagree, but are you asking him to give you the world literature?

MR. BROWN: I'm asking him to give me what he has relied upon in forming his opinion, whatever that is.

A. I produced publications that I

provided today not in specific response to your subpoena but as general support and to illustrate my opinions in this case. I will -- I reserve the right as an expert to render an opinion on whatever particular literature or evidence that's out there in the world

literature that you present to me or ask me to review or that anyone else asks me to review. I haven't provided the world literature today as the basis for my opinion, but I do in fact have knowledge of and will respond to individual questions that are related to my opinion that are supported by literature that I did not provide today.

THE VIDEOGRAPHER: Counsel, I need to change the tape.

MR. BROWN: All right.

THE VIDEOGRAPHER: Time is now approximately 5:11 p.m. and we are now off the record.

(Discussion off the record.)

THE VIDEOGRAPHER: Time is approximately 5:12 p.m. and we are back on the record.

Q. (BY MR. BROWN) All right. Sir,

which study from Dr. Dennis Paustenbach have you relied upon in any amount or any way in formulating your opinions in this case?

A. Specifically I did not review Dr. Paustenbach's studies in arriving at my opinion in this case. I can recall studies that

Dr. Paustenbach has performed that are -- that inform with respect to the quantitative epidemiologic assessment of the risk associated with AML following exposure to benzene. I can specifically refer to and provide those studies, but I didn't provide them today.

Q. And you've told me at the beginning you brought the studies you thought that you needed and were necessary to express support for the basis of your opinions?

A. That are representative, yes.

Q. All right. What I'm asking you to do here today is, would you please bring the other studies that you think play a role in your opinions, to provide those as part of your deposition here today. We've requested in the subpoena -- I know there's been objections to them, but I'm asking you to provide any of the studies that you are relying upon as an expert

or any other information that you are relying upon as an expert in this case to express your opinions. Is that fair?

MR. SCOTT: There haven't been any objections to studies. Just so we are clear there. I object to the form of the question to

that extent.

Q. (BY MR. BROWN) Is that fair that you would provide me with those?

MR. SCOTT: Object to form of the question.

A. With respect to my specific opinions in this case and specific literature, I believe that that's reasonable. I don't believe that it's reasonable for you to ask me to provide the world literature on the pathogenesis and development of MDS, AML, every study that has ever been published on benzene or has anything to do with potential development of disease associated with benzene.

Q. (BY MR. BROWN) I'm not asking for that. I'm asking for the studies that you are relying upon specifically to express your opinions in this case.

A. I think that, yes.

Q. Do we have those in the notebook that you provided?

A. I could provide some more. I did not do so because I didn't believe that that's what I was asked to do.

Q. How many more could you provide or

would you provide as being something you specifically relied upon in preparation for your opinion?

A. Again, this is what I specifically relied upon. If you are asking me to provide everything in the literature that supports my opinion, that's a totally separate matter.

Q. What you've got here in your notebook marked as Exhibit 1 is the complete list of documents that you have specifically relied upon, correct?

A. In -- in -- specifically related to this case, but not in terms of my overall opinion with respect to the quantitative risk associated with exposure to benzene and the development of AML.

Q. So when we get down to trial and there's questions asked how do -- what do you support your opinions with, Doctor, you are not

going to be telling the jury at that time or telling Mr. Scott at that time, well, I relied upon Dr. Dennis Paustenbach's study and it's very informative, anything like that?

A. Specifically -- specifically I'm going to rely on the studies that I've provided

here. If you or Mr. Scott or anyone else asks me my opinion with respect to any other study, I will provide it.

Q. All right. I'll pass the witness and reserve the rest of my time for after Mr. Scott's examination.

EXAMINATION

BY MR. SCOTT:

Q. Dr. Irons, would you tell us a little bit about where you grew up and your educational background.

A. I grew up in the San Francisco Bay Area. I attended the University of Pacific and the University of California, San Francisco, for my undergraduate and early graduate training. I attended the University of Rochester School of Medicine and Dentistry for graduate and medical training, and graduated with a Ph.D. in toxicology in 1974.

Q. Did you start out as a medical student, then?

A. I was in -- I was taking classes in both the school of medicine and the graduate school when I was at the University of Rochester and was undecided as to specifically what

direction I would take until relatively late in my education.

Q. When did that occur when you altered or picked a course as between being a physician and the research course that you've chosen?

A. I received a grant from the National Institute of Arthritis, Metabolism, and Digestive Diseases to study pathology as it related to toxicology, and I combined that with training in clinical pathology, anatomic and surgical pathology for a period of approximately three -- between three and four years after finishing my Ph.D. requirements. At the end of that period I decided not to continue medical education but to go into full-time research at that time.

Q. Many of us have seen television programs involving pathologists and that sort of thing. Did you do that sort of pathology at any

point in your educational career?

A. Yes. That was part and parcel of what I did during that three- to four-year period. I was involved both in research and in training in anatomic and clinical pathology.

Q. And what sorts of things would that

pathology -- that pathology experience include?

A. Participating in autopsies, clinical pathology, clinical laboratory studies. I focused on pathology in general but also specific areas of pathology that were defined on the basis of my grant as toxicologic pathology. My focus was on pathology related to toxic exposure, immunopathology, and what is now referred to as hematopathology, but at the time hematopathology was not a recognized discipline.

Q. How have toxico -- toxicopathology and hematopathology changed since the years you first studied?

A. The classification of diseases has turned over several times. The criteria for the diagnosis of diseases has basically gone from observational and in many cases clinical to primarily a quantitative and laboratory-based process, although certainly morphologic analysis

is still very important in the diagnosis, differential diagnosis of hematopoietic diseases.

Q. We are all familiar with the Human Genome Project and genes and chromosomes in particular and what we read in the newspapers

about those today. How has that changed toxicology and pathology, particularly hematopathology?

MR. BROWN: Object to the form.

MR. SCOTT: What's the objection?

MR. BROWN: It's leading.

MR. SCOTT: That's the objection?

Q. (BY MR. SCOTT) If that's it, you can answer that question.

A. Clinical laboratory, molecular laboratory studies now are the fundamental basis for the differential diagnosis of hematopoietic and lymphoid diseases. Genetic abnormalities, immunophenotypic abnormalities, including flow cytometry, immunohistochemistry, molecular studies looking at specific gene involvement, gene rearrangement, cytogenetics, as well as classic histochemistry and staining are now all integrated into the criteria for classification

of hematopoietic and lymphoid diseases.

Q. How did you -- what interested you in the field of toxicology to begin with?

A. Understanding -- what I was most interested in was understanding the basis for the development of disease and pathogenesis of

disease in general, but particularly that associated with exposure to drugs or chemicals. I was fascinated with understanding the mechanism, and I also felt that the study of the effects of drugs and chemicals provided a very powerful tool for understanding the pathogenesis of disease.

Q. Do we fully understand today the mechanisms involved in the development of diseases, leukemia, and particularly acute myelogenous leukemia?

A. No. We understand a great deal more than we did five years ago, and a tremendous amount more than we did ten years ago. Even so, how it all fits together and what the natural history is and the specific events that occur in the pathogenesis of AML in general and specific AMLs in particular, we still don't have those answers.

Q. Before I get on to some of your other experiences, let me ask you about your family. Are you married?

A. Yes.

Q. How long have you been married?

A. Over 25 years.

Q. Do you have children, and tell us who your children are and what they are doing these days.

A. Yes, I have three children. Katy is a librarian in Tacoma, Seattle area, David is an executive on Madison Avenue, and Sarah is a 21-year-old college student who has just returned from California to Boulder.

Q. Let me -- and you live here in Boulder, Colorado; is that correct?

A. There's a rumor to that effect.

Q. I understand you spend a lot of your time elsewhere from Boulder these days. Can you describe for the jury what your primary job is these days.

A. I'm director of the Joint Sino-US Clinical Molecular Laboratory in Shanghai, China.

Q. How long have you had that position?

A. Basically since -- officially since 2001.

Q. And what are your job duties in that position?

A. The laboratory is both a clinical laboratory responsible for the diagnosis of

leukemia and lymphoma and hematopoietic diseases for 31 hospitals in Shanghai and it's also a research laboratory that's conducting a series of studies on the pathogenesis of hematopoietic diseases, different types of leukemia and different types of lymphoid diseases, and is also involved in studies of the health effects of benzene in workers in the workplace.

Q. I want to hand you some documents that --

MR. SCOTT: Darren, you may have marked these. Did you mark the papers, studies and other papers related -- yeah. We called that Exhibit --

MR. BROWN: 3.

Q. (BY MR. SCOTT) 3. Can you describe for us -- if you will go through each of the separate studies, articles, papers that makes up Exhibit 3 for us. Tell us what it is and what

your role with regard to that document is.

A. Well, in general these are peer-reviewed publications that we have recently published that present results from our studies in Shanghai. The first one is entitled "Chronic exposure to benzene results in a unique form of

dysplasia." This is a study that characterizes a very -- a very unique pattern of abnormalities occurring in the bone marrow of a group of individuals that were exposed to -- chronically exposed to very high levels of benzene, and that we recognized in fact was not previously described in terms of the long-term persistent bone marrow pathology that had been described for benzene in the past.

Q. What is the unique form of dysplasia that results from chronic exposure to benzene, based on the study -- your study that you described?

A. It includes -- it's a specific pattern with respect to the pathologic abnormalities that are prominent in individuals that have been exposed to benzene. Some of these cases correspond to the WHO classification of myelodysplastic syndromes, refractory

cytopenia with multilineage dysplasia. I believe there is one that would qualify as a refractory anemia. Other forms of bone marrow aplasia, many of which previously would have been diagnosed as aplastic anemia in certainly the classic historical literature.

The specific pattern that we see that's unique involves the inflammatory changes that are present, characteristics such as hematophage ptosis, a unique form of eosinophilic precursor abnormalities, dysplasia, a constellation of changes that were found to be consistent for a majority of these individuals that haven't been described together associated with dysplasias in the past.

Q. I notice on Table 1 of the Chronic Exposure to Benzene article, they are a listing of 23 -- are these cases --

A. Yes.

Q. -- on Table 1? These are all people who have documented exposure to benzene?

A. These are all people who have documented exposure to benzene and who previously have been diagnosed with benzene poisoning, which in China the best way to

describe it is they present -- they have presented -- they presented previously with bone marrow findings consistent with bone marrow suppression associated with benzene exposure at the time that these findings were observed.

Q. What levels of exposure to benzene,

based upon your experience, are necessary for a person to present with bone marrow suppression?

A. In general, the literature is consistent or the literature suggests, and this -- the quantitative -- the actual levels of exposure, quantitative levels that have been associated with bone marrow suppression are not as well documented as the epidemiology associated with acute myelogenous leukemia or myeloid leukemia because most of these studies were done before actually that type of epidemiology was even done, and most reported in the literature is anecdotal.

Certainly the experience that we have that is provided or reflected in this publication is associated with exposures that are extremely high relative to the levels that we typically associate with the development of AML.

With respect to these exposures, I think we -- we describe full shift exposures that average, so this is an average, between 50 and 300 parts per million. That's -- that's not ppm-years. That's the average exposure that these individuals were exposed to for any period

of time, for long periods of time, for many years in many cases. Some of the cumulative exposures are typically in excess of 5,000 part per million years.

MR. BROWN: Object to responsiveness.

Q. (BY MR. SCOTT) Let's go to the, if we can, the next paper in Exhibit 3. So I would like to move through these and get some description what each one of these papers involves.

A. One is the "Prospective study of 174 de novo acute myelogenous leukemias according to the WHO classification: subtypes, cytogenetic features and FLT3 mutations." This is basically -- this is our first -- this was our first series reporting on the pattern and specific characteristics of AMLs that have been diagnosed by the laboratory in the general

population, and provide one of the first -- actually the first characterization, detailed characterization of the pattern of AMLs seen in individuals associated -- who have been diagnosed using the WHO classification.

Q. Okay. The WHO classification is a

relatively new classification for hematopoietic disease; is that correct?

A. It was first -- it's been in development for a long time, but it was officially promulgated by the World Health Organization in 2001.

Q. Let's go to the next paper, if we could.

A. This is a paper that characterizes the prevalence of MDS, myelodysplastic syndrome, subtypes in Shanghai and compares the WHO, World Health Organization, classification with the previous predominant classification that was used, and that was the French, American, British classification.

Q. What are the significant factors or findings in this study, Prevalence of MDS subtypes?

A. This provided -- this is -- this --

this paper really provides two pieces of -- overall two pieces of information that are -- that have not -- that are unique. One is this is -- this, I believe, is the first direct comparison of the two diagnostic classification schemes in a clinical study. So we used both

classification schemes in the diagnosis of these cases and compared the prevalence of the individual subtypes based on that classification.

This is important with respect to interpreting studies on the incidence of AML, as well as MDS, because the classification of MDS using these different -- using the different classification -- the rubrics that are used, actually redefine the incidence and prevalence of AML as well as MDS because they are on a continuum.

So in the case of the WHO classification for the same group of individuals looking at a mixture of cases of myelodysplastic syndrome and AML, with the WHO classification more people have AML than with the FAB classification. So this defines those quantitative differences. This paper does.

In addition to which, we uncovered or discovered a very unique prevalence for a type of -- a new classification, a new MDS subtype defined under WHO, refractory cytopenia with multilineage dysplasia. That occurs in a very -- the incidence for it with respect to

age, the prevalence of this disease in Shanghai, there is a very high prevalence in younger individuals, individuals basically below the age of 30, which is a unique finding and suggests that the pattern of MDS certainly in Shanghai is different than that that's been described in the west.

Q. Let's go on to the next paper, if we can.

A. The next paper is an abstract that is published in Chinese and I provided a translation for it. That describes a prognostic analysis of, again, a group of cases, 166 cases of MDS, using WHO in China. This was provided -- this was presented at a conference in China as a means of communicating our results to the general medical community in China.

Q. Okay.

A. I have an abstract here that was

presented at an international conference on benzene in Munich in October of 2004, and it is the original -- it's the first presentation of the data that eventually led to our description of benzene-induced dysplasia, BID, and this described our initial findings with respect to a

group of 18 of the 23 workers that we -- that were the basis for the original publication. And this described the exposure characteristics of the group as well as some of our initial findings.

Q. All right. Let's go to the next one.

A. I have an abstract that presented the initial findings of the prevalence of MDS using the WHO classification that was published in Blood, American Society of Hematology. More recently we have published an abstract describing the relative prevalence of lymphoid neoplasms in Shanghai and the relative frequency of subtypes of non-Hodgkin lymphoma diagnosed using the WHO classification. To date there are not any quantitative case series that have used the WHO classification previous to this. So this is one of the first studies that describe

the different subtypes of NHL in lymphoid neoplasms used in the WHO classification.

Q. All right.

A. The last one in this series is an abstract of a presentation that I provided at the International Conference on Malignant

Lymphoma in Lugano, Switzerland, in 2005, and it describes a very, very rare type of leukemia called aggressive natural killer cell leukemia that we've described in Shanghai. In the world literature over the last 20 years or so, there are a total of maybe slightly less than 100 cases ever reported anywhere, and I believe as of now we have somewhere around ten to twelve that we've diagnosed in Shanghai in just the last couple of years.

Q. Generally, what -- what is the number of cases of lymphohematopoietic disease you and your group have observed or seen in the Shanghai health study today?

A. If you look at all hematopoietic and lymphoid diseases, not just AML per se or its subtypes or what historically has been referred to as NHL, but virtually all hematopoietic diseases, it's on the order of -- I think the

last, my last -- last time I looked at it, it was about 3,000, 3,200 cases.

Q. We are familiar with publications done by investigators, specifically Dr. Hayes, Dr. Rothman, and Dr. Linet and others that have worked with the NCI out of China as well. Are

you familiar with the series of papers or studies that I'm talking about?

A. Yes.

Q. What is the number of cases that they report they have seen, that group has seen?

A. Well, the cohorts for those studies, which means the total number of individuals that were defined within their study criteria, not diseases but individuals, was very, very large, on the order of 30,000 plus, 33,000. The actual number of cases of so-called NHL or AML that are in those -- or MDS in those studies is much smaller. The total number of NHLs reported in that study I believe is 17, of which 14 were associated with exposure to benzene and three were in their control group. I think their total number of AMLs was on the order of 33. I may be off on that by one or two.

Q. Again, how does that compare with

the number of NHLs and AMLs that your group has seen?

A. Their study design is basically a cohort study, which looks at a defined segment of the population, compares exposure, and then looks at the incidence of disease. We have a

totally different design. Their study was a retrospective study that looked at historic exposure and historic diagnosis. Our study is prospective. It's based on concurrent -- previous concurrent, which means present exposure, and looks at diagnosis today, and also follow-up and prognosis of cases after initial diagnosis.

Q. I know there are some other papers in Exhibit 3. Let's be sure we've talked about all of those. They may not be studies or abstracts, but what else is in Exhibit 3?

A. I have provided two descriptions that I have that -- that in response to Mr. Brown's request for documents, that describe the laboratory that I prepared in support of efforts of the laboratory to expand its research and funding base.

Q. What role does Fudan University play

in the studies and the work that you're doing in China now?

A. Fudan University is where the laboratory is located. I am a member of the faculty at Fudan University. So the clinical and research operations of the laboratory are

basically conducted under the auspices and at Fudan University.

Q. What role does the University of Colorado Health Sciences Center in Denver play in the studies and investigations you are working on now?

A. The University of Colorado Health Science Center is the lead institution in terms of the research studies and also provides support both in terms of training and technical expertise in support of the clinical operation in Shanghai.

Q. Have you learned Chinese as part of your efforts with this work, or have you learned Chinese?

A. I'm not a Chinese scholar, but I have a passable knowledge of Mandarin and am China-proof. I can survive in China in Mandarin.

Q. We've talked about the papers related to your current work. How many papers have you authored regarding -- authored or co-authored regarding generally the subject of benzene over your 30-plus-year history, career?

A. Probably between 150 and 160. I'm

not sure exactly.

Q. How many papers have you authored or co-authored regarding lymphohematopoietic diseases, understanding there may be some overlap?

A. There is -- there is -- there is a great deal of overlap. I would say I've probably published on the order roughly of 160 publications. I would say 80 to 90 percent involve benzene and 99 percent involve hematopoietic or lymphoid diseases. I did some earlier work on the liver and liver toxicity and methods of quantitative analysis, but certainly the vast majority of my career has been spent focused on understanding the pathogenesis and cause of hematopoietic and lymphoid diseases.

Q. Have you edited any books regarding the subject of benzene or which deal with the subject of benzene and lymphohematopoietic

diseases?

A. Yes. Toxicology of the Blood and Bone Marrow is a book that I edited and authored many years ago.

Q. Mr. Brown mentioned that the authors of Casarett and Doull or the editors of Casarett

and Doull had cited some of your work in their textbook on toxicology. Have you appeared -- has your work appeared in other textbooks and scientific publications?

A. Yes. Some of which I've authored, some of which I haven't.

Q. Can you give us some idea of the journal, the professional or scientific journals that your work has appeared in?

A. The journal Blood, Proceedings of the National Academy of Sciences, Journal of Clinical Investigation, Toxicology and Applied Pharmacology, Journal of Experimental Pathology, Journal of Immunology, International Journal of Experimental Hematology, Leukemia, Leukemia Research, International Journal of Hematology, European Journal of Hematology.

Q. Mr. Brown marked as Exhibit 2 to this deposition your curriculum vitae. Is that

a pretty good summary of your educational experience, your work experience, your scientific publications and endeavors over your career?

A. I think so, yes.

Q. Have you done work with -- Mr. Brown

mentioned NTP, National Toxicology Program. Have you done work with any of these organizations: NTP, National Institute of Environmental Health Sciences, Occupational Safety and Health Administration, Environmental Protection Agency, any of those sorts of scientific -- governmental advisory or regulatory agencies?

MR. BROWN: Object to form.

MR. SCOTT: Is it just that I've got them all together?

MR. BROWN: I will withdraw it if he makes it clear in his answer.

MR. SCOTT: That's good.

A. Yes.

Q. (BY MR. SCOTT) You need to be more complete than that or I've got to start all over.

A. Earlier in my career I served as a

consultant for the National Toxicology Program on pathology working groups and in the evaluation and peer review of chronic analyst studies. I've served as a consultant to the Environmental Protection Agency and a member of their scientific advisory panel, scientific

review panel. I have served as a -- in that role for a variety of organizations. What other organizations did you mention?

Q. OSHA.

A. I have not specifically done any work for OSHA.

Q. Have you received any awards from any of those organizations?

A. I've received a certificate of commendation from the Environmental Protection Agency for my work on their scientific review panel.

Q. Mr. Brown mentioned earlier Dr. Yin and asked if you were aware of the Yin study. How long have you known Dr. Yin?

A. Longer than I would care to admit. It certainly dates both of us.

Q. And specifically how did you first come into contact or meet Dr. Yin?

A. I met Dr. Yin at a British Royal Society of Medicine -- at the British Royal Society of Medicine London in 19 -- I'm sorry, this is really dating me. Probably 1980, maybe '80. In that general area.

Q. Other than where you met him, have

you had any professional association with him over the years?

A. Yes.

Q. Can you describe that for us.

A. We've been colleagues. Obviously we have common interests in China, he being Chinese and me spending a lot of time there. And I've been in China since -- my first trip to China was in 1988 at his invitation, and we've been colleagues. We obviously have a common interest in benzene. We agree on some things. We disagree on others. We've had a continuing relationship now for a long time.

Q. Let me scroll back a little bit here. You also discussed a little bit in answer to Mr. Brown's questions about the peer review process, and I want to explore that a little bit. You sort of quickly answered and enthusiastically answered that you had had

studies or articles which had not been accepted by journals. I want to talk about the peer review process. First, does any good scientist who publishes and investigates have articles or studies that are not immediately accepted by a particular journal?

MR. BROWN: Object to form.

A. My -- certainly my experience and my knowledge is that most scientists have had the unenviable experience of having their work critiqued and/or not accepted upon initial presentation. I would object to your characterization of my response to Mr. Brown as enthusiastic. It's just a -- it's a part and parcel of the scientific process.

Q. (BY MR. SCOTT) When a journal -- when a scientific journal does not accept a study or an article, what kinds of reasons are those decisions based upon?

A. Well, as I said before, it can be lack of topical interest. It can be lack of relevance with respect to the specific focus of the journal. A journal may decide that a particular manuscript or article is way too specific or deals with too specific a subject

matter than the journal is comfortable -- or is typically published, and they will recommend that you send it to a more specialized journal.

Sometimes a journal is -- sometimes the peer review process results in reviewers who object to usually design and/or interpretation,

usually design, of data and whether or not the methods and the data appropriately support the conclusions of the author. That is subject to individual opinion, and the peer review process is one that allows for usually more than one but certainly one or more scientists with independent -- or independent of the work to render an opinion. If an editor decides that those criticisms are significant enough, he may decide not to accept the paper or to suggest revisions, which is usually up to the individual editor. This is a process that's been in place for well over 100 years.

Q. Have you served as a peer reviewer for scientific journals such as those that you mentioned to us earlier?

A. I've served as a peer reviewer, as an editor, and as a member of editorial boards. I am not currently on any editorial boards

because my time doesn't permit, but I still review publications as a peer reviewer.

Q. When you served as an editor, did you make those decisions about whether a particular submission met the topical interest of the journal, study design was something that

the journal was interested in, that sort of thing?

A. Yes.

Q. You also told Mr. Brown when he asked you about recent instances in which you testified that, quote, I'm busy. What did you mean by that?

A. I spend most of my time in Shanghai. Certainly more than half my time, probably on the order of 60, 70 percent. I'm also a member of the faculty here at the University of Colorado Health Sciences Center and I teach. That doesn't leave much time for me to engage in consultation activities. And so for the last certainly couple of years, my consulting activities have been limited.

Q. How many times have you testified by deposition during the year 2006?

A. None. This is -- this is my first.

Q. Same question for 2006 and testified in a trial?

A. None.

Q. Just so that it's clear here, based upon the state of scientific evidence and literature, what is the level of exposure to

benzene which has been demonstrated to cause acute myelogenous leukemia?

MR. BROWN: Object to form.

MR. SCOTT: What's the objection?

MR. BROWN: Incompetent, lacks foundation.

MR. SCOTT: My question is incompetent or the witness?

MR. BROWN: I'm not saying that. I'm saying it lacks foundation. You didn't set up any foundation for your question.

Q. (BY MR. SCOTT) Okay. Do you remember the question?

A. Could you repeat it.

Q. You bet. I have it written down. Based upon the state of scientific evidence and literature, what is the level of exposure to benzene which has been demonstrated to cause acute myelogenous leukemia?

MR. BROWN: Same objection.

A. I don't think there is any question that the scientific and medical literature supports a causal association with respect to benzene and the development of acute myeloid leukemia at concentrations of 100 ppm-years or

higher. I also believe that quantitative epidemiology studies suggest that accumulative exposure maybe as low as 60 and in one case as low as 45 ppm-years. In general, I think that the literature support -- I think the most likely cumulative exposure that's associated with the development of acute myelogenous leukemia falls somewhere in between those extremes, but I do believe that the literature doesn't support a significant excess risk of AML below 45 ppm-years.

Q. (BY MR. SCOTT) Is there a particular paper or study or investigation that you rely upon in that regard?

A. Two that -- two that I think inform with respect to that, the lower cumulative metric, one would be the Hayes study associated with AML or acute -- acute nonlymphocytic leukemia, which is the China study. The other

is a case control study by Rushton, Leslie Rushton, that reaches a conclusion that in any case, there's no evidence of an increased risk of AML below 45 ppm-years.

Q. Are there published studies -- reliable published studies in the scientific

literature which indicate a higher level of cumulative exposure as the lower bound of those associated with the development of AML?

A. Yes, I believe so. I believe that certainly -- certainly there's one quantitative exposure assessment that's been published by Dr. Wong associated with the -- again, an analysis of a pliofilm factory, which is the basis for most -- is the first epidemiology study demonstrating a quantitative relationship. That suggests an increased risk significantly above 100 parts per million years. I personally think that that may be too high.

The actual pliofilm study, which was originally authored by Dr. Infante and reanalyzed by Dr. Rinsky and coworkers, demonstrated an excess risk on the order -- that was significant, on the order of 60 ppm-years.

Q. When did a consensus develop among

the medical and scientific community that benzene was a human carcinogen?

MR. BROWN: Object to form.

A. The relationship between benzene and -- benzene as a carcinogen, which specifically relates to acute myeloid leukemia,

was the subject of controversy for a long time, primarily because there were no quantitative studies, and the appreciation that something that could cause bone marrow suppression could also cause leukemia really was not a generally held medical or scientific theory until the -- until we had sufficient experience with chemotherapeutic and radiation treatment in the therapy of other cancers. This occurred -- these studies began to appear in the '60s, in the late '60s, and a consensus with respect to a defined quantitative risk of AML associated with benzene occurred in the mid to late 1970s.

Q. (BY MR. SCOTT) When did the U.S. government first conclude that benzene was a cause of acute myeloid leukemia?

MR. BROWN: Object to form.

A. If by that --

Q. (BY MR. SCOTT) Let me change my

question because it's not -- it's a good objection. When did the U.S. government or any agency of the U.S. government first publish anything that -- in which the conclusion that benzene was a cause of acute myeloid leukemia was published?

MR. BROWN: Object to form.

A. As I sit here, I'm not sure I recall the specific date, but I think it was on or about 1980. I could be wrong.

MR. BROWN: Object to responsiveness.

Q. (BY MR. SCOTT) What does -- are you familiar with the ATSDR?

A. Yes.

Q. What is that?

A. It's a -- it's basically a document put out by the federal government that summarizes the knowledge with respect to health effects, what's known about the adverse effects of chemicals and specifically benzene.

Q. And it is not independent research; is that correct? The ATSDR on benzene, for instance, is not independent research?

MR. BROWN: Object to form.

A. No, there's no original research. It's a compilation of studies and a summary of the official policies of government and various agencies relating to benzene and its toxicity. So it provides a summary of literature as well as summarizes the regulatory policies that

relate to benzene.

Q. (BY MR. SCOTT) I want to turn now specifically to the case of Mr. Oliver McCarty. What opinions have you reached regarding the -- whether or not there is evidence, sufficient evidence to conclude, based upon the scientific literature and the information you've received about Mr. McCarty, as to whether or not benzene exposure was a cause of his MDS and acute myelogenous leukemia?

A. Based upon my -- excuse me -- based on my review of the data made available to me in terms of Mr. McCarty's alleged exposure to benzene, as well as my review of the medical records and the diagnosis of his disease, it's my opinion that there is insufficient evidence to conclude that in fact his disease was caused by exposure to benzene.

Q. Now, Mr. Brown asked you some

questions related to your conversation with Mr. Plisko earlier this week. Had you reached the conclusions which you have just stated prior to your conversation with Mr. Plisko?

MR. BROWN: Object to form.

A. Yes, based upon the other factors

that I've discussed today that I evaluated with respect to Mr. McCarty's disease.

Q. (BY MR. SCOTT) You have reviewed the deposition of Frank Parker. Did Mr. Parker do a quantitative exposure assessment for Mr. McCarty?

A. Not in any of the documents that I've read.

Q. That would include his deposition; is that correct?

A. That's correct.

Q. What is the incidence of AML in the general population, U.S. population, just give or take?

A. It's been a while since I looked at that.

Q. Sure.

A. In the general population it's less than -- I believe it's less than ten per

100,000, but I have not reviewed that. I haven't reviewed certainly any recent incidence literature on that.

Q. Where would you look -- if, for instance, we were interested in how many cases of acute myeloid leukemia, new cases of acute

myeloid leukemia we would see in the United States this year, where would you look for that information?

A. Basically what's referred to in the trade as the SEER data, SEER study, which is a compilation of incidence data provided by -- provided from around the country that I believe is sponsored by the U.S. government.

Q. Is it true that the vast majority of cases of acute myeloid leukemia in this country are -- occur to people who have no occupational exposure to benzene?

MR. BROWN: Object to form.

A. Yes.

Q. (BY MR. SCOTT) Where would one look for that sort of information?

A. If you look at the -- basically if you look at the incidence of AML in the general population, the vast majority are referred to as

de novo, which means no -- no known cause or etiology. That -- there are two different types in general: those that are de novo and those that are referred to in general as secondary.

Secondary means that there's some association with an etiology that's known -- a

cause, an exposure or an agent that has been associated with the -- previously with the development of AML.

The most frequently encountered secondary AMLs are associated with chemotherapy, with radiation. That constitutes the vast majority of secondary AMLs that are reported in the United States. The incidence of AML related to benzene exposure is most reliably determined by looking at the quantitative epidemiology studies that have been published on the subject.

Q. You mentioned earlier a report from IMPATH Labs related to the observance of multiple ring sideroblasts. What is the significance of ring sideroblasts with regard to Mr. McCarty's case and your opinions?

A. Ring sideroblasts are a morphologic feature that is associated with certain forms of myelodysplastic syndrome, primarily those

associated with, if not exclusively, de novo MDS, MDS not associated with secondary exposure. And certainly in the case of benzene, previous descriptions of MDS and AML that have been associated with benzene don't provide evidence of the presence of ring sideroblasts, which is,

I think, significant in that ring sideroblasts are very frequently -- refractory anemia with ring sideroblasts and the presence of ring sideroblasts in MDS are very frequently found in the United States.

Q. Let me -- and I don't mean this in a critical way at all, but just to see if I can put it in plain English so I will understand it -- is that the same thing as saying that in the studies in which people who have been exposed to benzene have been observed to develop myelodysplastic syndrome, that the scientists have not observed ring sideroblasts?

MR. BROWN: Object to form.

Q. (BY MR. SCOTT) If I've said it wrong -- I'm trying to get this into more plain language.

A. Yes. I specifically don't know of any cases that have been reported, and if

they -- if there are cases, they are vanishingly few.

MR. BROWN: Object to responsiveness.

Q. (BY MR. SCOTT) In opposition to that, in those cases where people with

myelodysplastic syndrome -- who have developed myelodysplastic syndrome have no known occupational exposure to benzene, is the finding of ring sideroblasts somewhat common?

A. Yes.

Q. I think the last question I have is, you mentioned, and I think you said 3,000 cases of -- that the Shanghai health study or JCML Lab had seen of some lymphohematopoietic cancer or MDS. How many of those cases have you personally been involved in the evaluation of, in the diagnosis of?

MR. BROWN: Object to form.

A. All of them.

Q. (BY MR. SCOTT) What is your involvement in that -- Mr. Brown asked you questions earlier about whether you could diagnose disease. How does that fit with what you just told me about your involvement in the

JCML evaluations?

A. In China, in JCML, I'm director of the laboratory, both clinical and the research arms of the laboratory. I'm responsible for the overall management and I personally sign out cases based on my evaluation of the clinical

laboratory data and the histopathology.

Q. I believe that's all I have.

MR. BROWN: You got one more minute left.

THE VIDEOGRAPHER: The time is approximately 6:11 p.m., and we are now off the record.

(Discussion off the record.)

THE VIDEOGRAPHER: The time is approximately 6:15 p.m. and we are back on the record.

EXAMINATION

BY MR. BROWN:

Q. Dr. Irons, is it impossible to have a benzene-related AML where there is some ring sideroblasts present, even when there is a myelodysplastic syndrome transformation to AML with a loss of chromosome 5?

MR. SCOTT: Object to the form of

the question.

A. I'm sorry. Could you repeat that.

Q. (BY MR. BROWN) Yes, sir. Is it impossible to have a benzene-related AML where there are some ring sideroblasts present even when there is a myelodysplastic syndrome

transformation into AML with a loss of chromosome 5?

MR. SCOTT: Object to the form of the question.

A. I can't say that anything is impossible. As a scientist I can't say that. I can't prove a negative, as I've said before. What I -- what my opinion is, is that there are no cases that I know of in which those criteria are met.

THE VIDEOGRAPHER: Doctor, would you please wear the microphone.

THE DEPONENT: Sorry.

Q. (BY MR. BROWN) All right. So that we get your answer good on the tape, you can't say it's impossible, correct?

A. I can't say -- as a scientist I can't offer an opinion that anything is impossible. I can't say that it's impossible.

My opinion is based upon the fact that I'm not aware of any cases in which those specific features are present in a case that's been previously associated with benzene exposure.

Q. Are you aware of any studies out there that say if you have ring sideroblasts,

there is no way that the leukemia or myelodysplastic syndrome can be related to benzene exposure?

MR. SCOTT: Object to form of the question.

A. As I said, I have no -- I can't say that it's impossible. There are no studies that say -- there are virtually no studies that say anything is impossible. That's not the nature of scientific inquiry.

Q. (BY MR. BROWN) Are there any studies that address ring sideroblasts being present in AML and whether or not benzene is a particular cause of it?

A. Only by its absence as a feature described in the cases.

Q. So specifically there is no study, correct?

A. No, that would be -- that's

virtually unheard of.

Q. You said you reached your conclusions and opinions about Mr. McCarty's disease and its cause prior to your conversation with Mr. Plisko, correct?

A. I reached -- yes, I did.

Q. That's what you testified to Mr. Scott just now?

A. That's correct.

Q. So you didn't need any type of exposure assessment by anyone to reach your conclusions and opinions in this case, did you?

A. That's not -- that's not a fair description of my testimony or my opinions.

Q. Sir, let me just ask you. You said --

MR. SCOTT: Let him finish, please.

Q. (BY MR. BROWN) You said you didn't need Mr. Plisko's quantitative exposure assessment to reach your opinions, correct?

MR. SCOTT: Object to the form of the question.

A. I testified that I had reached my conclusions based -- with respect to the likelihood that Mr. McCarty's AML was associated

with benzene exposure based on the evidence that I had prior to my discussion with Mr. Plisko. Subsequent to that discussion, I also am relying on that quantitative exposure assessment. But prior to that, in the absence of a quantitative exposure assessment, I had reached an opinion

based on the nature and the characteristics of Mr. McCarty's disease.

Q. (BY MR. BROWN) Let me object to responsiveness. You reached your conclusions and opinions about Mr. McCarty's disease and its cause prior to your conversation with Mr. Plisko where he gave you a quantitative exposure assessment on Mr. McCarty's benzene exposure, correct?

A. Yes, that's correct.

Q. And so by common sense, anybody can see you didn't need Mr. Plisko's exposure assessment for Mr. McCarty to form your opinion that Mr. McCarty -- that his AML and his myelodysplastic syndrome were not related to exposure to benzene, correct?

A. In the absence of a quantitative exposure assessment, I have an opinion with respect to the characteristics and features of

Mr. McCarty's disease and whether or not the literature related to exposure to benzene is associated with those characteristics.

Independent of that and in addition to that, I have an opinion with respect to the likelihood -- the same likelihood that includes

a quantitative exposure assessment. But in the absence of an exposure assessment, I'm still prepared to offer an opinion with respect to the characteristics and features of Mr. McCarty's disease and its presentation.

Q. That's the same as saying you don't need Mr. Plisko's notes or the handwritten notes you made from your conversation or anything he told you to come up with your opinions about Mr. McCarty and his disease, correct?

A. I have an opinion with respect to the likelihood of Mr. McCarty's disease in the absence of a quantitative exposure assessment and in the presence of a quantitative exposure assessment. It's the same opinion, but it's based on those different pieces of information.

Q. There was some discussion about your work in the area of pathology. Let me ask you, sir, in the United States if you signed off on a

diagnosing -- or a diagnosis of a condition based on a bone marrow chromosomal study or analysis, for instance, would you be in violation of the laws that prohibit the unlicensed practice of medicine?

A. Could you repeat the question.

Q. Yes, sir. If you signed off on a diagnosis of a patient who had an abnormal morphology study or chromosomal analysis, you would be in violation of the laws of the United States that prohibit the unlicensed practice of medicine, correct?

MR. SCOTT: Object to form of the question.

A. If I did?

Q. (BY MR. BROWN) Yes.

A. Yes.

Q. Are you a nosologist?

A. No.

Q. There's some confusion I had about -- I think it was paper 5 where you were asked how many lymphohematopoietic cases in Shanghai had you reviewed to date, and I think your answer was about 3,000, 3,200 cases; is that correct?

A. To date, yes.

Q. All right. And then, sort of comparing apples and oranges, you were asked about the other cohort by I think Mr. Yin, Mr. Hayes, and said that that was about 33,000 total in the cohort, correct?

A. My study is not a cohort study.

Q. I understand.

A. Well, you said other cohort. It's not a cohort study.

Q. You said the cohort in the other study was a 33,000-member cohort.

A. The Yin study has a cohort. It's a different study design. It has a total of 33,000 individuals, not 33,000 cases of disease.

Q. I understand that. What I wanted to get to is, you weren't asked by Mr. Scott about how many total lymphohematopoietic cases there were in that 33,000 cohort; you were only asked how many cases of NHL, AML, and myelodysplastic syndrome. Can you tell me how many cases of lymphohematopoietic disease were in the cohort of 33,000?

A. Total?

Q. Yes.

A. As I sit here, I can't recall specifically. I can't recall specifically. It may be twice as many. I don't recall.

MR. SCOTT: Twice as -- I'm sorry.

Q. (BY MR. BROWN) With regard to your opinions on the minimal -- or the minimum level

at which somebody would develop AML as a result of benzene exposure, and you've said 45 ppm-years is the lowest number you've heard of -- is that correct?

A. That's the lowest level for which I believe there is quantitative epidemiology to support an increased risk associated with AML.

Q. All right. Have you seen, and I know you have, the Australian study which says that levels below 5 parts per million have resulted in leukemias?

A. I don't believe that that is a fair conclusion. I have seen the Australian Healthwatch study and I provided the most recent publication in my production here. I don't believe that's an accurate characterization of the results of that study.

Q. What is your understanding of the results of that study in terms -- when it says

levels below 5 parts per million was associated with causing leukemia?

A. I don't believe that I can reach that conclusion. I have here the most recent publication and summary of the update of that study and the conclusions that are reached in

it. And the final conclusion of the authors is that it's uncertain whether benzene exposures, particularly past levels of exposure, have been high enough to cause acute nonlymphocytic anemia, which is AML.

Q. There is nowhere in the study where you could discern or detect that the authors were suggesting that exposure below 5 parts per million were contributing to leukemia or other cancers of the blood or hematopoietic system?

A. This is the most recent publication from this group and constitutes the current conclusions of the authors with respect to the sum total research conducted in that study. At the present time there -- that's the conclusion that the authors have reached.

Q. That's an Exxon-funded study, correct?

A. I couldn't tell you. I believe that

the Australian Healthwatch is supported by a variety of different sponsors, but I couldn't tell you specifically whether Exxon is supporting them or not.

Q. When you testified in the case in 2003, do you remember testifying that it was an

Exxon-funded study?

MR. SCOTT: Object to the form of the question.

A. No, and I don't believe at the time I -- at this point, I don't recall knowing specifically whether it was Exxon-funded or funded by someone else. I believe that it probably says somewhere in this document what its support is.

Q. (BY MR. BROWN) It should, shouldn't it?

A. It's supported by the Australian petroleum industry, which I would conclude probably includes Exxon, but doesn't say specifically.

Q. If the study did in fact find originally that leukemias were found in excess at exposure levels below 5 parts per million, that would have been a finding that Exxon and

the other members of the petroleum industry in Australia would not have liked, correct?

MR. SCOTT: Object to the form of the question.

A. I can't speak for any particular company or any individual in any particular

company with respect to what they would have liked or disliked.

Q. (BY MR. BROWN) Well, you've got common sense, don't you, sir? Industry, companies don't want there to be publications out there finding that excess leukemias at low levels because it causes them to spend money, doesn't it?

MR. SCOTT: Object to the form of the question. Come on, this is just argument and harassment, Darren. This isn't an expert subject.

MR. BROWN: Yes, it is.

MR. SCOTT: Object to the form of the question.

A. I believe that's very overly simplistic and not realistic. I think by the same token that a company may not -- or any individual in the company may not be happy to

hear that exposure to a given chemical at a given level causes a disease, I think that responsible individuals within any company want to know what in fact the relationship is between exposure to an agent or a chemical or a workplace condition and the development of the

disease.

Q. (BY MR. BROWN) And money has nothing to do with it in your opinion; is that correct?

MR. SCOTT: Object to form of the question.

A. Money has nothing to do with it as far as I'm concerned, in my opinion.

Q. (BY MR. BROWN) This is not actually an update of the Australian Healthwatch study; that's a completely different study; isn't it, sir?

A. No, this is the same group, I believe. It's defined as Healthwatch, Australian Healthwatch.

Q. You are citing the update of "Mortality and Cancer Incidence in the Australian Petroleum Industry Cohort" by R.T. Gunn, correct?

A. Yes.

Q. It's your testimony under oath that that is an update of the original Australian study?

MR. SCOTT: Object to form of the question.

A. I'm reading what it says. It says

here, "The Australian petroleum industry surveillance program Healthwatch was established in 1980," and then goes on to describe the history of the cohort and that this is an update.

Q. (BY MR. BROWN) You agree that the Yin study differs with you in terms of what the lowest required levels of exposure are to develop leukemia, correct?

MR. SCOTT: Object to form of the question.

A. Specifically if by the Yin study you mean the studies published in collaboration between the Chinese and the National Cancer Institute, it's typically referred to in terms of the exposure assessment provided in the Hayes analysis, no. I believe that the Hayes study provides -- if you take the Hayes study on face value, it provides evidence of a significant

excess risk above 45 ppm-years.

Q. (BY MR. BROWN) It also says that excess leukemias were found at levels of exposure below 10 parts per million, correct?

A. It refers to the findings of individual cases for which were characterized

average exposures. Again, an average exposure is not a quantitative exposure or a time-related average and includes, certainly within a description, individuals who may have been exposed at much higher levels.

Q. Sir, with regard to the amount of work that you have done over the last 15 or 16 years serving as an expert for defendant oil companies and chemical companies in litigation, with regard to your history of working at the Chemical Industry Institute of Toxicology which was funded by industry, do you feel like you have any conflict of interest whatsoever in doing the Shanghai study, particularly when you have testified, as has Otto Wong, time and time again that benzene exposure only at high levels can cause certain diseases?

MR. SCOTT: Object to form of the question.

A. I don't believe that characterizes my opinions or my testimony. And I certainly believe that my publication record speaks for itself, and the design and conduct of the Shanghai health study is independent of the sponsors, as is required by U.S. and Chinese law

and has been adequately documented.

Q. (BY MR. BROWN) How much have the sponsors, the five petroleum company sponsors, contributed to the study thus far?

A. Total, which is not -- which is not just the studies that I'm responsible for in the laboratory, I believe is probably on the order of $23,000,000. Specifically I believe the clinical and research aspects of the study that I'm responsible for, the overall budget has been on the order of 19,000,000.

Q. Per year?

A. No. Total.

Q. And it's been going on since 2000, correct?

A. Began -- began -- design and laboratory construction began in 2000, 2001.

Q. And who are the five industries or five companies again who are the contributors to

the study?

A. Sponsors are Exxon-Mobil, Chevron, British Petroleum, Conoco-Phillips, Shell Chemical.

Q. Have they -- in addition to contributing money to your efforts, have they

also contributed money, to your knowledge, to Fudan University in China or the university here where you work?

A. Those are all the same.

Q. So the $23,000,000 is total and there's not been one more dime contributed to the efforts in the study; is that correct?

A. Not to my knowledge.

Q. Who else has contributed money besides these five entities?

A. Those are the entities that are supporting this project, and they are the only entities supporting this project.

Q. This is an industry-funded project then, isn't it, sir?

A. It's an industry-funded project, yes.

Q. Do you or the API have any plans to include the National Cancer Institute or NIOSH

in the study?

MR. SCOTT: Object to form of the question.

A. The American Petroleum Institute is not -- is simply an administrator in this project and doesn't have a management or

sponsoring role. I have no intentions of including the National Cancer Institute in the conduct of this project, any more than I would include anyone else at this stage.

Q. (BY MR. BROWN) Well, wouldn't you agree it would take away some of the -- what might be a perceived bias if the National Cancer Institute or NIOSH were included in this study?

MR. SCOTT: Object to form of the question.

A. I think that's an outrageous, outlandish idea. To my knowledge, the National Cancer Institute has neither the interest nor the expertise to conduct a study such as this, nor do they have an interest in providing clinical services to the City of Shanghai.

This study has a totally different design and totally different purpose and, to my understanding, focus than the NCI study, which

was a classic retrospective, historical epidemiology study. This is a prospective study of the pathogenesis of disease in the hematopoietic system. A relatively small part of it is associated with or deals with potential exposure to benzene.

Q. (BY MR. BROWN) Sir, wouldn't you agree that it would be best for you and those who are doing this study that if there was an appearance of no influence by the companies who are funding your study or appearance of impropriety or bias in that study?

MR. SCOTT: Is there a question? I'm sorry. Object to form.

A. Is there a question?

Q. (BY MR. BROWN) Yes. Wouldn't it be best for you in your efforts if there were an appearance of no influence by the companies who are funding your study or an appearance of no potential bias because of oversight or by way of oversight from the National Cancer Institute, NIOSH, or some other governmental entity?

MR. SCOTT: Object to the form of the question.

A. I think it's totally meaningless and

ineffective. I think our -- I think that the Shanghai health project as it exists has both the appearance and the reality of being independent from the study sponsors in meeting with U.S. and Chinese law, as well as the ethical guidelines that have been set forth by

the World Health Organization and by the independent committees that oversee the science and oversee the conduct. It's independent of the sponsors, which is consistent with any other clinical research project in the United States and most conducted worldwide.

(Irons Deposition Exhibit 8 was marked.)

Q. (BY MR. BROWN) I want to show you what we've marked as Exhibit 8 to your deposition and ask if you've ever seen that document before.

A. No.

Q. All right. I will represent to you that this is a document produced by Monsanto in another litigation, and it looks like at the top there is a Craig M. Parker. Does the name Craig M. Parker -- do you know Craig Parker?

A. No.

Q. In any event, it looks like it's a PowerPoint presentation about the proposed studies on the risks of benzene-induced diseases in China; that's your study, isn't it?

A. It's not mine. I've never seen it.

Q. Well, is it referring to your study?

A. I don't know whether it is specifically or not. That's not the title of my study, and I have not seen this document before. I certainly didn't produce it.

Q. All right. In any event --

A. Monsanto is not a sponsor.

Q. Well, it's a document that was produced by Monsanto in other litigation. That's what I will represent to you. And people that were -- efforts on the part of those who were contributing to acquire other contributors to assist in funding the study. You are familiar with that, aren't you, sir?

MR. SCOTT: Object to form of the question.

A. In general I believe that the sponsors had discussions with other companies, but specifically I'm only aware of one particular discussion, and that was with Dow

Chemical. I have no knowledge of any conversations or discussions or presentations with Monsanto or any other company.

Q. (BY MR. BROWN) You are not saying that somebody didn't try to recruit Monsanto to be a contributor to help fund the study,

correct?

MR. SCOTT: Object to the form of the question.

A. I'm saying I have no personal knowledge of that.

Q. (BY MR. BROWN) You would agree that it would be wrong, it would be ethically wrong, it would be professionally wrong for somebody to undertake a study where they had already had some expectations of what the results of the study would be, correct?

MR. SCOTT: Object to form of the question.

A. Yes, which is precisely why U.S. regulation and international guidelines for clinical studies ensure the independence of the study conduct and the investigators in clinical studies.

Q. (BY MR. BROWN) Let me object to

everything after "yes." It would be criminal for somebody to rig an epidemiological study to use for their benefit in litigation; do you agree with that?

MR. SCOTT: Object to form of the question. This is just harassing.

MR. BROWN: No, it's not, Bob.

MR. SCOTT: Of course it is. It's ridiculous.

A. You know, I can't -- I'm not an attorney and I don't know whether or not it would be criminal. It would be certainly unethical and reprehensible.

Q. (BY MR. BROWN) All right. Well, here is the reason I'm saying criminal. These epidemiological studies are used to perform risk assessments and set PELs for certain different types of chemicals that are involved in these studies, correct?

MR. SCOTT: Object to form of the question.

A. I'm not -- I'm not at all -- I'm not sure what you are talking about in terms of these epidemiology studies. I have absolutely no knowledge or concern over whether or not the

studies that I'm conducting support regulatory standard setting or not from the standpoint of an investigator. It's totally -- that's not the goal of the study, and that's certainly not my focus in the conduct of the studies.

Q. (BY MR. BROWN) What you are saying

is you just let the chips fall wherever they may and it doesn't matter how much money these companies fund, correct?

MR. SCOTT: Object to the form of the question. It's nonsensical, Darren. What do you mean what he's saying?

A. I don't understand the question.

Q. (BY MR. BROWN) Is it your understanding that with regard to your study, you let the chips fall wherever they may. You design the study, and you don't look back on it, and there is no changes made if a bad result is determined, anything like that?

MR. SCOTT: Object to the form of the question. This is harassing.

A. What I'm saying is the conduct of these studies, their design, the performance of these studies and the results that are published for the study are not in any way related to or

predicated on the basis of what influence they will have on regulation. That's certainly not the focus of me or the investigators.

The focus of these studies is to understand the pathogenesis of these diseases and to provide additional information that may

inform with respect to the specific relationship between exposure to benzene and the development of any of these diseases.

To the extent that that data becomes useful for further regulation or other activities, I think that's a laudable use of it, but it's certainly not the focus or the driving force as far as the conduct of the study is concerned.

Q. (BY MR. BROWN) Well, if your study is used to support higher PELs for dangerous products and people die because of the study that you helped do, and the study was predetermined from the beginning, that would be criminal, wouldn't it, sir?

MR. SCOTT: Object to form of the question. Darren, we are over your six hours.

MR. BROWN: No, we're not.

MR. SCOTT: Yeah, we are.

MR. BROWN: No, we're not.

MR. SCOTT: I'm pretty sure we are. I'm not stopping you, but come on. Let's ask some reasonable questions. Object to form.

Q. (BY MR. BROWN) That would be criminal, wouldn't it, sir?

MR. SCOTT: Object to the form. It's already been asked. It's harassing of this particular witness.

A. That is absurd. Your allegations are absurd. That that in no way characterizes either the conduct or the intent of the study.

Q. (BY MR. BROWN) That somebody would think that the results of the study would be predetermined, that's absurd?

A. Yes.

MR. SCOTT: Object to form of the question.

Q. (BY MR. BROWN) Let me read to you Exhibit 8, what is said here by this PowerPoint presentation with Craig Parker's name at the top. It says --

MR. SCOTT: Can you tell us who that is?

MR. BROWN: It's a safety guy or

some health professional, as I understand it, who worked for Monsanto.

MR. SCOTT: You are vouching for all of his credentials and all of that?

MR. BROWN: Sir, are you going to interrupt my deposition and coach the witness?

MR. SCOTT: I should because you are over six hours, but I'll let you go on.

MR. BROWN: No, I'm not.

MR. SCOTT: Object to form of the question.

Q. (BY MR. BROWN) Sir, he says right here, "The information gained from these studies will assist contributing companies in achieving product stewardship goals and will be useful in defending the industry's risk reduction programs. Additional benefits from this collaboration approach are outlined below." How do you feel about that statement, sir?

MR. SCOTT: Object to form of the question.

A. This statement as you've read to me and as I read it says that the information gained from the studies will assist in -- contributing companies in achieving product --

product stewardship goals and will be useful in defending the industry's risk reduction programs. That to me sounds like a very laudable goal, associated with stewardship and in risk reduction. I don't see where that implies any nefarious or predetermined result.

Q. (BY MR. BROWN) How would somebody know in February of '01 before you even have your results of your study that the information is going to be useful in defending the individual risk reduction programs?

MR. SCOTT: Object to form of the question.

A. I have no idea.

MR. SCOTT: Do you know what the risk reduction programs are, Darren?

Q. (BY MR. BROWN) How would somebody know that, sir?

A. I have no idea.

MR. SCOTT: Object to form of the question.

A. I mean, I would never have said that because -- that particular point in time I certainly wouldn't be convinced that our results would do that or not.

Q. (BY MR. BROWN) Were you aware of these statements made by Dr. Infante in his article published called "The past suppression of industry knowledge and the toxicity of benzene to humans: Potential bias in future benzene research" where he states, "The

PowerPoint presentation apparently sent to Craig Parker, manager of toxicology and product safety, Marathon Oil, states that the planned research results will enhance industry's ability to achieve the above-mentioned objectives and the findings of the Shanghai study are expected to 1 -- or A, provide strong scientific support for the lack of risk of leukemia or other hematological disease at current ambient benzene populations to the general population." Have you ever heard of anything like that before?

MR. SCOTT: Object to form of the question.

A. First time I heard that was when I read that particular article, which is absurd and outlandish.

Q. (BY MR. BROWN) Did you call up Dr. Infante and tell him that?

A. I wouldn't waste my time.

Q. Have you written anybody at the --

at this --

MR. SCOTT: International Journal of Occupational and Environmental Health. Joe LaDou is the editor.

Q. (BY MR. BROWN) Have you written

anybody for that organization, sir?

MR. SCOTT: Object to form of the question.

A. No.

Q. (BY MR. BROWN) I mean, what you're saying is -- I mean, Dr. Infante is making a serious allegation against you and your study, correct?

A. Dr. Infante has made a number of statements there that -- most of the statements I disagree with, and I certainly don't -- I don't have any knowledge that -- or personal knowledge that indicates that they are true. As a matter of fact, I think they are not true. And I think that there are many statements in there that are unfounded and certainly misrepresent my understanding of what I'm doing and what the study is about.

Q. Well, and I'm not -- I don't want to

imply that you are doing anything wrong. What I want to get to the bottom of is if you've got a study where industry expects the results of the study are going to be favorable to them before the study is even complete, that is just simply wrong, correct?

MR. SCOTT: Object to form of the question.

A. Yes, that would be wrong. And that not the case in the Shanghai health study.

Q. (BY MR. BROWN) And even though you disagree with what Dr. Infante says, you've never written anybody for the journal or never written Dr. Infante or published any written document to respond to what he said, correct?

A. No. As I said, I wouldn't waste my time.

Q. That's a pretty serious allegation, isn't it?

MR. SCOTT: What's a serious allegation?

Q. (BY MR. BROWN) That there is -- that's a pretty serious allegation, isn't it, sir?

MR. SCOTT: What -- object to form

of the question.

MR. BROWN: Okay. Object to the form.

Q. (BY MR. BROWN) The ones that I just read to you about that Dr. Infante said.

A. As I have said before and I will say

it again, they are absurd and outlandish, and to my knowledge they are totally unfounded.

Q. Give me one minute.

MR. BROWN: How much time do I have left?

THE VIDEOGRAPHER: On this tape, approximately 25 minutes.

MR. BROWN: No, I'm talking about there is a six-hour limitation. How much time have I used?

THE VIDEOGRAPHER: You are right in that six hours.

MR. BROWN: Okay.

THE VIDEOGRAPHER: I didn't know the exact time that Mr. Scott started asking.

MR. BROWN: I will pass the witness.

MR. SCOTT: I just have a couple of follow-up questions.

EXAMINATION

BY MR. SCOTT:

Q. First, I want to understand this discussion of how many cases the NCI China study has observed of lymphohematopoietic cancers as compared to those that the Shanghai health study has seen. You said twice as many, but I'm not

sure what that related to. Can you describe how many cases you all, your study, has seen and how many NCI has seen.

A. My understanding of the question was how many cases of other hematopoietic disease were reported in the overall series of studies published by Hayes and all, and that includes a variety of different publications by Travis, Linet, Hayes, Yin, and what have you. I -- as I sit here, I recall that the overall list of abnormalities and diseases was more than the number of cases of AML or NHL. That's what I was referring to.

Q. And how many cases of AML and NHL were there approximately in the NCI China study?

A. There were 14 cases of NHL, three of which were confirmed by pathologic diagnosis. I believe there were on the order of 30 and then a few cases of AML, some of which were confirmed

by histopathologic diagnosis and some that weren't.

Q. How many of the cases that the Shanghai health study is -- has evaluated have been confirmed by histopathology -- describe for us the process that the Shanghai health study

goes through to diagnose these cases and what it includes.

A. After a patient presents at Shanghai Hospital, the laboratory performs sampling including biopsy, analysis of blood, bone marrow, and then performs a series of molecular diagnostic studies in keeping with -- in strict adherence to the WHO criteria for classification. Histopathologic and hematologic, morphologic review is performed. Cytogenetic analysis and molecular genetic analysis is performed. A diagnosis is made typically by two independent morphologists and then integrated into a study diagnosis, sometimes three.

Each of those studies is then independently peer-reviewed by a board-certified hematopathologist from the United States, who for study purposes reviews the cases. And then

additional tertiary review is applied in selected cases involving a third expert in the United States.

MR. BROWN: Object to responsiveness.

Q. (BY MR. SCOTT) How does that compare

with the evaluative techniques that were utilized by the NCI kind of study?

A. There is virtually no molecular or phenotypic or cytogenetic analysis performed in those studies. Some of the diagnoses were anecdotal or verbal, involving no documented laboratory or pathology analysis. The criteria that were used in the diagnosis of those cases were not standardized, which is a problem in China. A relatively small number were available for independent histopathologic review in the United States and have been reported in those studies, as I said, three out of 14 lymphomas exposure group. A larger number of AMLs I think were -- there was material available for review.

Q. I want to -- with regard to this editorial of Peter Infante in the International Journal of Occupational and Environmental Health, I notice on the editorial board of that

journal -- the following individuals sit on that editorial board: Peter Infante, Barry Levy, and Dan Teitelbaum. Are you familiar with those men?

A. Yes.

Q. Are you familiar with whether or not

every one of those men has testified on multiple occasions for plaintiffs in cases involving benzene?

A. In cases that I have been a consultant on, they are frequently involved as consultants.

Q. When you read Dr. Infante's editorial in the International Journal of Occupational and Environmental Health did you notice whether he disclosed that he makes a lot of money testifying for plaintiffs in benzene cases?

A. What I noticed was there are no -- there are no statements of potential conflict of interest or financial support, and yet that's a preeminent policy of the journal, at least stated in their policies.

Q. That's all I have. Thank you.

MR. BROWN: I don't have any further

questions.

THE VIDEOGRAPHER: This includes the August the 29th deposition of Dr. Richard Irons. The time is approximately 6:55 p.m. and we are now off the record.

CORRECTION PAGE
WITNESS: RICHARD D. IRONS, Ph.D. August 29, 2006

PAGE	LINE	CHANGE	REASON

20 _____

21 _____

22 _____

23 _____

24 _____

25 _____

SIGNATURE PAGE

I, RICHARD D. IRONS, Ph.D., have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted on the correction page.

_____

RICHARD D. IRONS, Ph.D.

THE STATE OF TEXAS)
COUNTY OF JEFFERSON)

Before me, _____, on this day personally appeared RICHARD D. IRONS, Ph.D., known to me (or proved to me under oath or through _____) (description of identity card or other document) to be the person whose name is subscribed to the foregoing instrument and acknowledged to me that he/she executed the same for the purposes and consideration therein expressed.

Given under my hand and seal of office this _____ day of _____, 2006.

_____

NOTARY PUBLIC IN AND FOR
THE STATE OF _____
My commission expires: _____

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CAUSE NO. E-170.351

IN THE DISTRICT COURT OF JEFFERSON COUNTY, TEXAS, 172ND JUDICIAL DISTRICT

YOLANDA MCCARTY ROBERTS, ET AL
VS.
SHELL OIL COMPANY, ET AL

REPORTER'S CERTIFICATION
DEPOSITION OF RICHARD D. IRONS, PH.D.
TAKEN AUGUST 29, 2006

I, Joanne Blair, Certified Shorthand Reporter in and for the State of Colorado, hereby certify to the following:

That the witness, RICHARD D. IRONS, Ph.D., was duly sworn by the officer and that the transcript of the oral deposition is a true record of the testimony given by the witness;

That the deposition transcript was submitted on September 8, 2006, to the witness or to the attorney for the witness for examination, signature and return to Esquire Deposition Services, L.L.P., by October 6, 2006.

That the amount of time used by each party at the deposition is as follows:

Darren Brown..... 5 HOURS:38 MINUTE(S)
Robert Scott.....01 HOURS:01 MINUTE(S)

That pursuant to information given to the deposition officer at the time said testimony was taken, the following includes counsel for all parties of record:

Darren Brown - Attorney for Plaintiff
Rodney Barnwell - Attorney for Plaintiff
Robert Scott - Attorney for Defendant
Univar U.S.A., Inc.

I further certify that I am neither counsel for, related to, nor employed by any of the parties or attorneys in the action in which this proceeding was taken, and further that I am not financially or otherwise interested in the outcome of the action.

Further certification requirements pursuant to Rule 203 of TRCP will be certified to after they have occurred.

Certified to by me this 5th of September, 2006.

JOANNE BLAIR
Esquire Deposition Services
303 East 17th Avenue, Suite 565
Denver, Colorado 80203
(303) 316-0330

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FURTHER CERTIFICATION UNDER RULE 203 TRCP

The original deposition was/was not returned to the deposition officer on _____;

If returned, the attached Changes and Signature page contains any changes and the reasons therefor;

If returned, the original deposition was delivered to Darren Brown, Custodial Attorney;

That $_____ is the deposition officer's charges to the Plaintiff for preparing the original deposition transcript and any copies of exhibits;

That the deposition was delivered in accordance with Rule 203.3, and that a copy of this certificate was served on all parties shown herein on and filed with the Clerk.

Certified to by me this 6th day of September, 2006.

_____

JOANNE BLAIR
ESQUIRE DEPOSITION SERVICES
303 EAST 17TH AVENUE, SUITE 565
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