CAUSE NO. 17958-JG01

JAMES BOREN AND GINGER BOREN	)	IN THE DISTRICT COURT OF
VS.	)	BRAZORIA COUNTY, TEXAS
THE DOW CHEMICAL COMPANY, ET AL.	)	239TH JUDICIAL DISTRICT

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ORAL DEPOSITION OF
GERHARD K. RAABE, MD
OCTOBER 6, 2004

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ORAL DEPOSITION OF GERHARD K. RAABE, MD, duly sworn, produced as a witness at the instance of the Plaintiff, was taken in the above styled and numbered cause on the 6th day of October, 2004, from 10:05 a.m. to 4:12 p.m., before Valerie B. Corrales, CSR, Certified Shorthand Reporter in and for the State of Texas, reported by machine shorthand, at the offices of THOMPSON & KNIGHT, LLP, 333 Clay Street, Suite 3300, Houston, Texas, pursuant to the Texas Rules of Civil Procedure and the provisions stated on the record.

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

FOR THE PLAINTIFFS:

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EXHIBITS

(RAABE Exhibit No. 1 was marked.)

GERHARD K. RAABE, MD,

having been first duly sworn, testified as follows:

EXAMINATION

QUESTIONS BY MR. LUBEL:

Q. Please state your full name.

A. Gerhard Karl Raabe.

Q. Where do you reside?

A. New Hope, Pennsylvania.

Q. Who hired you in this lawsuit?

A. Mr. Shoebotham.

Q. On behalf of what company does he represent?

A. Dow.

Q. Dow Chemical Company?

A. I'm not sure of the specifics of that.

Q. Have you ever heard of Dow Chemical Company?

A. Yes.

Q. Have you ever done any work for them?

A. Not that I recall.

Q. And I'm not talking about just in a lawsuit capacity. I'm talking about have you ever done any consultation or consulting work with them?

A. I don't think so.

Q. Do you -- do you know anybody that's employed by Dow Chemical Company?

A. Yes.
Q. Who are they?
A. Currently employed?
Q. Yes, sir.
A. Jim Collins, Greg Bond -- but I have not seen him in many years. I think he's still employed.
THE REPORTER: You think he is what?
THE WITNESS: I think he's still employed. I'm sorry.
A. That's -- that's what comes to mind as we speak.
Q. (By Mr. Lubel) Are there any people that you do not believe are still working for Dow Chemical that you've had relationships with in the past?
A. Yes.
Q. Who are they?
A. Dr. Ralph Cook. That's the one that comes to mind.
Q. How do you know Jim Collins?
A. I've known Jim for a number of years. He was considering taking a position with Mobil at one time. And I believe he went to Monsanto. And I knew him when he was Director of Epidemiology at Monsanto. And we've continued our relationships over the years.
Q. Have you served on any committees or subcommittees with Mr. Collins?
A. Yes.

Q. Which committees or subcommittees?
A. Dr. Collins and I were both on the American Industrial Health Council's Epidemiology Committee. And we -- we both are fairly active in the American College of Epidemiology. So I see him there periodically, but we're not on any committees together.
Q. Have you ever seen Dr. Collins at any American Petroleum Institute or CMA meetings?
A. No API meetings. I don't think so at CMA.
Q. Any other industry organizations or trade groups for which you recall seeing Dr. Collins at?
A. Yes, one other. We -- there was a group that we had in the '80s called the Epidemiology Forum, and we participated in the some of those meetings.
Q. Epidemiology Forum?
A. Yes.
Q. Is that group still together?
A. Don't actually know. It was an ad hoc group that would meet about once a year.
Q. Was it petroleum or petrochemical company people?
A. It was epidemiologists that worked for essentially industry, not particularly any industry -- specific industry.
Q. When is the last time you've spoken to Dr. Jim Collins?
A. I -- probably -- the most recent time was probably

fall of 2003.

Q. What was the nature of that visit?

A. We had dinner together during one of the American College of Epidemiology meetings.

Q. Was the dinner that you had business related or purely personal?

A. No, just personal.

Q. Have you had any conversations with Dr. Collins about the benzene studies in China?

A. I did ask him about that at dinner, whether Dow was participating.

Q. Is that the extent of y'all's conversation about that?

A. Yes.

Q. So you would not have shown the POWERPoint presentation regarding the benzene China studies to Dr. Collins. Correct?

A. No.

Q. Is that correct?

A. Yes, that's correct.

Q. Nor any other Dow people?

A. Not -- yes. No other Dow people that I'm aware of.

Q. So the only communication between, at least, yourself and any Dow people regarding the Chinese benzene studies was the few moments that you spoke to Dr. Collins at that dinner

about it. Correct?

A. Yes.

Q. And at least nobody on your behalf nor did you send any written communications to anybody at Dow concerning the benzene studies in China. Correct?

A. Not that I'm aware of.

Q. Is Dow Chemical participating in any form or fashion in the benzene studies in China?

A. I don't know what the current status is. They weren't when -- as of the time we spoke.

Q. Did Dr. Collins give you any indication as to why Dow was not participating when y'all had that dinner?

A. I'm not sure if he knew.

Q. Did he voice any concerns or objections to you about the design of those studies?

A. No. We didn't talk about them that long.

Q. Your conversation with Dr. Collins -- or conversations, plural, I should say -- did not go to the extent of getting into the actual study design, those types of things. Correct?

A. No, not at all.

Q. Are you familiar with the study design?

A. No, sir.

Q. The Chinese study.

A. Basic -- the basic concepts that were being discussed

in 1999 but not since then.

Q. When were you first approached about the industry-funded Chinese benzene studies?

MR. SHOEBOTHAM: Objection, form.

A. It was in -- first approached? I'm not sure if...

Q. (By Mr. Lubel) How did you become introduced to the fact that they were considering the studies?

A. Well, during that period of time, I was actively involved with the American Petroleum Institute on their health and safety committee. We were interested in what were the research opportunities that could provide more information on health effects of benzene. And that was an evolutionary process that went on several years before China became more focussed.

Q. Who started the discussions about there needing to be a study in China on benzene funded by industry?

A. I don't think there was any single individual. I think the group of industry toxicologists and epidemiologists and other professionals trying to explore is there any place in the world where you can do additional research to better understand the health benefits.

Q. And you recall our -- generally our discussion when I deposed you in the James Cowey case, I believe, in -- around December of 2003. We had a brief discussion about the Chinese benzene studies. Correct?

A. Yes.

Q. And at that time you had told me that one of your responsibilities for the American Petroleum Institute was to go out and show different companies a POWERPoint presentation regarding the studies. Correct?

A. I think that somewhat misstates what I said or at the very least what I -- what I -- what I meant. I gave -- as chair of the committee, once we decided on the approach, I gave what I believe are two presentations at API meetings that had -- had industry management types of different levels present.

Q. Who prepared the POWERPoint presentations that you're referring to now?

A. Well, it was just essentially one presentation. And part of it, I took input from Dr. Wong and Dr. Schnatter and possibly even Dr. Irons. And collectively we came up with a set of slides that described the general thoughts of what we were trying to accomplish.

Q. And this was presented to the executives that had appeared on behalf of the industry at the meeting or presentation?

A. I don't remember the levels of the executives, but I think that's probably true. They were API meetings. It would have been petroleum representatives.

Q. And when were those present -- when was that presentation made?

A. I don't know the -- the months. Probably 1999.
Q. Who are the stakeholders regarding the benzene Chinese -- China studies?
A. I don't understand what you mean by stakeholders.
Q. You've never seen the term "stakeholders" referenced in the documents of the committee that is responsible for the industry-funded Chinese studies regarding --
A. I've never seen that term used, no.
Q. Who are the companies that have funded those studies to date?
A. I don't know the current list. I -- at the time all I can tell you is -- is who -- who were heading -- heading to funding when I left the industry.
Q. Who was that?
A. Exxon-Mobil, Shell, Chevron. I think those are the only ones that, at the time I was leaving, had probably even then not made formal commitments but seemed to be committed to funding some portion.
Q. How about BP? Were they part of that group?
A. Don't recall.
Q. And did you have conversations with Patrick Beatty about these studies?
A. Well, Patrick was a member of the health products stewardship committee, but the studies had not started when I left -- when I left the industry.

Q. Who designed the protocol for the studies?
A. I can tell you who the primary investigators were: Dr. Otto Wong for the epidemiology components, Dr. Richard Irons for molecular biology, and they had -- they were assembling an advisory committee to help work out details.
Q. And is Dr. Irons in charge of the study or just a part of it?
A. Just a part of it is my understanding. I mean, when you say "in charge of," I'm talking the principal investigator. I mean, I don't know how the project's managed at all.
Q. Would you agree that one of the purposes behind the benzene study in China being sponsored or funded, if you will, by industry was to understand the health effects of low level benzene exposure?
A. Yes, that was one of the objectives.
Q. And another objective was to refute information that had come out of the Australian Institute of Petroleum's health watch study that was released in 2001?
A. That was not an objective that I'm aware of since it came out two years after when I was working on that project.
Q. That was not an objective that you recall being discussed by the American Petroleum Institute or its members?
A. Well, we weren't trying to refute anything. We were trying to improve the state of the science and the -- protect our workers. So the -- without knowing what the results of the

studies were, it's hard to imagine what -- what it would all be used for.

Q. Well, do you recall that part of the purpose of this study was to gain a quantifiable understanding of the relationship between benzene exposure and potential associated cancer risks?

A. Yes. Definitely.

Q. And one of the reasons -- or would it be a fair statement that the primary reason that China was picked as the location for these studies was because they had a unique study environment, including databases documenting exposure?

A. Yes. I mean, ultimately that's -- that's one of the reasons it became a favorable location.

Q. Do you know anything about the databases?

A. No. Not -- not -- only very general, not particular.

Q. Well, what was it about the databases in China that made that location, if you will, unique to study benzene and the effects of cancer?

A. Well, I'm not sure -- "unique" sounds like the only place in the world. We -- we -- we started out by asking the question whether -- where were there places where benzene exposures were high enough that you could develop a range of exposures and still identify cases. The databases around Shanghai and -- and -- and some other places in China, they have a -- essentially an epidemiologic surveillance registry

that, amongst other things, reports new cases of -- new cancer cases and new leukemia cases, and it's tied into the region which is very, very large, so the opportunity to collect a number of cases over a short -- relatively short period of time existed.

Q. And, in fact, didn't the organization acknowledge that Shanghai was a unique study environment because there was a range of exposure levels?

A. Yes. They did a site -- some individuals did a site visit, and they felt it was -- offered a lot of opportunities.

Q. I take it that there was an agreement between these companies that agreed to fund the studies and the investigators. Would that be a fair statement?

A. I left the industry before anything formal was established. I don't know.

Q. Would it surprise you that members of the companies such as Chevron, Texaco, and Exxon-Mobil that had funded the Chinese benzene studies that were being performed by Dr. Wong and Dr. Irons and others, that they had the right to get essentially data as the study was ongoing to review information?

MR. SHOEBOTHAM: Objection, form.

A. I have no knowledge about that.

Q. (By Mr. Lubel) Would that surprise you?

MR. SHOEBOTHAM: Objection, form, calls for

speculation.

A. I don't -- I know nothing about it.

Q. (By Mr. Lubel) Let me ask you this: From an epidemiological standpoint, is that appropriate for the investigators to send data as the study is ongoing and not completed to review the information?

MR. SHOEBOTHAM: Objection, form.

A. I've worked on a number of industry-sponsored studies looking at multiple segments. It's not unusual for the investigators to report in quarterly or semiannual or annual, depending on the duration of the project, progress reports that would say the number of cases that are identified or the -- the total number of -- in the cohort at this time or in each distributions, but these would be all demographics. They would not have any outcome information in them.

Q. (By Mr. Lubel) What about laboratory results from tests? Would that be appropriate from an epidemiological standpoint for -- on an ongoing basis before the study has been completed and reports have been drafted to furnish that information to the companies that are essentially pouring billions of dollars into the studies?

MR. SHOEBOTHAM: Objection, form.

A. I've not seen the protocols, so I don't know what type of information we're talking about. It's just speculation, so I -- I can't answer that.

Q. (By Mr. Lubel) But as a general rule, I mean -- well, you're an epidemiologist, correct, by trade?

A. Yeah.

Q. As a general rule, is it appropriate for the investigators to send reports of laboratory results for tests that are being done in association with the study to the companies that are funding the -- the study and before the study is complete?

MR. SHOEBOTHAM: Objection, form.

A. I don't know what type of laboratory tests we're talking about. If it's exposure related type of information on -- on the set of data being collected, it -- it could make sense. I don't know what we're talking about.

Q. (By Mr. Lubel) Let me ask you -- let -- let's do it this way then: What type of information is appropriate for the investigators to share with the sponsoring or funding companies before the study's ever completed, as it's ongoing?

MR. SHOEBOTHAM: Objection, form.

A. I've never participated in a study that had molecular biology components to it, so I have no experience to speak from in that area.

Q. (By Mr. Lubel) Are you -- have you been privy to the various committees that have been created concerning the study and membership organization, things of that nature?

A. No. No, I have no information.

Q. Are you familiar with the American Petroleum Institute's responsibilities with regard to this industry-funded study in China?

A. Vaguely as it was evolving in 2000.

Q. What's your understanding of API's responsibility?

A. Well, as it was evolving at that time, they were going to provide administrative support for the project in the sense of handling the building and organizing oversight committee meetings, things like that.

Q. Including negotiating agreements with investigators?

A. I -- I think part of the activity was they would -- they would write the contracts with part of the administrative component.

Q. Were you aware that one of the reasons behind this study was to determine whether benzene causes all forms of AML or only certain subtypes?

A. That was one of the objectives of the -- the research. Or is, I guess, an objective of the research. I -- I would use the word "confirm" what we believed to be already the substate of the science.

Q. Which is what?

A. That benzene is only associated after high doses and long duration with certain forms of acute myelogenous.

Q. Which forms?

A. Well, most typically M7 -- those with five to seven

dilutions.

Q. Those the only ones that benzene causes?

A. By far the most frequent. There -- there are other -- some other chromosomal abnormalities and translocations that -- that have been associated but of lower frequency.

Q. Which one?

A. Those are reviewed by -- pretty well by Zhang.

Q. Let me ask it this way: Is there any chromosome abnormalities that you believe are not related to benzene exposure?

A. Based on my reading of the literature, which is still emerging, inverse 16 is not related to benzene, and translocation 15, 17 of the form that Mr. Boren has is not -- or it does not appear to be related to benzene.

Q. And do you have studies that state that translocation 15 and 17 is not related to benzene?

A. There is a fair amount of epidemiology about promyelocyte leukemias that the world generally concludes that there are no known environmental or occupational associations. I mean, there are some statistical associations here and there, but -- but benzene is not on any of those lists.

Q. I'm just asking you if you brought with you today studies that conclude that benzene cannot cause translocations of chromosomes 15 and 17?

A. No, I have not.

Q. Have you brought with you any studies that state in conclusion that benzene exposures can't cause translocation or breaks in 16?

A. Well, actually, let me rephrase even the answer to the first question. I have brought paper with me that examines the various frequencies and types of -- of translocation and chromosomal breaks that are associated with benzene. And you do not see clear. The statements are all made in the positive of what you see. Okay? And what you don't see are the -- are any translocations of 15 and 17 without lots of other chromosomal abnormalities associated with it. And you don't see any inverted 16s probably at all. But these are reviews of all the chromosomal abberations detected in humans exposed to benzene.

Q. Will you pull those studies out, if you've got one?

A. That one. That one actually reviews the benzene.

MR. LUBEL: Stickers.

THE REPORTER: That one actually what? I'm sorry. What did you just say?

THE WITNESS: That paper was specific to what has been observed in benzene.

MR. LUBEL: And we're going to mark what you just called that paper as Exhibit 2. Okay?

(RAABE Exhibit No. 2 was marked.)

A. Yeah. It's the paper by Zhang and Martyn Smith is the last author.

Q. (By Mr. Lubel) Before you move to the next paper, can you show me where in this paper these authors reach the conclusion that translocations of 15 and 17 are not related to benzene exposures? Or can't be related to benzene exposures.

A. Well, I -- I stated that these papers are in the affirmative. They -- they are reporting what has been seen.

Q. I understand you said that. Now I'm asking you: Can you find for me in that article the location where they say --

A. No, you cannot.

Q. -- it can't cause it?

A. No, you would not.

Q. In any of the studies -- you've done studies on benzene. Correct?

A. Yes.

Q. They've been published?

A. Yes.

Q. In any of your studies, did you conclude that the translocations 15 and 17 can't be related to benzene exposures?

A. As some of these papers criticizing our epidemiology portray is that we have not -- it's only in the last several years that the appreciation of the cyto -- cytogenetics and the subspecies, the subclassifications of AML, is very important. So the -- pretty much all the epidemiology and certainly the

epidemiology that is exclusively dealing with mortality studies just can't do that.

MR. LUBEL: Objection, nonresponsive.

Q. (By Mr. Lubel) My question, Dr. Raabe, is: In any of the studies that you performed on benzene, did you make any statement or draw a conclusion that is in written form that benzene exposures cannot cause the chromosome translocations of 15 and 17?

A. None of our studies had a subtype available. So the answer is no, we did not.

Q. Now, you also referenced chromosome damage to 16. Correct?

A. Invert -- inverted 16.

Q. Inverted 16?

A. That's an M4. That's not related to Mr. Bolen.

Q. You call that an M4?

A. Well, it's -- it is a form of M4. Pure inverted 16 is the marker generally for classified -- classification of M4.

Q. Does Exhibit No. 2, which you've told us addresses translocations 15 and 17, also address inverted 16?

A. It addresses what has been found in benzene-exposed workers.

Q. Right. Now, I interrupted you from your process of gathering the other articles on this issue. So I'm going to give you --

MR. LUBEL: Let's take a short break so you can pull those out, if you don't mind.

THE WITNESS: That's fine.

(Recess from 10:35 a.m. to 10:41 a.m.)

Q. (By Mr. Lubel) Have you pulled the studies we talked about before the break?

A. Yes. I pulled the studies that I think are related to the discussion of what can cause acute promyelocytic leukemia and what is known about secondary benzene or other chemical-induced leukemia.

MR. LUBEL: Can we mark those as Exhibit 3, all of them?

THE WITNESS: Yes, that's fine with me.

(RAABE Exhibit No. 3 was marked.)

Q. (By Mr. Lubel) Do any of the studies that we've marked as Exhibit No. 3 that you've brought with you today conclude that benzene exposures cannot cause APL?

A. The very first one, for example, concludes that there are no known -- it -- it reviews what is known about the occupational and therapy related and other possible etiologies for -- may we call it APL?

Q. (Mr. Lubel nods.)

A. APL. And draws the conclusion that it -- that there are currently no known associations.

The second paper examines a number of risk

factors associated with a large study of APL in -- in Italy -- I believe it's Italy -- and concludes that while they find a few associations, none of them were related to trades or crafts that would be thought to have potential for benzene exposure.

The last one is another case control study of acute promyelocytic leukemia. And it -- the only association it finds is related to shoe making in Italy and weak associations with radionuclide exposure and hair dyes.

The last -- the next paper is therapy-related acute promyelocytic leukemias, discusses what appeared -- what makes APL very unique in that even secondaries look pretty similar to the -- the primaries in terms of their treatment outcome and things like that and discusses the types of materials that are potentially capable of causing secondaries. And benzene, at least in terms of exclusion, does not seem to fall into those categories.

And the last paper is a -- is a review paper of what's known about secondary myelodysplastic syndromes in leukemias not specific to APL.

MR. LUBEL: Objection, nonresponsive.

Q. (By Mr. Lubel) My only question, Dr. Raabe, is: In Exhibit No. 3, can you pull out the section in any of these studies that conclude that benzene does not cause APL?

A. No.

Q. Exhibit No. 1 is a copy -- copy of your deposition

notice. I take it that Mr. Shoebotham, on behalf of his client Dow Chemical, forwarded that before you got here today?

A. Yes, sir.

Q. Have you brought all the materials that you're relying upon for your opinions?

A. Yes. I believe generally these are the ones that would be directly responsive to my opinions in Mr. Boren's case.

Q. Have you brought with you the POWERPoint presentation that you used for the benzene studies in China?

A. No, I have not.

Q. Do you still have it?

A. It's still on my desktop, yes.

Q. Is there any particular reason you hadn't -- have not brought that?

MR. SHOEBOTHAM: I filed an objection to that section of the duces tecum yesterday.

Q. (By Mr. Lubel) Is there any particular reason you have --

MR. SHOEBOTHAM: I advised Dr. Raabe there was an objection that I filed.

Q. (By Mr. Lubel) So based upon recommendations of Mr. Shoebotham, you've not brought it. Correct?

A. Correct.

Q. Is there anything proprietary or secretive about the

POWERPoint presentation?

A. No. Other than it's not related to anything about Mr. Boren.

Q. Isn't the Chinese studies about acute leukemias?

A. It's a series of studies that have just begun. And I don't know the timeline, but I wouldn't expect any results for three to four or five years. So it's difficult to imagine how anything about those studies could relate to Mr. Boren.

Q. Aren't there statements --

A. Unless it's historically.

Q. Don't you make statements in the POWERPoint presentation about AMLs?

A. Yes.

Q. That's not related to Mr. Boren? Isn't he an AML?

A. But the statements are that we want to have more additional study on dose response for AML. But that's not going to happen for years.

Q. Well, in fact, the purpose behind the study, at least one of the purposes, is the fact that industry has been plagued with a lack of evidence on whether there's a threshold for benzene and AMLs. Correct?

A. No.

Q. Isn't that the reason they're going to China to find the threshold?

A. No.

Q. That's not part of the purpose of the study is to find out if there's a threshold for benzene exposure for which no effect can be seen?

A. Right now there is only -- there are very few cohorts and only one that has been deemed acceptable for use in risk assessment which is dose response analysis. And one of the objectives of the -- of the Chinese studies is to conduct at least one additional study, if not more, that would -- could be used to assess whether what we have out of the Pliofilm cohort is correct.

Q. So you were not aware that one of the primary objectives of the benzene industry-funded China studies was to assess whether there's a threshold level of benzene exposure for which no effect can be seen?

A. Well, we have one study of that form to date. Certainly one of the -- when I say "dose response analysis," a consequence of dose response analysis would address the question: Are there levels of benzene exposure for which there is no increased risk of leukemia? We have one study that we use in the United States that gives us data on that. And it would be nice to have some other well-designed, well-conducted studies.

Q. And is that the -- one of the main objectives of the Chinese benzene study is to determine what threshold, if any, there is?

MR. SHOEBOTHAM: Objection, form, asked and answered.

A. There's an epidemiology component that will also -- that hopefully will address dose response analysis, but there's also an enormous molecular biology component that hopefully will provide new science and insight on to the course of occupationally-related benzene-induced disease, not just AML.

Q. (By Mr. Lubel) So if I've got, you know, documents from this -- these group of companies that are funding this study in China on benzene that suggest that one of the primary reasons that this study is being put together is to make a determination of the dose response relationship between benzene exposures and effect, you just disagree with that?

A. I thought I just said that is one of the objectives.

Q. And the companies that fund this study hope that this research may establish that there is an absolute threshold for benzene-induced hematological disorders below which there's no effect. Correct?

A. I don't know what they hope. It would -- certainly would be a -- if true, it would -- if true and confirmed in those studies, would be -- it might very -- I think everyone would feel happy if we had more data on that subject.

Q. And you were aware, were you not, that at least the two primary reasons behind the study, as you testified in the Cowey case, was to defend the companies in benzene-related

litigation, number one; and number two, to have documented proof that helps the companies in a regulatory setting?

A. That misstates my testimony in that -- in that case.

Q. In the Cowey case?

A. Yes, it does.

Q. You didn't say that in the Cowey case?

A. What I said in the Cowey case is we went through a long list of reasons for doing those studies: worker protection, more insight on dose response, more insight on course of disease. We went through a very long list.

You asked me then what would be some of the benefits of having data that supported, you know, what -- what we currently believe already exists. I mentioned the -- that it would have impact on some of the regulatory issues.

You badgered me some more about litigation, and at that point, I acknowledged that that could be a consequence. But it was not and never was an objective of those studies.

Q. Well, you'd agree that we can go back and look at your testimony in the Cowey case -- it's about ten months old -- and we can see what was said. Correct?

A. Yes.

Q. And you stand by the opinions -- statements you gave in that testimony under oath. Correct?

A. Yes.

Q. Are you relying on any information from Dow Chemical

in this case for your opinions?

A. Some of the information that supports my opinion comes from some of the epidemiology studies done in the Freeport operations, yes. It's not a -- I don't exclusively rely on it, but it supports my opinion.

Q. Let me ask you this: Are you relying upon any of the published studies by Ott or Bond, any of that work?

A. Well, we -- they're of historical interest because they -- they provide some limited information on -- on the risk of leukemia from benzene-exposed workers out of the Midland operation which was a totally different operation than where Mr. Boren worked.

Q. What were they studying in Midland?

A. Very similarly they -- the initial studies were all designed to look at the health of the workforce at the plant.

Q. They weren't studying benzene exposures only?

A. Well, they focussed down on benzene exposures.

Q. So you're telling me they were looking at all malignancies in that study? In -- in the Midland studies that were published by Ott, Bond, and others.

A. Well, the published studies are focusing in on benzene.

Q. Have you seen any unpublished studies that focussed on something else?

A. I have a recollection that there -- that there must

have been -- I mean, they're nested within a cohort. So I have this recollection that they must have conducted a -- a study that looked at all cancer mentality and then drilled down. But offhand, I don't remember the citation.

Q. Can you pull for us the Dow-Midland studies that you have with you today --

A. Yes, sir.

Q. -- please?

A. Freeport. That's Midland.

MR. LUBEL: Jonathan, what -- what is your objection to the POWERPoint presentation? Is it privilege or what's your objection?

MR. SHOEBOTHAM: My objection is stated in the objection that was filed yesterday. My recollection is that the grounds were -- were primarily relevancy. But I think there may have been some other grounds. I would just rely on the written objection that we filed before the deposition began.

Okay. Let's take a break. I'll be right back.

MR. LUBEL: Can I continue asking him questions?

MR. SHOEBOTHAM: What's that?

MR. LUBEL: Can I continue to ask him questions?

MR. SHOEBOTHAM: Just wait a second.

(Recess from 10:56 a.m. to 11:01 a.m.)

Q. (By Mr. Lubel) Dr. --

A. I've given you two -- two papers from Midland.

Q. You actually gave me one, and the lawyer here gave me the other one. Correct?

A. That's correct.

Q. That was -- the 2003 version was not within your stack of documents. Correct?

A. I didn't bring all the Dow documents with me.

Q. And as you've told me, you're not relying upon any of these articles for your opinions in this case. Correct?

A. Not as they relate to Mr. Boren, no.

Q. Do you recall whether or not in the '86 published article from the Dow Chemical personnel whether or not they found a significant excess of AMLs in the benzene-exposed population in Midland?

A. I don't recall if it's statistically significant. I'm looking through the paper.

My quick examination of the paper suggests that they don't speak to acute myelogenous leukemia, that they just report results for leukemia. And I don't see statistically significant -- statistical significance even for the low leukemia.

Q. So you don't see a specific reference to the subtype of leukemia called AML. Correct?

A. I don't see that in this paper.

Q. All right. And you're referring to the '86 version

of the Dow Chemical study in Midland?

A. Correct.

Q. All right. Do you have -- when you flip through there, do you see any reference to what the chemical exposures were to those benzene-exposed workers as far as levels?

A. Yes. In this paper they had -- they had -- for the -- all leukemias, they attempt to classify them in three different exposure ranges of accumulative exposure.

Q. What are those ranges?

A. If I'm reading it correctly: 0 to 499, 500 to 999, and greater than a thousand.

Q. Okay. And have you seen references to there being excess leukemias of the AML variety at the Dow Chemical plant in Midland at exposure levels in the approximate range of 25 parts per million?

A. I don't recall.

Q. You've never seen that?

A. I...

Q. That you can remember?

A. I just don't recall.

Q. At any rate, you didn't see any reference in this article --

MR. LUBEL: And we'll go ahead and mark this as Exhibit 4.

(RAABE Exhibit No. 4 was marked.)

Q. (By Mr. Lubel) -- to AML specifically. Correct? You just --

A. No, I didn't. I -- I just now did not read the text. But in terms of the tables that I was looking at, I didn't see any AML specific tables.

Q. And as you sit here, you don't recall there ever being a reference in any form or fashion in Exhibit 4 of this Dow Chemical study about AMLs specifically?

A. You're going to make me read the paper. I mean, I just don't recall --

Q. Read the abstract. You need to go no further than the first paragraph.

A. Ah. Good. Thank you.

Q. What does it say?

A. It says that the leukemias they observed were all AMLs.

Q. And that there was a significant excess, correct, in that same category?

A. Yes. But it's not presented that way in the table. So it must be the -- the significance analysis must be in the text -- in the body.

Q. Did they break down the subtypes of AML in this study?

A. No. It's a mortality study, and it's unlikely that that data would have been available.

Q. In your benzene study, did you break down the subtypes of AML?

A. No, we did not.

Q. In any of Dr. Wong's longstanding benzene research, did he break down the subtypes of AML?

A. Once again, these are mortality studies, and it's unlikely he would have had that information. So I don't believe he has.

Q. Does Dr. Irons, in any of his work -- have you seen a breakdown of subtypes of AML regarding what types he thought were related to benzene and what types he didn't?

A. I don't routinely follow all of Dr. Irons' work. So I -- I -- I can't answer to what he may have done.

Q. In the Rinsky work, was there a breakdown of the subtypes?

A. No.

Q. In the China work that has been written about by Hayes, Yin, and others, did they break down AML subtypes?

A. No.

Q. Is it generally accepted within the international scientific community that benzene exposures can cause AML?

A. At expo, the -- the descriptive epidemiology we have to date at high exposures of long duration can cause AML, yes.

Q. So at this date, there is general acceptance amongst the international scientific community that some benzene

exposures can cause acute myeloid or acute myelogenesis leukemia. Correct?

A. Yes.

Q. Otherwise known as acute nonlymphocytic leukemia?

A. That may include a few other categories, but basically, yes.

Q. And the title of Exhibit No. 4 -- this is the Dow study from '86. Correct?

A. Yes.

Q. Is "An update of mortality among chemical workers exposed to benzene"?

A. Correct.

Q. And as you've pointed out for us, this involves Midland, Michigan plants?

A. Correct.

Q. Have you seen any published study that involves the Freeport, Texas plant?

A. Yes.

Q. And benzene workers?

A. They're not quite conducted exclusively to -- to benzene.

Q. What I'm trying to find out is if you've seen a comparable study of benzene workers regarding the Dow-Freeport plant?

A. Well, the -- the -- the studies of Freeport that I've

seen don't show an excess of leukemia. And it --

Q. Can you show me those studies?

A. Well, here's the -- Burns and Carson is a 2003 update. It's the most recent that relates to Freeport. And then there are -- there's an '85 and then the published and unpublished version.

Q. If you'd pull those out, we'll go ahead and mark them all.

So you've handed me a stack of articles that you believe relate to Dow-Freeport specifically. Correct?

A. Yes.

MR. LUBEL: We're going to mark -- mark them different numbers.

A. Well, Freeport and the -- and the few of them include other Texas operations, but -- but they all include Freeport as best as I can see.

Q. (By Mr. Lubel) All right. The first one I'm going to mark as Exhibit No. 5, and it's titled "Cause-specific mortality among employees of Texas-based chemical manufacturing facility, 1940 through 1996." Correct?

A. Yes.

(RAABE Exhibit No. 5 was marked.)

Q. (By Mr. Lubel) Does this article -- well, we know by its title, it doesn't specifically address benzene workers. Correct?

A. Correct.

Q. Is there any section of this article that addresses specifically benzene workers?

A. I don't -- no, there's not -- no special analysis for benzene-exposed workers.

Q. All right. And I'm going to ask you essentially the same series of questions as we go through. The next article you handed me I'm going to mark as Exhibit No. 6.

(RAABE Exhibit No. 6 was marked.)

Q. (By Mr. Lubel) As we can tell from this article, benzene is not referenced in the title. Correct? As it was in the Midland study?

A. Correct. This -- this is titled, "Mortality Among a Large Cohort of Chemical Manufacturing" Workers.

Q. Does that study address benzene workers specifically?

A. No, it does not.

(RAABE Exhibit Nos. 7-8 were marked.)

Q. (By Mr. Lubel) Same question for Exhibit 7?

A. No, it does not.

Q. Same question for No. 8, Exhibit 8?

A. No, it does not.

Q. Do 5, 6, 7, or 8 -- any of those studies that we've marked as exhibits -- have a breakdown for the form of leukemia called acute myeloid or acute myelogenous leukemia?

A. I've not exhaustively gone through all the text, but

I don't believe that -- at least in the tables, that they specifically discuss AML.

Q. Do you have any knowledge regarding the number of AML cases out of the Dow Chemical-Freeport plant?

A. I don't believe I've seen any data.

Q. And I'm not limiting it to data. Has Mr. Shoebotham or anybody that represents Dow Chemical kind of under the table told you: This is how many acute myelogenesis leukemias we had at Dow-Freeport? Anything like that?

MR. SHOEBOTHAM: Objection, form.

A. No, they have not.

Q. (By Mr. Lubel) Have you seen the Otto Wong study that was done on approximately nine plants in the mid '80s?

A. Yes.

Q. Do you know whether Dow was a part of that study?

A. I believe they were.

Q. I've, I think, seen the same study you're referring to. And would it surprise you if there is no reference made specifically to Dow within the study?

A. No.

Q. That would not surprise you?

A. No.

Q. Why not?

A. My understanding of the agreements for participation were that the individual company data would not be identified.

Q. Is that common for industry-funded studies?
A. Yes. Well, it's common for industry-wide studies whether they're funded by industry or not.
Q. Not to identify the plant that's being studied -- or plants?
A. It's not typical.
Q. So when we look at the Otto Wong study from the mid '80s regarding these nine chemical plants or petrochemical plants, you would not expect to see the identity of those plants contained or listed by the author of the study?
A. Correct.
Q. Have you seen any of the data that was shipped by Dow Chemical to Otto Wong regarding their plant and what diseases they had out there?
A. No, I've not.
Q. Are you familiar enough with that study to know that it was not a study that addressed specifically benzene workers?
A. Are we talking about the -- what is often called the CMA?
Q. (Mr. Lubel nods.)
A. The study that Otto -- Otto Wong participated in?
Q. In the mid '80s?
A. Yes.
No. The objective was to have one population of benzene-exposed workers and one population of nonexposed

workers.

Q. So you're saying that that was a benzene-specific study?

A. It was -- the attempt was, yes.

Q. And do you recall whether or not in that study Otto Wong broke out subcategories that included references -- subcategories within leukemia that included references to the types of leukemias, such as AML?

A. I'm not sure in the original report, but I think it's on the subsequent reports. You get whether there's some information on AMLs versus other cell types.

Q. And do you recall whether that was ever -- ever further broken down to provide subtypes within AML such as M3 or M4?

A. It wouldn't have been available.

Q. So it wouldn't list that?

A. No.

Q. And we won't know from reading the published material by Otto Wong specific information as to Dow Chemical recording the number of AMLs out of that plant. Correct?

A. I don't believe you'd be able to ease that out.

Q. Where would you get that information? Do you -- do you know whether or not that information would have been provided to Dr. Wong?

A. I...

Q. Just from the general standpoint of an epidemiologist.

A. From the general standpoint, I believe Dr. Wong would have known what the different submissions were; in other words, what companies were submitting what data.

Q. Have you seen an update from that study?

A. There has not been one to my -- my knowledge.

Q. Did you read the deposition I took of Dr. Collins, the Dow Chemical Director of Epidemiology?

A. Yes.

Q. Did you recognize and appreciate that when I asked him why there had not been an update of that study specific to the Dow-Freeport plant, his response to that was: There was no need for it, because we had the studies coming out of Dow-Midland, and those exposures were substantially similar to Dow-Freeport? Did you see the conversation?

MR. SHOEBOTHAM: Objection, form.

A. I don't recall it specifically. Are you -- but are you implying that Freeport was the group that went into Dr. Wong's?

Q. (By Mr. Lubel) I wasn't.

A. No. But that's what -- more or less what you just said.

Q. Well, what was Dow -- is it your understanding that the Wong study in the mid '80s that you've also called the CMA

study or some refer to it as the CMA study, that that included Dow-Freeport or was that Dow-Midland or do you know?

A. I don't know.

Q. It doesn't say in the study?

A. But it -- in your previous question to me, you characterize Dr. Collins' answer, which I don't recall being that, that we -- we didn't need to do Freeport because -- because we had other data on Midland.

Q. I think you're right. Let me see if I can rephrase it.

I understood Dr. Collins when he gave his testimony under oath --

A. I don't recall it in that detail.

Q. Yeah. So let me just give you --

A. I'm not -- I'm not prepared to actually debate that with you.

Q. Let me give you basics; that is, I understand Dr. Collins to have stated that Dow-Freeport didn't need to be studied any more than it had been because Dr. Wong had studied it when he did the mid '80s report.

A. Okay. I don't --

MR. SHOEBOTHAM: Objection, form.

A. -- recall that.

Q. (By Mr. Lubel) All right. Well, that's number one. Number two, do you agree or disagree, if this is a correct

interpretation of Dr. Collins' testimony, that it would not be necessary to study Dow-Freeport specifically regarding benzene workers if you've got reliable data from Dow-Midland benzene workers if the exposures are substantially similar?

MR. SHOEBOTHAM: Objection, form.

Q. (By Mr. Lubel) Do you agree with that statement?

A. I can't agree -- agree or disagree. Because each occupational medicine department or epidemiology group would have different criteria for what studies to update, when to update them. And I don't have enough specific information to agree or disagree with Dr. Collins' statement.

Q. Let me ask you this: When -- when Dow started studying the benzene workers in Michigan, would it be a fair assumption that they knew that they had benzene workers down in Texas at Freeport?

MR. SHOEBOTHAM: Objection, form.

A. I don't now how they're -- I think it would probably be a fair statement, but I don't know how they're organized, whether the epidemiology group at corporate knew the locations of all the different material, the production facilities, and -- and what they made.

Q. (By Mr. Lubel) Had you been in the epidemiological department back in the late '70s for Dow Chemical -- let's say you were the director -- would you have recommended that just the Dow-Midland benzene workers been studied, or would you have

recommended bring in the Dow-Freeport benzene workers to study them all?

MR. SHOEBOTHAM: Objection, form.

A. There -- there's no simple answer to that question.

Q. (By Mr. Lubel) What's the complicated answer?

A. Well, the complicated answer is you would tend to start where you had what you perceived to be the best data available to start. And then based on -- based on what you learned in that study and what you knew from industrial hygiene at your other facilities, you could -- depending on what other priorities you had -- benzene is not the only material Dow -- Dow makes -- make decisions as to what level of detail and how frequently and what -- what do you want to study next.

So you just can't say, you know, if I -- what -- you just can't estimate what their decision process was.

Q. Well, you'd agree that in the field of epidemiology, it's generally accepted amongst people that do what you do that the power of the study can have an impact on the conclusions?

A. Yes. Well, not -- not -- would have no impact on the conclusions. Would have impact on the -- how strongly you could extrapolate the results. The conclusions would still be just as valid whether it's a big study or not. It's just whether or not they would apply broadly.

Q. But generally speaking in the field of epidemiology, you'd like to study more people than less. Correct? You'd

like to have a bigger size?

A. As long as bigger is as manageable as the next level down, yes.

Q. So from an epidemiological standpoint, generally speaking, if all else is equal, it would have been better for the Dow people when they're studying benzene workers to study all the benzene-exposed workers which would include the Dow-Freeport people?

MR. SHOEBOTHAM: Objection, form.

Q. (By Mr. Lubel) True?

MR. SHOEBOTHAM: Objection, form, calls for complete speculation.

A. It goes back to my earlier answer. I don't know what the underlying motivation was to do the Midland study. So whether to include Freeport or not, I just can't -- I have no way of knowing what -- what -- what would've worked there.

Q. (By Mr. Lubel) As you sit here today, you don't know whether there's ever been a benzene workers study of the Dow-Freeport people. Correct?

A. I have not seen any material on that.

Q. Has anybody told you that there has been a study to that effect?

A. No.

MR. SHOEBOTHAM: You're talking about other than Wong's study?

MR. LUBEL: He doesn't know that Wong's study is. Nobody does because it's not in the published literature.

MR. SHOEBOTHAM: He testified that -- that the Freeport population was part of the Wong study.

THE WITNESS: I didn't. Collins did.

MR. LUBEL: He doesn't know.

THE WITNESS: Well, he said Collins did.

MR. LUBEL: He said he doesn't know.

THE WITNESS: I just --

MR. LUBEL: Raabe's not willing to take that leap of faith as he sits here right now. Right?

THE WITNESS: I don't -- I mean, I was at some of those meetings, and I don't recall what Dow's submission was. So if -- if Dr. Collins says it was Freeport, I -- I accept that. I'm not trying to argue with it, but I don't recall that, and it's not -- wouldn't be in the papers.

Q. (By Mr. Lubel) Was Dr. Collins there at that time, in those meetings?

A. No.

Q. He wasn't even working for Dow, was he?

A. No.

Q. He was at Monsanto?

A. No. And I -- and I don't recall who the Dow -- Dow representative was at those meetings.

Q. Well, what plants were studied?

A. I don't -- I don't know.
Q. If you're at the meetings?
A. Well, I mean, I know -- I know of one plant that was studied.
Q. Your plant, Mobil?
A. Yes.
Q. Any others?
A. Any others?
Q. That were studied other than Mobil and maybe Dow.
A. The only plant-specific information I recall is -- is Mobil. Mobil.
Q. And what did Mobil supply to Dr. Wong?
A. Every --
Q. In the way of information.
A. To my understanding, because it was -- it was done by others, it was all of the -- we have a very distinct olefins and aromatics production plant in Beaumont. So all of the production people with potential benzene exposure were provided as the exposed group, and an unrelated plant -- and I can't even remember what it -- what it was. It wasn't a refinery. It was something totally different provided the, quote/unquote, nonexposed portion of the cohort.
THE REPORTER: Of the what?
THE WITNESS: Of the cohort.
MR. LUBEL: Nonexposed cohort.

Q. (By Mr. Lubel) Did Mobil supply Dr. Wong with the exposure information that he was supposed to rely upon?

A. It wasn't done quite that way. Do you mean -- ask me a question.

Q. Sure. Did Mobil have exposure information for the people that were being studied?

A. Yes.

Q. Did they supply that information to Dr. Wong?

A. No.

Q. Who did they supply it to?

A. I -- an exposure dictionary -- job exposure matrix was established. And the exposures were appended to the matrix. So we would have submitted a matrix of the job titles with timelines and exposure information. The raw data was not sent to -- to Dr. Wong.

Q. Who interpreted the raw data to assemble these matrixes -- matrixes that were actually sent to Dr. Wong?

A. Industrial hygiene and plant personnel at the Beaumont facility.

Q. And what were the exposure levels at Mobil?

A. Very low.

Q. How low?

A. I think the highest was about four or five ppm.

Q. Over what period of time?

A. The plant was built in the early '60s, so it was from

the start of the plant to whenever the cohort cutoff date was, which was '78 or '80, something like that.

Q. So about 20 years?

A. I'd have to go back and look at the cutoff pit dates, but...

Q. At any rate, do you recall whether Mobil had actual monitoring data that went back to the beginning of the plant, 1960 or so?

A. They had grab samples in some of the early dates, and then later, different qualities of --

Q. When did they move on from grab samples to actual monitoring of workers?

A. Don't recall.

Q. Was the progression of exposure information that Mobil had, did it start off with grab samples and then move to area monitoring and then move to personal -- personnel monitoring?

A. No. It was technology driven. In other words, in the very early days, there were -- well, that was my understanding. You didn't have the -- any reasonable capabilities to get reliable data on -- on individuals with any routine process. I mean, there were certainly technologies, but -- but...

Q. When did that start?

A. I don't remember. I'm not -- I'm not familiar with

state of the art issues in industrial hygiene monitoring. But it was not -- it was some time shortly after that, because there were -- there were data in the mid '60s that started to be personnel.

Q. How was the grab sample taken to your knowledge?

A. Don't know. Just described to me as a grab sample.

Q. Well, how was the information -- the exposure information translated, if you will, from the grab sample to the individual worker in the study?

A. I can only speak generally on the process. Generally what was done was the job matrix had a combination of location and tasks and information on -- on the task. And the location would be assembled, whatever they had in the time periods. And then it would be interpolated and assigned to that task in that time period.

Q. And who would perform that responsibility?

A. Combination of industrial hygiene and plant personnel. I don't know specifically who was involved.

Q. So Mobil employees would assemble this information and then send it to Dr. Wong in some type of spreadsheet. Is that --

A. Yeah, that's a fair --

Q. -- accurate?

A. -- fair summary.

Q. Is that generally what's been going in these -- at

these studies that we've seen on benzene over the last 25 or 30 years?

A. No.

Q. Has it changed?

A. Yes.

Q. In your '95 study that you and Dr. Wong have published -- do you recall that study? Is it around '95?

A. Give me the subject matter.

Q. On benzene.

A. The -- our review of the refinery workers?

Q. Right.

A. Yeah. Okay.

Q. How did y'all receive exposure information for the refineries that were the subject of the study?

A. Okay. That study does not have benzene-specific exposure information in it.

Q. You did not receive any benzene information?

A. That study summarizes all of the cell type specific leukemias observed in all the different refinery cohorts.

Q. Does it reference --

A. It references --

Q. -- M3s?

A. No.

Q. So it doesn't get into that level of detail?

A. Correct.

Q. The subtypes of AML, for example?
A. Correct.
Q. Y'all broke down leukemias to the like AML, CLL, CML?
A. ALL. True.
Q. That type of breakdown?
A. Correct.
Q. So the refinery study is not a benzene-specific study, if you will?
A. The -- the summary tables of cell type specific apply to refinery workers. There are -- there's considerable other data and other studies that are discussed in the text, but those tables are not specific to benzene.
Q. Can you pull that study out?
MR. LUBEL: Can we take a short restroom break?
MR. SHOEBOTHAM: Sure.
(Recess from 11:35 a.m. to 11:44 a.m.)
(RAABE Exhibit No. 9 was marked.)
Q. (By Mr. Lubel) Exhibit No. 9 is the study that you pulled out that you and Dr. Wong authored?
A. Yes. You know, a case -- case study.
Q. It's frequently referred to as the '95 study? You have --
A. Not by me.
Q. You've published other stuff?
A. Yes.

Q. But in '95, it was the only one you published on that area. Right? Benzene?

A. Well, there are two versions of it that are essentially identical: one in '95, one in '96.

Q. What is the difference between them?

A. One -- there's no substantive difference between them. Slightly different discussion because it was being written after the '95 symposium, and it appeared in the Environmental Health Perspectives. There's no real difference.

Q. I only need to read the Exhibit 9 version --

A. Yes.

Q. -- to find out --

A. Exactly.

Q. -- how you --

A. Exactly.

Q. -- what you believe. Okay. Now, what -- y'all actually listed the actual plants that are studied in your '95 study?

A. Correct.

Q. How did you get permission to do that when apparently Otto Wong couldn't get permission to list the chemical plants in the mid '80s study that he did?

MR. SHOEBOTHAM: Objection, form.

A. The -- the cohorts themselves were already published previously. They did not have cell type specific information.

So who the cohorts were was sort of general knowledge. The actual cell type specific analysis does not show the company.

Q. (By Mr. Lubel) In the '95 study that we've marked as Exhibit No. 9, did you do an individual plant-by-plant investigation?

A. That -- only to the extent that each individual cohort provided us the specific number of person years and the specific cell types that they observed. So we cal -- so we could calculate the -- the cohort specific cell -- leukemia cell type, SMRs.

Q. In other words, did you get exposure information from each plant?

A. No.

Q. Did you get exposure information as a whole for all the plants?

A. No.

Q. Exposure was not part of the study. Correct?

A. Only to the extent that they were all classified as refinery workers.

Q. Yeah. But you don't know, for instance, what the exposures were to the -- to the workers in the Amoco plant?

A. Correct.

Q. Or the Chevron plant?

A. Correct.

Q. Or any particular plant other than Mobil where you

worked?

A. Correct.

Q. But you do know -- and I take it it's reported in your study that we've marked as Exhibit No. 9 -- the number of, for instance, AMLs at Dow? Not Dow. At Shell?

A. At one time we did.

Q. Is that reported in this study?

A. The number?

Q. Per plant.

A. Well, the number per plant is, but not -- it doesn't identify which plant. So I can tell you that --

Q. Why not?

A. Because the -- for the companies to agree to let us summarize it this way, they asked us to code the identity.

Q. I understand that you wouldn't mention the name of the person that died of AML in the study. I'm just saying why wouldn't -- for instance, let's say Texaco had eight AMLs over the period of the study -- let you reference how many AMLs they had?

MR. SHOEBOTHAM: Objection, form.

A. I don't -- I don't recall the specifics on a company-by-company basis. But since the objective was to summarize all the petroleum workers that we could, it was easiest for us to get participation by saying we would not show the specific number of AMLs for any individual cohort or plant.

So, I mean, it was us telling them that, and the few companies that I recall that were having heartache suddenly had less heartache.

Q. (By Mr. Lubel) So what -- I mean, as part of this study that you and Dr. Wong did on the refineries, you did not look at each individual leukemia case and look at their particular exposures. Correct?

A. Correct.

Q. Nor did you look at -- in general at the exposures of the benzene workers at each of those plants?

A. Well, all refinery workers would have benzene exposure at higher rates than background populations. So this was just summarizing refinery workers --

Q. Did you --

A. -- in that context.

No, we did not do individual specific benzene unit analysis.

Q. But you didn't get data from the different benzene-containing units for Amoco or Chevron or any of the other particular plants and analyze the data that way?

A. Correct.

Q. Now, this study did not find a significant excess of AMLs?

A. In refinery workers, correct.

Q. Right?

There's more than this study that does not find an excess of AMLs to the benzene-exposed population. Correct? This isn't the only one?

A. No, that's correct. But it -- I mean, it summarizes some of the cohorts it did find early -- early excesses of leukemia in refinery workers.

Q. But you're not taking the position under oath that just because a study or a number of studies does not find an association in that study population between benzene and AML, you're not saying that stands for the proposition that there is no link?

A. No.

Q. That wouldn't be good science, would it?

A. No. One would have to examine the body of the science and considerable information specific to the individual to -- to associate the two.

Q. And it's still good science in your opinion when you look at the body of the science to say that there is a link for certain benzene exposures to acute myelitis leukemia. Correct?

A. Correct.

Q. Even though there are some studies that don't find an association?

A. Well, presumably those are -- the reason for all of that is because the exposures are low.

Q. Well, but irrespective of the exposures, the fact

that a particular study does not reflect an outcome that's reflected in other studies does not mean that the outcome found in the other studies ain't true. Right?

A. You have to examine the study in the context of what information it provides and where that information fits with the others -- the other published studies.

(RAABE Exhibit No. 10 was marked.)

Q. (By Mr. Lubel) Is Exhibit No. 10 the Otto Wong study that you brought with you, at least one version of it, that you've also or previously called today the CMA study?

A. Yes.

Q. And there's -- there's two versions of this? There's one and two?

A. No. There just -- there -- there -- I wouldn't use the term "versions." Paper one describes the general findings from the cohort, who the population studied, what they did, blah, blah, blah. And paper two is specific to the dose response analysis that was conducted for benzene.

Q. And this particular study is the study that we were talking about previously about -- Wong did the study of a number of plants, but the study itself doesn't tell you which plants were studied. Correct?

A. Correct.

Q. Now --

A. May I see this just briefly?

Q. Yes. And you know something about that study because you participated in meetings about it. Correct?

A. Yes, I did.

Q. As a Mobil employee. True?

A. Yes. Thank you.

Q. Now, it was your recollection that the way this study worked was that the companies assembled exposure data in-house and then supplied a summary of that to Dr. Wong in his capacity as investigator?

A. Based on the protocol that was developed for the study on how to do that.

Q. So who designed the protocol, if you know?

A. Well, Dr. Wong, I believe Dr. Tabershaw was involved early on, and Dr. Abraham Lillianfelds (phonetic) was an advisor. So some combination resulted in a process.

Q. So they designed the protocol for the study. Correct?

A. I believe that to be correct.

Q. And then somehow that protocol would've been communicated from those people to the -- the chemical companies or refineries, whenever the companies were in issue?

A. Right. Assignments were given for...

Q. But generally speaking as you recall the way it worked, at least for Mobil -- you can't speak for the other companies. Correct?

A. Correct.

Q. For Mobil, industrial hygiene people and other staff assembled exposure information that they had for the Beaumont plant and then put that in some type of summary chart and sent that to Dr. Wong?

A. Correct.

Q. Did you ever see that information?

A. At the time I believe I did, yes.

Q. Since this was a mortality study, you're looking at death certificates. Correct?

A. Correct.

Q. You ought to -- not do what's frequently called a morbidity or incident study?

A. There was no way possible to conduct such a study.

Q. So you -- the investigator relies on the information in the death certificate as being accurate and reliable. Correct?

A. Well, it is because the expected information is calculated the same way.

Q. My point is, though, if whoever fills out the death certificate puts the wrong disease on there, then that's a problem that you may not know about?

A. Epidemiologically it's not a problem, because if -- because all -- you're comparing to large populations where the same errors are likely to occur. So while the absolute numbers

of cases may -- could possibly shift if you had additional information, the resulting observers expect the SMR is considered a perfectly scientifically valid number.

Q. Even if you're talking about a rare disease?

A. In a general sense unless you believe that, you know, regionally or locally there's, you know, some reason to believe that -- that there is a bias on one side and not the other.

Q. But, I mean, you could take a real rare disease where you wouldn't even expect to see many in either the study cohort or in the control group, and if one -- if one death certificate were to be mislabeled and not have that particular rare disease on there, it could have an influence on the interpretation of that study. Correct?

MR. SHOEBOTHAM: Objection, form.

A. Unlikely.

Q. (By Mr. Lubel) But you're not willing to go out on a limb and say, "For all diseases, it matters whether or not the people filling out the death certificates are doing so accurately." You're not willing to make that jump, are you? That it never matters whether the information on the death certificates are reliable or not?

MR. SHOEBOTHAM: Objection, form.

A. Obviously accurate information on the death certificates is -- is -- is important to these studies.

Q. (By Mr. Lubel) Now, for the CMA study that we've

marked as Exhibit No. 10, did you receive drafts of this before it was published?

A. The process wasn't that way. There was a -- Otto could do whatever he wanted on the publication.

Q. I'm just asking you --

A. Dr. Wong.

Q. -- in advance --

A. No. What I'm saying is that there is an unpublished report that was handed to CMA. And that -- that report was communicated immediately to the government. We never saw a draft. I mean, we made comments on what Otto -- what Dr. Wong provided. And I don't know whether there was any changes made, but the report -- the first report that we saw was the report that was sent to OSHA and NIOSH when we first saw it.

Q. Well, let me ask you --

A. But the publication was totally independent of that.

Q. Let me do it this way: You referenced an unpublished report?

A. Correct.

Q. And then you referenced a published report which we see is Exhibit 10. Right?

A. Correct.

Q. Do you know if the unpublished report looks anything different than the published report?

A. It's going to be much thicker because it has lots of

information that you'd never get into in a published report. One's unpublished analysis tends to have a lot of tables that essentially look at the populations, the age distributions -- a number of things that are not always likely to show up in the published version of the report.

Q. Well, what -- what version does the peer reviewers look at? The unpublished or the published version?

A. They look at whatever the author sends to the peer reviewers.

Q. Okay. In this case, do you know which version they got?

MR. SHOEBOTHAM: Objection, form, calls for speculation.

Q. (By Mr. Lubel) Have they got the unpublished or the published version?

A. Well, it's clearly unpublished when you send it to the peer reviewers.

Q. I understand that. But the thicker version, did the peer reviewers get the thicker version or do you know?

A. Don't know, but that would be very uncommon in epidemiologic activity.

Q. Do you know who peer reviewed this?

A. Peer review was a -- is a -- I'm looking for a word -- a confidential process. One does not know who peer reviews your studies. It's up to the editors who they want to

send it to.

Q. Okay. Well, before this study was published, the CMA study -- by the way, CMA refers to the Chemical Manufacturing Association?

A. Yes.

Q. Did -- did Dr. Wong's work get sent around to, for instance, reputable scientists to examine his work before he sent it out for publication?

A. Dr. Wong's unpublished report that he submitted to CMA as part of the contract was sent to NIOSH, OSHA, made generally publically available to whoever wanted to read it.

Q. But in -- in particular, do you know if Dr. Wong's work was sent specifically to scientists to review his work?

A. I...

Q. To edit it or to tell him what changes ought to be made or anything?

A. I don't know. Not to my recollection.

Q. Now, before the published work -- strike that. Before the unpublished version, the one that you said was the thick version --

A. Can we call it the final report? Because that's what it -- what it is: the final report.

Q. My question is: Did you ever see any draft reports?

A. No.

Q. I'm not talking about just you. I'm talking about

anybody at the CMA. You're saying they never saw a draft report?

MR. SHOEBOTHAM: Objection, form.

A. I don't believe they did.

Q. (By Mr. Lubel) So nobody within the CMA had the right, as you recall it, to review Dr. Wong's analysis or his assessments before the final report was completed and sent to the people at OSHA and whoever else asked for it?

MR. SHOEBOTHAM: Objection, form.

A. I don't recall there being a draft for comments that was not the report that was made immediately available to anybody who wanted it.

Q. (By Mr. Lubel) But you recall CMA people making comments about Dr. Wong's paper and writing. Correct?

A. After the report was --

Q. Before he could fix it?

MR. SHOEBOTHAM: Objection, form.

A. Well, it was not so much fix it. The contract process that was in place at that time resulted in Dr. Wong providing a report that would be distributed. And I don't recall there being like a comment period in the report. That's all I'm saying.

Q. (By Mr. Lubel) Was that not common within industry organizations such as the CMA for there to be a period of time for people within the organization to review and potentially

make comments on the studies?

A. Normally, in my experience in -- in -- in industry-wide cohort studies, it's typical for the industry to have it for a month or two to provide comments back to the investigator, and the investigator, at that point, can either take the comments or not and then issue his final report. But there is a -- a window of -- of opportunity for the -- for the contractor to provide comment.

Q. And you don't think that happened for the Wong CMA study?

A. Correct.

Q. Is that something that he requested not be a part of the process?

A. Don't know.

Q. Well, surely the industry would want to have that ability to make comments. Correct?

MR. SHOEBOTHAM: Objection, form.

A. I -- I don't know how that process emerged.

Q. (By Mr. Lubel) When did the -- the study was apparently published in -- around the '86 time period?

A. Yeah, I think it was submitted probably around that time. Submitted in August, '86.

Q. When did that study start?

A. I think discussions for who would participate probably started around probably 1980 or maybe earlier.

Q. So the study lasted five or six years?
A. No.
Q. No. How long? Before the final report was published, how long did it take?
A. Well, there is a final report, and then there's a published paper from the report.
Q. When did the work start in relationship to when the final report was done? Did it start in the late '70s? I mean, when did this start?
A. When I speak to the final report, I'm talking the report that we were just discussing that goes -- the thicker report that goes to --
Q. I'm with you. I'm with you.
A. Okay. I think that was probably completed -- I don't know when the project started, because it was already starting -- started when I joined industry -- joined Mobil. I think it's -- the -- the -- the unpublished final report probably was issued '84, in that timeframe. '84, '85.
Q. And do you know whether the work had started in the '70s at some point?
A. Don't know.
Q. Do you know how Dr. Wong was brought in to -- to do this work? How he was chosen?
A. No. No, I don't.
Q. Do you know what Tabershaw's relationship to the

study was?

A. I believe that -- that early period, Dr. Wong worked for Dr. Tabershaw.

Q. What kind of epidemiologist is Dr. Tabershaw?

A. Well, into that -- we're now going into a very early era. Dr. Tabershaw was an occupational physician that went into epidemiology in the '60s and did a lot of historical work with various occupational groups.

Q. I take it that he was picked to run this CMA study because of his reputation in the field? If you know.

A. I can only speculate. I don't know.

Q. Does he have -- does he enjoy a good reputation in the field of epidemiology?

A. I'm hesitating only because I'm trying to think. I think he's passed away, but...

Q. When he was alive, did he enjoy a great reputation --

A. Yes.

Q. -- in the field of epidemiology?

A. Yes. Epidemiology had to be done with calculators in that day, so what he did with the technology available was very good.

MR. LUBEL: Good time for the lunch break?

MR. SHOEBOTHAM: Sure.

(Recess from 12:08 p.m. to 1:03 p.m.)

Q. (By Mr. Lubel) Did you have a good lunch?

A. It was satisfactory.
Q. Good.
The POWERPoint presentation regarding the China studies that you said was sitting on your desk back at home, have you forwarded a copy of that to Mr. Shoebotham to review?
A. No, I've not. And it's not sitting on my desk.
Q. I thought you said on your desk?
A. On my desktop computer.
Q. It's in your computer.
Have you talked to Mr. Shoebotham about what information is provided in the POWERPoint presentation?
A. Very superficial.
Q. What did you tell him?
A. I said it outlines the broad objectives for the research program that we were trying to -- trying to fund in China. And that's about the level of detail it was.
Q. Did you tell Mr. Shoebotham that any of the information contained therein was confidential or subject to a confidentiality agreement?
A. Never came up.
Q. Did you tell Mr. Shoebotham that anything on the POWERPoint presentation contained some trade secrets or proprietary information?
A. No.
Q. Are you taking the position that any of that

information is proprietary or trade secret?

A. No.

Q. Is there anything particularly secret about it to your knowledge?

MR. SHOEBOTHAM: Objection, form.

A. Not that I'm aware of.

Q. (By Mr. Lubel) Have you done any work regarding benzene exposures and skin absorption?

A. I've tracked the debate over the years between Dr. Paustenbach and some of the NIOSH scientists, but I've not done any work myself.

Q. From a toxicological standpoint, you hadn't run tests on any animals in that regard, have you?

A. No, I've not.

Q. Have you seen any Dow Chemical studies that address the issue of how much benzene can be absorbed through the skin?

A. I don't recall.

Q. Have you seen any studies done by the Chemical Manufacturers Association or the API on that issue?

A. Definitely no for CMA. I don't recall if API ever did any of that work.

Q. As you sit here, given the number of years that you've been involved in the -- what I call the benzene debate, have you seen or can you recall any literature that you believe addresses in a reliable fashion benzene exposures and what can

be absorbed through the skin?

A. It is quite a large body of literature out there. I have not looked at it, so I can't offer any opinions on that.

Q. That's not something you're prepared to do for this particular case at this time. Correct?

A. Correct.

Q. Do you recall if Mobil ever ran any studies on that issue of how much skin absorption there could be from benzene exposures?

A. As you know, Mobil had a fairly active benzene research program. I don't recall the general absorption specifically, though.

Q. Do -- do any of the literature that you've brought with you today, did you -- I take it you didn't bring any literature here today that addresses specifically any opinions you intend to offer on skin absorption for benzene?

A. No, I didn't bring any literature.

Q. Do you recall discussions within the CMA about the Dow cytogenetic studies that had come out in the late '70s?

A. Not at CMA, no.

Q. Do you recall a fellow by the name of Epstein? He may have been with API.

A. The name Epstein sounds familiar, but I don't recall it with any association to API, no.

Q. Do you recall any discussions within the API

regarding the Dow cytogenetic studies concerning benzene exposures?

A. I don't recall.

Q. So you don't recall the CMA as an industry organization having a broad problem with the Dow cytogenetic studies in the late '70s?

A. I was not that close to the CMA activities, so I -- I don't know what -- what they were up to.

Q. I've seen your name -- name on some of the CMA meetings, but it appears to me -- and correct me if I'm wrong -- that the times that you would appear on behalf of Mobil Oil Company would be when the regular members didn't show up?

A. That's probably correct.

Q. And how do you pronounce -- is it Bue -- Buell (phonetic)?

A. Can I see the...

Q. I don't have the one with -- let me find the one with his name on it.

A. I think I know who we're talking about, but I'm blocking on his name now. John Behun?

Q. I don't think that's it.

A. I think it -- I think that was him.

Q. Was he the regular member as you recall it?

A. Yes.

Q. What does the RMA stand for?

A. The RMA?

Q. I see that referenced in some of the CMA documents saying that the API and the RMA need to get together to...

A. I don't know as we sit here today.

Q. I also saw a reference to the Wong CMA studies that we referred to earlier being a ten-plant study. Can you explain why it is that the published literature reflects something less than ten plants?

A. Only -- yes, I can.

Q. Will you? I'm waiting.

A. But without any specifics, my understanding was that there were a number of plants, not all CMA members, that were, I think, originally approached, but not all of them ultimately participated.

Q. Did they back out?

A. I don't know the circumstances.

Q. I also see a fair amount of discussion about the different plants, collecting the plant data on exposures and then sitting down with a group to discuss it. Do you know anything about that?

A. I recall participating in one such meeting, yes.

Q. What do you recall from your meeting?

A. Well, the objective was to -- there were two difficulties in trying to come up with summary metrics. One was relating to peaks: How does one characterize a peak? How

does one even know when they occur? And that's been an issue inside epidemiological research for -- forever and still is.

So there were several discussions as to how to set the peaks and -- and -- and what -- what metric would make sense scientifically.

And the other objective was to, I think, try and decide what the range of the cut points would be. In other words, is there under one? I'm not saying that that's -- what I'm giving you here now is the sort of discussion but not -- not specifically. You know, is it we've got one category under five and five to ten?

So it was trying to make some sense of the distribution of the numbers and how to categorize them. That's my recollection of the meeting I participated in.

Q. Why wouldn't that have been a decision by the investigators as opposed to the --

A. I believe the investigators were -- well, at least, I believe the investigator was present during part of that.

Q. But why would -- why wouldn't the investigator make the decision on how the exposures were classified as opposed to having, you know, the industry make the decision?

A. Well, the industry -- I don't think the industry did make the decision. I think it -- it was a -- the meeting was a group thing as to how to remember the -- the protocol set out that there'd be a job exposure test specific matrix that would

be -- that would be mapped. Each company would map the exposure methods to that test matrix. The test matrix would be rolled up into jobs.

And as a consequence of all that, I think -- you know, Otto needed -- I mean, people -- this hadn't been done very frequently before, so this was a somewhat new activity, and how to do it was a -- was partially just discussed in a broad setting. It wasn't an industry decision as to how to do it. It was what are our choices and which ones make sense.

Q. But ultimately there was a uniform rating for each exposure classification. Correct? Or job?

A. No. Well, I'm not sure if we're communicating here. What there was is there was a set of categories that each company would map their task into.

Q. Like, give us an example of what you're --

A. Well, let's say there was a task of drumming.

THE REPORTER: Of what?

THE WITNESS: Drumming. D-R --

A. And so -- so there was a drumming on the task list. Now, you may or may not have had any drummers in your cohort, but there was a drumming task. But if you had drummers, you would have to assign an exposure category to drumming.

Q. (By Mr. Lubel) Per plant or for all the plants?

A. Per plant is my -- my recollection.

Q. Then that's what I was driving at was -- so your

understanding is each plant assigned a value for that job classification?

A. A long time ago, but I think that was at least part of the initial process. I don't recall the specifics.

Q. Now, at least in theory, each plant would look at exposure data that they had learned about before the meeting and take that information and use it as the number that they used for that job description. Correct?

A. That's my presumption.

Q. So Mobil may have one number for drumming and Dow may have a different number and Union Carbide may have a different number and on down the road. Correct?

A. I -- could well be, yes. I mean, I don't know specifically, but makes sense.

Q. But you wouldn't anticipate that each company would have the same number for a particular job classification for that exposure, would you?

A. Well, there was no reason to think that they would be that dissimilar in a particular time period. But, you know, remember, they're putting it into categories. So, I mean, if they started out with 2.1 and mapped it into two, I -- you know, I don't know.

Q. But the raw data that the person or staff at Mobil, for instance, that they used to come up with the -- this exposure classification was not given to the investigators

themselves. Correct?

A. I don't -- I don't recall it being given to the investigator. They did site visit the facilities first. I'm sure they've seen it in the general discussion sense, but I don't believe that they were actually provided copies of that.

Q. Do you recall the site visits?

A. Before my time.

Q. So you don't know what actually took place in their site visits?

A. No. And when I say "before my time," I don't mean before my time joining Mobil, but before I got active into the project.

Q. So when you talk about summary metrics, what are you referring to?

A. Well, it's a -- it's a jargon term in epidemiology. What I'm referring to is the fact that -- that a body of industrial hygiene data would be summarized as either to a mean or median or geometrically. In other words, someone -- you would make a set of decisions that this is how we're going to summarize that data for this observation or this task in either context.

Q. Who performs that?

A. Typically industrial hygiene person.

Q. And then they provide that information to the investigator?

A. Well, they would work together so that they'd make sure everybody -- the investigator needs to understand very well how it was done so that he can interpret it properly when he uses it. So it's a team effort, but the lead is from industrial hygiene.

THE REPORTER: From where?

THE WITNESS: Industrial hygiene. Sorry.

Q. (By Mr. Lubel) There's going to be a number of communications between the individual company and the investigators. Correct?

A. At various times in the process, yes.

Q. They're going to be faxing back information to each other and mailing them information and sitting down and having meetings and things of that nature. Correct?

A. Not on a daily basis and not even frequently on a monthly basis, but there will be need for clarification of materials, yes.

Q. And at some point there's going to be a document from Mobil or whoever the studied plant is to the investigator. There's going to be a written document that's sent to them. Right?

A. Not necessarily.

Q. How do they get the exposure information?

A. Oh, you mean at the end of the process?

Q. Whenever.

A. I mean, a lot of those meetings are face to face.
Q. But they have to hand them something?
A. Yeah.
Q. They don't just verbally say, "Hey, our exposure levels are this"?
A. No. I can only speak for Mobil. But at the end of the process, a spreadsheet-like table was sent to the investigator.
Q. And have you ever seen those spreadsheets for Mobil?
A. I recall seeing it, you know, 30 years -- 25 years ago.
Q. Has that information been trashed to your knowledge?
MR. SHOEBOTHAM: Objection, form.
A. Don't know. Have no knowledge.
Q. (By Mr. Lubel) Do you recall what the recordkeeping or destruction policy was for Mobil regarding how long to keep the information that was sent for the studies to the investigators?
MR. SHOEBOTHAM: Objection, form.
A. I know what the general record retention policy for Mobil was in that time period, but what they -- when you've added on what they did with the investigators, I'd have -- I have no knowledge.
Q. (By Mr. Lubel) What was the general policy?
A. I think it was five years at that time.

Q. They would destroy the documents?
A. I don't know. I'm just telling you what the policy was.
Q. But what did you do with the documents that you had that were over five years old that related to epidemiological studies? Did you tell them -- did you send them down to the trash department to set a fire or whatever they do with them --
MR. SHOEBOTHAM: Objection, form.
Q. (By Mr. Lubel) -- or how did you treat that?
A. By the mid '80s, we had adopted a policy of long term record retention for study specific materials.
Q. Well, do you recall on the Cowey deposition that you gave nine or ten months ago that you referenced a 1964 Mobil that for the first time had been unearthed and I think you used the words surrendered to the Provost Umphrey firm? Do you recall that?
A. Yes. Because I had only recently come across it myself.
Q. Where did you find it?
A. When -- I found it because there were some library documents that Exxon did not take. And one of my former staff members down in Virginia said: Got some stuff. And there's some folders that you -- you know, it was clearly somewhat epi-related kind of stuff. Should we -- what -- what should we do with it? They don't want it. And then -- so I asked him to

just send -- I think it was one box worth of stuff -- send it up to me, because it was reprints and things like that.

Q. What did you do with the box?

A. Well, a number -- a fair percentage of it was reprints of papers that I coauthored, so I put those in my own reprint file. And some of it I had no idea what it was, made no sense to me. I threw that out. And the study I found and I kept.

Q. And -- and -- and do you still have that study?

A. The 1964 study --

Q. Right.

A. -- that was referenced right here?

Q. Can you send that to Mr. Shoebotham so I can see it?

MR. SHOEBOTHAM: Well, we -- I've objected --

MR. LUBEL: To that?

MR. SHOEBOTHAM: -- to the production of that study. There's a written objection filed with regard to that.

Q. (By Mr. Lubel) What's the -- what's the -- the 1964 study about?

A. It's an investigation conducted, as best as I can interpret from reading it, by statisticians from Metropolitan Life Insurance Company looking at the mortality patterns of Mobil employees during that period of time or prior -- you know, obviously prior to '64.

Q. Was it ever published?

A. No.
Q. So the Met Life study was sent to Mobil and that's how it got in the file that went in the box that was sent to you. Right?
A. Yes, I think that's probably fair to assume.
Q. So have you sent that 1964 study to Mr. Shoebotham to look at?
A. No.
Q. Is there anything proprietary about that study?
MR. SHOEBOTHAM: Objection, form, calls for speculation.
A. Proprietary is a -- is a -- something that epidemiologists don't...
Q. (By Mr. Lubel) Is there any trade secrets in it?
MR. SHOEBOTHAM: Objection, form, calls for speculation.
A. I don't think so.
Q. (By Mr. Lubel) Is there any Mobil processes described in it --
MR. SHOEBOTHAM: Objection.
Q. (By Mr. Lubel) -- I can go steal the -- from the study and go build my own Mobil plant and make a bunch of money?
MR. SHOEBOTHAM: Wait a second.
Q. (By Mr. Lubel) Is there anything like that in there?

MR. SHOEBOTHAM: Objection, form, calls for speculation.

Q. (By Mr. Lubel) Is there?

A. I didn't see anything that -- what I would be -- question as a trade secret.

Q. Does it identify by name the individual employees of Mobil?

MR. SHOEBOTHAM: Objection, form.

A. No, I don't believe so.

Q. (By Mr. Lubel) Does it identify by social security number any individual employees?

A. No.

Q. Give any of their home addresses of where these people live that have these problems?

A. No.

MR. SHOEBOTHAM: Objection, form.

Q. (By Mr. Lubel) Well, you've -- you've produced it to the Provost Umphrey law firm. Correct?

A. In a Mobil-related case, yes.

Q. Right. A benzene case?

A. Specific to a -- a Mobil facility, yes.

Q. But it had to do with benzene, I mean?

A. Well, the report doesn't, but the -- it's not a benzene-related report.

Q. Okay. But you produced it in a benzene-related case?

A. I was asked to produce all the Mobil documents relative to studies conducted by Mobil.

Q. But the -- the underlying case was an alleged benzene case as opposed to a car wreck or something?

A. Yes.

MR. LUBEL: So what's your objection, John?

MR. SHOEBOTHAM: My objection was stated in the objections that we filed yesterday.

MR. LUBEL: How do you know? You haven't seen the document. How do you know what to object to?

MR. SHOEBOTHAM: Well, I've objected to it.

Q. (By Mr. Lubel) Did you discuss with Mr. Shoebotham the specifics of the report?

A. No. I said it was a Metropolitan Life Insurance report. It has no bearing on -- on -- on any benzene-related matters that I can see.

Q. Did it address any benzene-related diseases like leukemia?

A. No.

Q. No leukemias were referenced in it?

A. In that era of time, it was predominantly cancer and heart disease. And there were few -- a few major cancers discussed.

Q. What does that mean, "a few major cancers"?

A. Well, I mean, there were -- my -- my only

recollection was colon cancer, lung cancer is discussed; there's a -- in other words reported. I don't even recall whether there was any leukemia reported in the study. I don't think so. It was more focussed on heart disease in workers, cardio -- cardiovascular risk.

Q. Was this information considered by Dr. Wong when he did the CMA study or do you know?

A. It would have been quite irrelevant, but I -- I can't imagine that he had even seen it.

Q. Wasn't he looking at the mortality of people that worked at the plant from all causes, essentially?

A. This is a report. This is not a data sect.

Q. But it's talking about mortality. Right?

A. I'm sorry, I don't -- yes, the report is talking about mortality, but I don't understand the...

Q. Wasn't Wong's study on mortality of Mobil and other plant workers?

A. I'm sorry. Now we're getting -- what study are you talking about?

Q. The CMA study.

A. No. The CMA study was specific -- had a very specific protocol that was not about general mortality experience.

Q. Okay. So you're saying that the CMA Wong study did not address, for instance, lung cancer?

A. It did in benzene-exposed workers.
Q. So it just looked at benzene-exposed workers?
A. What we've been calling the CMA study only assembled cohorts of benzene-exposed workers and nonbenzine-exposed workers for comparison as --
Q. As the control group?
A. Yes. And the mortality of the benzene-exposed cohort was fully enumerated in those reports. But it -- but it -- well, I already said it relates to those workers from those companies that were presumed to be benzene exposed.
Q. Well, you don't plan on throwing away the 1964 study. Correct?
A. No, I don't.
Q. Have you ever seen an answer to the question that was posed by the CMA: Do prior exposure to benzene at toxic concentrations make individuals more sensitive to subsequent exposures?
A. I'm not familiar with that statement.
Q. You ever seen an answer to that question?
A. No, I don't -- well, no, not -- not -- I mean, there's been discussions that I can -- without just, you know, being specific. It's a question. And it's a researchable question that we continue to have.
Q. Has it been researched?
A. I -- in some settings, probably.

Q. Do you know the answer to the question from the research that's available?

A. No.

Q. You spoke about peak exposures earlier. Do you recall that?

A. Yes, sir.

Q. Which exposures are more dangerous: the low level chronic exposures or the peak exposures? Or do we know the answer yet?

A. We don't really know the answer yet. The -- the -- with all the epidemiology, we can reliably develop. The state tends to focus on continuous exposures; and therefore, the metrics are expressed in ppm years.

Q. Do you know if the -- the industry-funded China studies that's ongoing, if it's seeking to address that issue of what matters more: the peak exposures as compared to the chronic low level exposures?

A. I don't know.

Q. Was that an issue that you thought was going to be addressed back when you were involved in '99?

A. Well, I think it was certainly known that it was -- would have been a -- it would be nice to have. But you always come back to the issue: Can you measure peak exposures reliably in a fashion that can be used in an epidemiologic investigation?

Q. Who is Paul Cheney? Apparently he went to a meeting for you of the CMA variety.

A. Paul Cheney, as I recall, was the plant contact of the data gathering in Beaumont.

Q. Why were some of the plants in the CMA Otto Wong's study plant-wide studies and some of them were not? Does that make any sense?

A. Yes. I understand the question.

The answer is that for Mobil, for example, the whole plant was an olefins aromatic plant, so pretty much excluding administrative personnel, all of the plant would have been considered benzene exposed at some level. Other facilities that were quite large would only contribute data from the -- from their BTX units or however they define their benzene subgroup.

Q. For instance, Dow, do you know what areas they chose to include in the Wong study?

A. I have no knowledge.

Q. Did you ever see the Dow exposure information that was submitted to Wong?

A. I don't believe so.

Q. Did the companies not share the information that they were sharing with the investigator?

A. Only to the extent that we would be sitting here around the room and trying to decide how to summarize it. I

mean, you might say, Well, I've got this, this, this, and you've got that, that, that, but not -- not in writing that I'm aware of.

Q. The summary itself for the raw data was shared between companies. Is that a fair statement?

A. Yes.

Q. I see some reference to discrepancies between tapes submitted and the cohort listings?

A. I believe you, but I don't remember any of it.

Q. And I'm focussed on what is meant by "tapes." What are they talking about?

A. Oh, I know what that answer is. In that era, there were magnetic tapes being shipped.

Q. They contained information?

A. Well, you know -- do you know -- do you know what I mean by mag tape?

Q. I've heard of it, but I -- is it like a microfilm?

A. You're not old enough.

Q. Is it microfilm like at the library?

A. No, no, no. No. Before diskettes, CDs, and things like that, all of these computer analyses were done on big mainframe computers. And these were magnetic tapes that were about 12 inches in diam -- in diameter. And --

Q. It's the old version of a disk today?

A. No. Then -- we had disks back then, too. But they

were so big and so cumbersome that they were not used to transport data. They were used on the computers themselves.

Q. Okay. Do people call you Jerry in those meetings -- in those meetings?

A. Probably.

Q. Who was Dr. Stille, S-T-I-L-L-E?

A. I think it's pronounced Stille, but I actually don't even remember who that is. I don't remember.

Q. Theodore Stille from the TOMA. I think it's Tabershaw Occupational Medicine --

A. Associates.

Q. Right.

A. I don't recall.

Q. Well, TOMA was the group that was the investigator for the CMA study. Right? That's who Wong worked for, wasn't it?

A. I -- I just don't recall if the company name was TOMA, but -- but now that we've said it four times, TOMA, yes, it probably was. You are correct. Didn't ring a bell when we first talked about it.

Q. And TOMA representatives, whether they be Dr. Tabershaw himself or Dr. Stille or Dr. Wong or whoever there was as their representative, was actively involved in the CMA meetings. Correct?

A. I don't recall that. I...

Q. You don't recall seeing them at meetings?
A. I wasn't at every meeting.
Q. At the ones you were at, do you recall seeing them?
A. I recall some meetings with Dr. Wong, and I recall a few very early meetings with Dr. Tabershaw, but I don't recall much of any other staff.
Q. You recall a fellow by the name of Hazleton?
A. No.
Q. Do you recall the five-day study?
A. No.
Q. Do you recall a 90-day benzene toxicology study to be conducted at Hazleton Laboratories?
A. Ah, Hazleton Laboratories, yes. They -- there were a number of benzene tox studies going on in the varying places around -- around the United States and around the world during that whole period. But I don't recall the purpose of the 90-day study. Although when you say 90-day, it sounds like it's a subchronic study.
Q. Well, do you recall the CMA asking Dr. Hazleton to revise his draft final report?
A. I have no recollection of that.
Q. Do you recall there being a lot of problems with the data that was -- that the plants had put together to give to Otto Wong for the CMA study?
A. I don't recall.

Q. Is it a true statement that analysis of urinary phenols was not considered to be a sensitive indicator of benzene exposure at low levels?

A. That's a true statement to my understanding.

Q. What is the appropriate weight of medically-monitored benzene exposed workers today?

MR. SHOEBOTHAM: Objection, form.

A. I am not familiar with what's currently being done. I mean, the urinary phenol was part of the OSHA for high -- high exposures expected to be over 10 ppm. And then CBCs, blood test, was the surveillance -- surveillance activity at the time when I left the industry.

Q. (By Mr. Lubel) Were there problems with looking at the phenol levels for benzene-exposed workers?

MR. SHOEBOTHAM: Objection, form.

A. They're only predictive at high exposures. And then that test has to be conducted within a reasonable period of time after the exposure.

Q. (By Mr. Lubel) Within 24 hours?

A. I don't recall. It's not my area.

Q. Do you recall the CMA having meetings with the authors of the IARC monograph on benzene?

A. I have no knowledge of that.

Q. Would that surprise you?

A. No.

MR. SHOEBOTHAM: Objection, form.

Q. (By Mr. Lubel) Talking about before the report was finalized, the monograph?

A. Whole monographs are written in residence in a week. And there are typically industry observers that are there to provide technical information into the process. But -- so I don't know what you're specifically speaking to.

Q. Have all of your studies been cohort studies on benzene?

A. Oh, benzene, yes. Well, the ones related to the refinery workers that are related potentially to benzene have all been cohort studies.

Q. Have you ever done a case control study?

A. Yes.

Q. Under what circumstances?

A. We did a case control study of workers exposed to asbestos.

Q. Why did you do a case control study as opposed to a cohort study?

A. Because you can develop a lot more detailed information on a smaller number of cases and controls than is typically feasible for a very large cohort.

Q. So what study is better for benzene exposures? A cohort or case control study?

A. Depends on what the objective of the study is.

Q. Under what circumstances would a case control study be better than a cohort study for benzene exposures?

A. Typically in a case control study, you can get more refined exposure information on the cases and controls.

Q. How do you get better information? Because they're alive?

A. No. That's a different element of the design. You just asked case control versus cohort. No, because you -- you now have the ability to identify very specific job history information. You can go to the medical records and get smoking history information. You can directly -- you know, you have just more opportunity to get more refined exposure and lifestyle and individual data on smaller numbers.

Q. Why can't you do that on a cohort study? Because the number -- the group's larger?

A. It's just typically -- trying to do that on 7,000 current and former workers just becomes technically and logistically -- one can never say impossible, but so difficult that -- that you --

Q. Practically impossible?

A. -- you -- that you go to the nested case control design.

Q. Would it be a fair statement that the case control design would provide more reliable specific detail than the cohort?

A. That depends on the individual study and how it's conducted.

Q. But everything else is equal, you'd expect to get more detail out of the case control design than the cohort design. Right?

A. I -- I -- I don't think you can make generalities that broadly. It depends on the size of the cohort relative to the size of the case control setting.

Q. Well, Dr. Collins, on behalf of Dow, objected to the China study being done by Wong and Irons that we've been talking about at some length today. And as I appreciate what he said, his primary complaint was the design of the study. And I -- did you read that when you read his testimony?

MR. SHOEBOTHAM: Objection, form.

A. I -- I read the same words that you did. My take away was that he was -- that he was speaking as an epidemiologist that liked cohort studies and nested case control studies within cohort studies. And that would not be feasible in the typical China setting.

Q. (By Mr. Lubel) To do a cohort study?

A. Unless you were going to do the -- all of Shanghai, it would not be feasible.

Q. Well, the study that was performed by Hayes & Yin and others in China, were those case control or cohort studies?

A. Those were cohort studies of multiple -- multiple

different facilities, which is part of the reason why nobody can quite understand how to interpret that study. It generated -- generates a lot of confounding information in the exposure setting.

Q. That's because they looked at too many factories?

A. Well, they've got painters; they've got chemical workers; they've got pesticide workers. And yes, ostensively, they may have shared some element of benzene exposure across those, but nowhere in those studies do they account for all the confounding exposures that are not measured. So if you were a pesticide worker and at any given time benzene was one level and pesticide was another, they're correlated.

Q. But that's one of the inherent problems with the cohort study is trying to get the specifics that you're referring to. Right?

A. That's why every -- each study has a different set of objectives.

Q. So the -- the industry-funded study that's ongoing right now by Irons and Wong, that's a case control study. Right?

MR. SHOEBOTHAM: Objection, form.

Q. (By Mr. Lubel) As you understand it?

MR. SHOEBOTHAM: Calls for speculation.

Q. (By Mr. Lubel) No. It's a cohort study. Right? I didn't mean to say case. Isn't it a cohort study?

A. My understanding of what Dr. Wong's piece is is a case control study with knowledge of the individual plants from which the cases are coming. I don't have any information on the molecular biology piece of what Dr. Irons is -- I don't know where Dr. Irons' cases are coming from.

Q. So it's your understanding that the China study, at least the part you're familiar with, is going to be a case control study?

A. In its basic design, yes.

Q. And Dr. Collins' objection to that was he preferred a cohort study. Is that what you understood him to say?

MR. SHOEBOTHAM: Objection, form.

A. I can't get into Dr. Collins' head. I mean, it was just a one liner, and -- and...

Q. (By Mr. Lubel) What does one line say, is what I'm getting at? What did he testify to?

MR. SHOEBOTHAM: Objection, form.

A. He said he prefers a cohort study.

Q. (By Mr. Lubel) How are they going to assemble the exposure evidence from the China study?

A. I don't have firsthand knowledge on how it's being done. I mean secondhand knowledge of how it's...

Q. From your '95 studies, how did you assemble the benzene exposure information?

A. Well, the papers that I believe you're referring to,

we had access to the Pliofilm cohort directly. We had the -- the raw data. And the refinery studies, as you recall our earlier discussion, we did not have exposure data specific to the leukemias.

Q. Did you have two '95 studies?

A. Dr. Wong has one that he's by himself on, and then there's the one that we have been referring to where we're both coauthors on. There's tables in that paper that talk about dose response for benzene that are generated out of the Pliofilm cohort, not the refinery cohorts.

Q. You talking about Exhibit 9?

A. Yes.

Q. But you -- your study in Exhibit 9 was not a study of the Pliofilm cohorts, was it? I thought it was refinery workers?

A. No. It discusses table ten in the paper. There's a very large section in the paper that discusses what we know about dose response and what we know about case control studies of different specific leukemias, subtypes and basic subtypes.

Q. But I thought Exhibit 9 was a refinery worker study?

A. Half of it, yeah. I mean, the -- the -- the -- it reports the refinery workers and then discusses what all is known about leukemia in a subtype, specific analysis, as it relates to benzene and refinery workers.

Q. All right. Now, for the Pliofilm portion of it,

where did you get the exposure information?

A. Dr. Wong received a copy of the Pliofilm cohort with the Rinsky exposure estimates and the Paustenbach exposure estimates.

Q. Who did he get them from?

A. That I don't recall.

Q. Did you ever see them?

A. Yeah.

Q. Did you ever analyze that data?

A. Out of the analysis, I saw some of the tables.

Q. What's the volume of exposure data that we're talking about? Are we talking about a box or ten pages of --

A. No. That cohort has been so extensively studied and documented that the cases are already assigned accumulative exposures and under -- there's three major exposures: Rinsky, Infante, and Paustenbach. And those have already been summarized as individual numbers on each case.

Q. I understand that. There's actually articles on them. Right?

A. Yeah. Lots.

Q. But have you seen the raw data, is what I'm asking? The raw exposure data.

A. No.

Q. From the Pliofilm cohort?

A. I have not.

Q. Has Wong seen it to your knowledge?
A. No. I believe he's -- no, I don't know. I don't know that actually.
Q. So when you or Wong -- strike Wong.
When you are involved in an article that talks about the Pliofilm exposures, you're interpreting the summary of the data as supplied from the -- from the Rinsky materials, from the Infante materials, and from the Paustenbach materials. Correct?
A. Yes.
Q. Now, who wrote the first article on the Pliofilm cohort?
A. Peter Infante.
Q. And it was followed by Rinsky. Correct?
A. Several versions of it.
Q. And then Paustenbach?
A. Paustenbach. Well, no.
Q. Crump & Allen?
A. You're confusing risk assessments using that data from the epidemiology studies using that data.
Q. Well, let's stick with the epidemiological studies.
A. Okay.
Q. What's the --
A. First Rinsky, Paustenbach -- excuse me. That's incorrect. Paxton then -- then another update by Rinsky.

Q. Where does Infante fall in that?
A. He was not involved in any of the updates except maybe a courtesy fourth or fifth author on the first Rinsky.
THE REPORTER: With what? I'm sorry.
THE WITNESS: Courtesy fourth, fifth author -- fourth or fifth.
A. I don't recall where he is, but he's near the end of the author list.
Q. (By Mr. Lubel) But the original study was done by Infante. Correct?
A. Without detailed exposure estimates, correct. The original cohort mortality study.
Q. And then Rinsky followed Infante. Correct?
A. Yes.
Q. With a study?
A. Correct.
Q. And he provided exposure information in the study. Correct?
A. Correct.
Q. How did Rinsky, if you know, locate the exposure information?
MR. SHOEBOTHAM: Objection, form.
A. NIOSH industrial hygienists reconstructed estimates for that cohort and people in that cohort.
Q. (By Mr. Lubel) Where did they get the estimates for

that cohort?

A. From data available in -- in the original cohort. I mean, it was -- it was data that had already been collected that -- but had not been analyzed.

Q. Collected by Infante but not analyzed?

A. I don't -- the government -- government does their studies differently. They hire lots of subcontractors that go out and see the things. So I don't really know specifics beyond what I just told you. But it was already in their possession either through their contractors that were assembling the data or their own.

Q. Here's why I'm asking you: There's at least three theories of the Pliofilm exposures. Correct?

A. Not theories. They're three estimates that are -- that only two -- two of which tend to be used today.

Q. But they're different, is my point.

A. But there's three different ones.

Q. They're not -- there's three different ones, is my point. They don't all agree?

A. Correct.

Q. So what I'm trying to find out is how Rinsky got his data and then how Paxton the got his data and then how Paustenbach got his data, if you know. If you don't know, just say you don't know.

A. Okay. I don't know how Rinsky got his data.

Paustenbach, who helped Paxton assemble that update, got the records under freedom of information from the federal government. So that's how they got the data.

Q. Do we know if Rinsky had that same data?

A. Yes.

Q. So basically what Paustenbach and Paxton did was is they came up with a different estimate based upon at least in theory the same data that Rinsky had?

A. Correct.

Q. And do we know whether the data that they all had was actual exposure monitoring, you know, an employee wearing a badge, or whether it was grab samples or whether it was area monitoring? Do we know that kind of detail?

A. I don't recall it, but there are published papers that discuss that.

Q. Okay. Do you recall whether that was one of the weaknesses with the study was the -- how the estimates were made and what they were based on?

A. The big -- the biggest debate or differences between the estimates refer to what the exposures were during the end of the war years. The very early part of the cohort are where the big differences -- well, the difference is enough to change the ppm year distributions -- occur. That I do recall.

Q. And in summary is the primary issue in that regard that the Paustenbach camp takes the position that because it

was the end of the war years, the plants were going at full capacity, if not in excess of full capacity, and therefore you'd expect higher exposures than what Rinsky had estimated?

A. I don't recall the specific arguments. I have not looked at them in years.

MR. LUBEL: Okay. Can we take a short break, John?

MR. SHOEBOTHAM: Sure.

(Recess from 1:57 p.m. to 2:09 p.m.)

Q. (By Mr. Lubel) I'm going to mark some exhibits from the documents you brought with you today. Okay?

A. That's fine.

(RAABE Exhibit Nos. 11-12 were marked.)

Q. (By Mr. Lubel) Number 11 is out of your file. Can you identify that for me?

A. Just a few shorthand notes that I took along the way discussing James Boren.

Q. Is that all the notes that you have for this case?

A. Yes, sir.

Q. Thanks. Are you relying on anything in your notes for your opinions?

A. Well, the notes state that -- yes, I am.

Q. Which ones?

A. Well, he was a pipefitter/welder. He's got an AML-M3 with the only cytogenetic abnormality being T15:17

transportation. He was a smoker and at the end of his -- when he left the petrochemical industry he started a boot/shoe repair shop. I mean, it's just pertinent facts of his history.

Q. Did you get that from his deposition?

A. Yes, I believe.

Q. The notes that we see in Exhibit No. 11?

A. Yeah.

Q. So you read Mr. Boren's --

A. Well, his --

Q. Did you read Mr. Boren's deposition?

A. Yes. Yes. I mean, I got the diagnostic information and some other documents.

Q. Medical records?

A. Yes.

Q. Is it safe for a person to stick their hands in pure benzene every day for three years?

A. It's probably unwise.

Q. Why?

A. I can't imagine anybody actually doing it, because benzene is so defatting to the skin that they would have had cracked skin. They would also increase their potential exposure.

Q. And why would it be unwise to stick your hands in benzene every day for several years? Pure benzene, from a health hazard standpoint?

A. Well, I don't believe anybody would, but the reasons for that is that it would so severely defat the skin, they would have severe skin problems, one; two, they would begin to accumulate some inhalation exposure and some dermal exposure to benzene.

Q. And what would be their hematological risk to those exposures?

A. I can't comment on that.

Q. Why?

A. Because I've not attempted to do an evaluation on the inhalation of dermal exposures specific to that scenario you just said.

Q. But you've been an epidemiologist for a number of years working specifically on benzene issues. Correct?

A. Yes.

Q. And in all the years that you've been doing this, have you ever seen the question posed as to whether or not it's safe for somebody to stick their hands in pure benzene?

A. I think I agree it's -- well, safety is -- I -- when you use the word "safety," I start to balk, but it's certainly not something that should be done.

Q. Would you as a -- you're a medical doctor, too. Correct?

A. No. Doctor of Public Health.

Q. Right. In your role as an epidemiologist that knows

a fair amount about benzene and the health hazards associated with benzene, if you had been approached by Mobil Oil, for example, and said, "Hey, we've got pipefitters and other crafts running around here washing their tools in benzene every day, pure benzene, what do you think about that, Dr. Raabe?" what would you have said to them?

A. Stop it immediately and don't let it continue.

Q. Back to my initial question which is: Have you ever seen the question posed as to what the exposure levels are for somebody that sticks their hands in benzene?

A. No.

Q. Would it surprise you if the industry organization, the Chemical Manufacturers Association, actually posed that question?

A. No.

Q. Do you know if it was ever answered?

A. I don't know.

Q. If it was answered, you don't recall what the answer is. Correct? As far as what the exposure levels are?

A. I don't recall either part of that process, that they asked it or that an answer was ever developed nor did I see an answer.

Q. But as you sit here as an expert that Mr. Shoebotham hired, you may be able to make general comments about whether it's wise or unwise for a worker at any time to stick their

hands in benzene, whether it be for a day or three years, but you don't feel as though you're in a position to address whether or not that type of exposure could lead to a hematological disease?

A. I would look to other scientific disciplines to give me a dose estimate that I could put into context with what we know about dose responses for benzene-induced disease.

Q. What discipline would you go to for that information?

A. Probably a combination, depending on the level of expertise. Industrial hygiene would be part, someone with familiarity with dermal exposure. Could be the same person or it could be someone else.

Q. Who are you thinking of that knows something about dermal exposures?

A. Well, it could be toxicologists in combination -- a toxicologist in combination with an industrial hygienist, or it could be an industrial hygienist with that kind of training.

Q. Have you ever consulted specifically with somebody in that area that specializes in dermal exposures to benzene?

A. No, I have not.

Q. Do you know if anybody has?

A. In the world?

Q. Anybody that you know that has done so.

A. I know John Spencer has done work in that area.

Q. In what regard?

A. Well, I'm aware he's done some work with liquid wrench, and I'm also aware that he has done some work in this case.

THE REPORTER: Who? Done some work with who?

MR. LUBEL: In this case.

THE WITNESS: In this case.

THE REPORTER: Before that who did you say?

THE WITNESS: With liquid wrench.

Q. (By Mr. Lubel) Have you talked to John Spencer about his opinion in this case?

A. No, but I've seen a one-page summary of exposure information.

Q. And does his one-page summary of exposure information address exposures to a person that sticks their hands in benzene every day for a number of years?

A. The piece of the document that I have, if there's more of it, does not speak to that.

Q. Does or does not?

A. Does not.

Q. Okay. Have you talked to Mr. Shoebotham about whether Mr. Spencer is going to create a document that addresses that exposure?

A. No, I have not.

Q. As you sit here today, do you know whether that exposure is sufficient to cause leukemia?

A. Mr. Parker also came up with some estimates that ostensively included derma. I have not seen any background material on those -- on those calculations, but the number he comes up with is substantially below the 200 ppm years that the literature suggests is the lowest level where increased AML risk occurred. Significantly occurred.

Q. Well, Rinsky's and White's data back in the early '80s came up with data that suggested 10 parts per million could increase the risk of leukemia. Correct?

A. That is total leukemia.

Q. Pardon me?

A. I believe you're talking about total leukemia versus acute myelogenous leukemia.

Q. All right. You've seen the Rinsky data and you've even seen the EPA 1998 breakdown on it where they have a table in there that talks about increased relative risk of AML specifically from exposures in the 40 part per million to the 200 part per million range?

A. I would have to go back and look at them. What I'm thinking of is Rinsky's updated paper where he doesn't see statistically significant increase risk for leukemia until approaching 400 ppm in his most recent update.

Q. What update was that?

A. 2002.

Q. What did the previous update say?

A. It was statistically significant in I believe the 200 range as well.

Q. Not in the 40 to 200 part per million range?

A. I don't recall.

Q. Well, surely you're aware that there are peer review published articles that address risk of AML being significantly increased by exposures to benzene less than 200 parts per million. Correct?

A. There are several papers out there, yes.

Q. One of those is the Hayes, Yin article regarding the China studies done by the National Institute of Health and the Chinese equivalent. Correct?

A. Hayes has a few tables that address exposures that are lower than 200 parts per million.

Q. In fact less than 10 parts per million. Correct?

A. I don't recall that as being statistically significant.

Q. In what level do you recall him taking the position that it was statistically significant?

A. I don't recall. We would have to pull the paper and start looking at it.

Q. Do you think the Hayes and Yin article is in here?

A. You have two piles of my papers.

Q. That's mine.

A. Oh. Yes, it should be, unless we've already pulled

it out earlier. I don't think. The Hayes paper should be there.

Q. Give it back to you?

A. I have the paper.

Q. Let's mark that as Exhibit No. 13. You can go ahead. I'll just write on it in just a second. Thanks.

A. I'm waiting for a question. What...

Q. Oh, you found the Hayes and Yin article?

A. Yes.

Q. What's the date of it?

A. 1997.

Q. There was a 2000 version, wasn't there?

A. Not of the new -- this is the most recent epidemiologic update.

Q. I recall talking to you in the Cowey case about the -- was it 2000 or 2001?

MR. BLACK: 2000.

Q. (By Mr. Lubel) -- 2000 Hayes and Yin article. It's the one that specifically addressed MDS and NHLs?

A. Oh, the -- the one that essentially responds to some of the criticisms?

Q. I don't recall that that was the nature of it, but...

A. I didn't bring that with me.

Q. At any rate, looking at the article that you have, do you recall that there was statistically significant excesses of

what I think they categorized as acute nonlymphocytic leukemias at low level benzene exposures?

A. The cumulative -- yes, there is.

Q. Do you remember what the -- you've got a chart, so --

A. Right. Looking -- make the record easy. Looking at table two, the first statistically significant risk occurs in the category 40 to 99, and it's also significant in the category over 100 ppm.

Q. 40 to 99 parts per million?

A. Yes.

Q. And is that for AML specifically?

A. AMLL.

Q. Which is acute nonlymphocytic leukemia?

A. Correct.

Q. What would that include other than AML? MDS?

A. No, it would not include MDS. It would include a few subtypes that are slightly different than AML.

Q. And I take it from the testimony that you've given previously that you still believe that benzene exposures at the right level can induce myelodysplastic syndromes, otherwise called MDS?

A. Yes.

THE REPORTER: I need just one second.

MR. LUBEL: Sure.

(Discussion off the record.)

Q. (By Mr. Lubel) Do you believe that the levels that are required for there to be a benzene-induced MDS are the same that are required for a benzene-induced AML? Or do you think they're different benzene exposures?

A. I don't -- they're probably similar but not exact.

Q. Which one is lower?

A. Depending on the nature of the MDS, probably MDS may be a little lower.

Q. Do you know where you're going with that, or you're just saying, "I think it's lower and I'm not sure where"?

A. I think it's lower and I'm not sure where.

Q. Fair enough.

MR. LUBEL: Do you have a paper clip?

(Discussion off the record.)

Q. (By Mr. Lubel) In your file I see a deposition and trial list that I'm going to mark as Exhibit No. --

MR. LUBEL: What are we on?

THE REPORTER: Fourteen.

Q. (By Mr. Lubel) -- 14.

A. Yes, that's what that is.

(RAABE Exhibit No. 14 was marked.)

Q. (By Mr. Lubel) Is that something you created?

A. Yes.

Q. Is it updated?

A. Best as I can.

(RAABE Exhibit No. 15 was marked.)

Q. (By Mr. Lubel) Exhibit 15 is a copy of your curriculum vitae?

A. Yes.

Q. Who are you employed by right now?

A. I'm principal scientist for a company that I own called Health Risk Sciences.

THE REPORTER: Health what?

THE WITNESS: Risk Sciences.

Q. (By Mr. Lubel) Do you have any employees?

A. No.

Q. You're the --

A. I'm the only full-time employee.

Q. I take it your wife helps? She is the chairman of the board? Come on, be honest?

A. She does good Xeroxing.

Q. I'll be sure and send her a copy of this.

(RAABE Exhibit No. 16 was marked.)

Q. (By Ms. Lubel) All right. What is Exhibit 16?

A. 16 is a list that I believe to be correct of materials sent to me by Mr. Shoebotham's office.

Q. Okay. Are you relying on all of the information contained within Exhibit 16 for your opinions?

A. No.

Q. Can you put a check mark next to the stuff you're

relying upon?

A. When you use "relying upon," do you mean -- I'm not sure I understand what relying on -- when you use the term.

Q. Something you may sit down in front of the jury if this case ever gets to trial and say --

A. And make statements from?

Q. Yeah.

A. Okay.

Q. Exactly.

A. I don't recall offhand the medical -- where I pulled those medical records from, so I'm just going to check the medical records that are listed here. Possibly need it.

May I just ask --

Q. Yes.

A. -- him to confer with me?

Q. Sure.

(Discussion off the record.)

Q. (By Mr. Lubel) What you have done at my request here is on Exhibit 16 you've checked off the stuff out of the materials you've been provided by the defense lawyer, Mr. Shoebotham, that you know at least as of right now that you intend to potentially use at trial if asked any questions?

A. Potentially.

Q. I'm not trying to be tricky, but you have marked a section that says "Documents Produced by TDCC," and then it's

got a bunch of Bates numbers, 1 through 2,534?

A. I believe those are -- you will see I checked -- there are a couple of other submissions of the same type that I checked. Some of those include -- the ones I'm thinking of mostly are the whole variety of studies conducted by Dow that were --

Q. What kind of studies are they?

A. We discussed -- I mean, I brought a few of them with me. It's essentially the production -- all the studies that that they did relevant to benzene and relevant to Freeport and Midland.

Q. And are you including toxicological studies?

A. No.

Q. Even if they are within these Bates numbers, you're not using those for your opinions. Right?

A. Own -- like when you say toxicologic, you mean purely animal study?

Q. Right.

A. No, I wouldn't be using those.

Q. Like fish.

A. I would not be using them.

Q. Are there any particular Dow studies that we've not already talked about in your deposition that you're relying upon for your opinions?

A. Well, we didn't specific -- I believe we numbered

them all, but we didn't discuss all of the Freeport updates.

Q. Other than those, the ones we've marked as exhibits here in the deposition. I'm trying to get you to think about stuff that we don't have in front of us here at the deposition?

A. Possibly going -- I possibly may need to go back to some earlier Midland -- Midland papers, doctor's original papers, for example, but I don't know at this time.

Q. Okay. Are you relying on Dr. Natelson's deposition?

A. Not directly. I checked it only when he makes statements that I support and agree with.

Q. Do you agree with all the statements he made in his testimony under oath?

A. I don't remember all the statements, but I probably don't agree with every single one.

Q. Do you remember the ones you disagree with?

A. Not specifically, no.

Q. Do you remember any of the disagreements you have with Dr. Snodgrass' deposition?

A. His -- the draft I received was very difficult to read, so I have not really comfortably reviewed all of that yet.

(RAABE Exhibit No. 17 was marked.)

Q. (By Mr. Lubel) Exhibit 17 is the John Spencer exposure information that you've received so far?

A. As well as Frank Parker's estimates, yes.

Q. But who prepared the document to your knowledge?
A. I don't know who prepared the document other than it contains the estimates from Frank Parker and the estimates that I have been told are John Spencer's.
Q. Who told you they were the estimates for Spencer?
A. I believe I got that out of reading the Natelson deposition and was -- I believe it was confirmed by Mr. Shoebotham.
Q. So you have not spoke to Mr. Spencer about this case?
A. No, I have not.
(RAABE Exhibit No. 18 was marked.)
Q. (By Mr. Lubel) And then Exhibit 18 is the affidavit of my client that Mr. Shoebotham sent you. That's in your file. Correct?
A. Yes.
Q. I take it you've relied upon that for exposure information. Correct?
A. I relied on it only to the extent that it's an allegation by Mr. Boren of what he did.
Q. But it's information that you in your capacity as an epidemiologist has to look at in order to make an assessment?
A. Yes.
Q. In addition to depositions and other things?
A. Yes.
(RAABE Exhibit No. 19 was marked.)

Q. (By Mr. Lubel) Exhibit 19, if you'll just generally summarize what that consists of?

A. These are essentially, for the most part -- well, correspondence between Mr. Shoebotham and myself and for the most part our transmittals or plan to meet announcements, and the last item attached to it is the notice of the deposition.

Q. Thanks. In Rinsky's 2002 article that you brought with you, did he give different exposure information from what he had in the previous versions, '81 and '87?

A. No. The cohort is terminated so the NIOSH estimates that he used in the original study are the same ones being used today.

Q. Do you know how he got different exposure information, or did he get a different exposure information?

A. There was no more -- there is no -- the plant is closed. There is no more exposure.

Q. Did he interpret the exposure information differently in the 2002 version, or did he just have an update of additional information that changed the numbers?

A. He updated the mortality of the cohort using the same exposure information.

Q. And that gave him different numbers, if you will?

A. It gave him additional cases.

Q. So his 2002 paper, if you will, on the Pliofilm cohort does not invalidate his '81 or his '87 version of the

paper. Correct? It's different?

A. No, it doesn't invalidate it.

Q. It's just different?

A. It's just the most recent information on the cohorts mortality.

Q. I'm going to show you some information. You can tell me what you're using it for. The American Cancer Society, Leukemia -- Acute Myeloid (Myelogenous), what are you using that for?

A. I'm using that for a general description of leukemia and acute myeloid leukemia in this country.

Q. So you're not using this article specifically for things like latency periods and that kind of stuff. Right?

A. Right.

Q. Same question with the National Cancer Institute article, pages 1 through 39, that you brought with you?

A. General epidemiology of leukemia and acute myelitis.

Q. I'm going to mark these series of articles as one exhibit, No. 20. Okay?

A. That's fine.

(RAABE Exhibit No. 20 was marked.)

Q. (By Mr. Lubel) If we get to an article where there's something other than general background information about leukemia you're using, will you tell me? Stop me, please.

A. If I recall it at the time, yes.

Q. The Estey article on Acute Myelogenous Leukemia?
A. This one discusses -- begins to discuss in more detail cytogenetic abnormalities and those that are potentially related to secondary or environmental exposures.
Q. All right. We're going to go ahead and mark that as Exhibit 21.
(RAABE Exhibit No. 21 was marked.)
Q. (By Mr. Lubel) Does that article exclude translocations 15 and 17 from being benzene induced?
A. It doesn't speak directly to the benzene.
Q. What does it speak to?
A. As -- if I'm recalling it correctly, it discusses what is -- what is known about the -- well, it talks about the basic epidemiology of M3, and it -- I believe it also talks about therapy related and does discuss latency, age, some of the -- related to therapy related or types of -- it doesn't speak specifically to benzene.
Q. Does it speak indirectly to benzene? Is the word "benzene" in here?
A. I don't believe the word "benzene" is in there.
Q. Okay. Move on to Exhibit 22 which appears to be an article titled Acute Myelogenesis Leukemia by Seiter, S-E-I-T-E-R, and others?
A. Yes.
Q. What are you using that for?

A. Same as the previous article.
Q. Okay. Can we keep that as part of 21 then?
A. That's fine with me.
Q. How about the next one, Cancer Epidemiology?
A. That's just general epidemiology.
Q. Keep part of 21 too or is that a different category?
A. It's not specific to APL. It belongs more with the general epidemiology.
Q. Is there any particular section in what I'm going to mark as Exhibit 22, Cancer Epidemiology and Prevention, Second Edition, that you're using for your opinions in this case?
A. Depending on what I'm asked, I may refer to it for what are known risk factors for acute myelogenous leukemia.
(RAABE Exhibit No. 22 was marked.)
Q. (By Mr. Lubel) What are the known risk factors that are generally accepted in the scientific community?
A. Radiation, a variety of chemotherapeutic drugs, and in some special circumstances some viruses, and of course benzene in high dose, long duration.
It's getting late in the day, but that's the ones that come to my mind right now.
Q. Those are the ones you typically see in a textbook. True?
A. Yes, generally.
Q. There may be articles out there that suggest that

there's other risk factors. True? Like smoking?

A. Yes, smoking. Very good. Thank you.

Q. You don't typically see smoking in the textbooks but that doesn't mean that smoking can't be related to AML?

A. Well, the surgeon general has recently concluded that it is.

Q. Well, that's my point, though.

A. Yes.

Q. Just because it's not mentioned in an -- every epidemiology textbook doesn't mean that there aren't articles out there that are sufficient to say otherwise?

A. Well, once again, individual articles would not do it. It would have to be a body of literature. The paper that we're talking about here, this chapter on leukemia, summarizes through '96 the state of the science about risk factors and epidemiology of leukemia.

Q. Well, of the known risk factors, let's go through them. Smoking. Did smoking contribute to cause Jimmy Boren's leukemia?

A. It may have confounded or contributed to his disease, yes.

Q. You say "may have." Based upon reasonable scientific principles, what is your opinion as to whether smoking contributed to cause in whole or in part his leukemia?

A. As it applies to M3s, we don't have a lot of data.

But it is -- and depending on -- and he didn't smoke that -- in the scheme of things, his pack years are not that high, if correctly reported. So I sort of view it as perhaps contributing to but probably not causing directly. In other words, at most it's contributing, it's adding, but I don't...

Q. Well, that's how cancer works. Very seldom can you pick one thing and say that's what caused it. Right?

A. Unless you have very specific dose -- dose information.

Q. But, I mean, if a person is exposed to multiple carcinogens, the theory that's taught in medical schools is that each and every exposure to those carcinogens can take you down the path of cancer?

A. Well, I hope they're not teaching that in medical schools today, but -- because that's a gross oversimplification and probably incorrect in many parts.

Q. Is that incorrect?

A. It's very specific to which cancer you're talking about. In other words, making it as a generalization is -- is potentially...

Q. Well, let's take smoking, for example, with Jimmy Boren. You're willing to say that it perhaps contributed to cause his leukemia, but you won't say that you are of the opinion that it was a cause. Not the sole cause. You won't say that it was a cause of his leukemia. Correct?

A. I reserve -- I guess I'd want to say I want to reserve that, because I plan to go back to the primary papers that the surgeon general cites and read those. I have not read those all. Right now I'm willing to say that it contributed, but how strongly I make that statement, I think I'll have to reserve my opinion.

Q. Based upon papers that you didn't have before you today. Correct?

A. Well, based on papers that I have not been able to retrieve yet.

Q. You don't have them here at the deposition?

A. Correct, but I don't have them anywhere either. I have got to get them.

Q. Well, the second risk factor that you referenced for AML was radiation?

A. Yes.

Q. Did radiation contribute to cause Mr. Borne's leukemia?

A. Based on the information I have of his work history, I would say no.

Q. Because you don't see any evidence of exposure to radiation before his diagnosis of leukemia. Correct?

A. Correct.

Q. Do you see any evidence that would suggest that one or more viruses contributed to cause Mr. Boren's leukemia?

A. I don't have enough information to write any conclusions on virus.

Q. One way or the other. Correct? What information would you need?

A. I don't think it plays for Mr. Boren, but in some other lymphomatic diseases I would like to know more about farming and animal husbandry and HIV and -- you know, there are a number of -- I didn't see any virus markers in any of his medical history that would be related.

Q. See evidence that would cause you to suspect or opine that Mr. Boren had previous chemotherapy treatment that caused his leukemia?

A. I have not seen anything in the records.

Q. We can rule that out. Correct?

A. Yes.

Q. And we can rule out the radiation. Correct?

A. Based on the data available to date, yes.

Q. And at least based upon the information you received on Mr. Boren's to date, we can rule out viruses for now?

A. Yes.

Q. What we're left with is smoking, which you want to look at the surgeon general opinions before you make a final opinion on that. Correct?

A. Yes.

Q. And then I take it that it's your opinion that

benzene didn't contribute one iota to Mr. Boren's leukemia. Correct?

A. Pretty much everything I have read says that the form of M3 he has is a spontaneously-occurring leukemia and is not related to environmental or occupational factors based on the current state of the science.

Q. So it's your opinion that no acute myelogenous leukemias of the M3 variety can be related to benzene exposures. Correct?

A. No. I'm speaking specifically to the photogenetic profile of Mr. Boren.

Q. 15:17 translocations?

A. Only.

Q. And you have earlier in the deposition told us the studies that you're relying upon, and we've marked those as exhibits for that proposition. Correct?

A. I believe so.

Q. The Otto Wong study dated 1995, "Risk of acute myeloid leukemia and multiple myeloma in workers exposed to benzene," are you relying on that for your opinions in this case?

A. Yes.

Q. In which regard?

A. Discussing the lower threshold of what AML risk -- when AML risk increases.

Q. And what -- how were -- how was the exposure information collected for this Otto Wong study?

A. It was provided by NIOSH.

Q. What was he studying?

A. Pliofilm cohort.

Q. And has the government accepted his article as authoritative on the issue of risk assessment?

A. I don't know what that means. What do you mean?

Q. For instance, with EPA, when you see them discuss risk assessment in relationship to the Pliofilm cohorts, you see them talking primarily about Rinsky's data.

A. Well, that is Rinsky's data.

Q. But I'm talking about Rinsky's articles, and you'll see them be very critical of long stuff and kind of exclude his summary of it. Have you seen that?

A. That's an incorrect characterization. The -- the difference between approaches is that the EPA still uses total leukemia for their risk assessments, and Dr. Wong and I and others believe that only AML is the relevant cell type that should be used in the calculation.

Q. All right. What assumptions did Wong make in this article about the exposure levels?

A. He used Rinsky's exposure levels in one calculation, and he used Paustenbach's in another calculation.

Q. Well, if he used Rinsky's exposure levels in one

calculation, shouldn't his conclusions have been the same as Rinsky's?

A. They are applied to acute myelogenous leukemia. Rinsky's are applied to...

Q. And that would be in table one?

A. At least, yes. Yes.

(RAABE Exhibit No. 23 was marked.)

Q. (By Mr. Lubel) And we've marked that as Exhibit 23?

A. Fine.

Q. Next article we'll mark as Exhibit 24.

(RAABE Exhibit No. 24 was marked.)

Q. (By Mr. Lubel) What are you using that for?

A. This and Rinsky's -- these are two letters to the editor that discuss that exposures much beyond 15 to 20 years; in other words, only the most recent 15 to 20 years of exposure are relevant to risk assessment.

Q. Do you agree with that?

A. Yes.

Q. Why is that?

A. Well, because -- let me just say the -- right now we have observational data from epidemiology studies and both Rinsky and Glass and others that -- that -- that when they lag the exposures, only the most recent exposures are relevant. And we -- and there -- biologically there's lots of speculation what that might be, but it suggests that the earlier exposures,

if they did -- if they occurred, the body recovers from it.

Q. Is there a consensus that exposures longer than 15 years prior to diagnosis are not significant disease-forming process?

A. Not in the absolute sense that you said it. I think there is a consensus that earlier exposures is relevant, but the specific cut periods may be dose -- dose rate dependent. So...

Q. Could depend on whether they're peak exposures or chronic exposures that are more important?

A. Only speculation.

Q. Nobody knows?

A. We just know that the early exposures -- we know that just the most recent exposures seem to be the ones that afford the risk, even in the Pliofilm.

Q. You're aware of the health watch study at Australia. Correct?

A. Yes.

Q. And I take it that you agree with the findings that they made 2001?

A. No. Their case control study? Like what?

Q. The Australian Institute of Petroleum.

A. The cohort study? Which study?

Q. The health watch study that Exxon-Mobil sponsored. They sponsored multiple studies.

A. There are multiple papers.
Q. Do you have them here?
A. I have the two most recent.
You want me to pull them out to save time?
Q. Yes. Thanks.
MR. SHOEBOTHAM: Take about a three-minute break.
MR. LUBEL: Sure.
(Recess from 2:59 p.m. to 3:06 p.m.)
(RAABE Exhibit No. 25 was marked.)
Q. (By Mr. Lubel) Can you identify Exhibit 25 from your files?
A. Yes. It's a paper by Deborah Glass. It's case control study of -- conducted in Australia.
Q. Was it a study of benzene-exposed workers?
A. Yes.
Q. And does the study address what benzene exposures caused leukemias and their cohort in their study group?
A. Can you restate that?
Q. Does the Glass study that we marked as Exhibit 25 address specific benzene exposures leading to certain outcomes like leukemia?
A. Yes.
Q. And what do they say about AML?
A. They find an association between acute nonlymphocytic

leukemia and benzene exposure.

Q. At any particular levels?

A. For AML?

Q. AML.

A. Significant findings above 8 ppm years.

Q. How many years?

A. Eight ppm years.

Q. See that real quick. In the abstract, the conclusion section, the last sentence, it reads, "No evidence was found of a threshold cumulative exposure below which there was no risk." Did I read that correctly?

A. That's a modeling result. You read it correctly.

Q. Do you agree with that statement?

A. No.

(RAABE Exhibit No. 26 was marked.)

Q. (By Mr. Lubel) Exhibit 26 from your files, if you can identify that for us?

A. This is a paper, first author is Gun. It's an update of the cohort mortality and cancer incidence study in the Australian petroleum industry.

Q. Same cohort that Glass wrote about?

A. Glass is case control study from that cohort.

Q. So it's a different type of study?

A. Yes.

Q. Do they reach different conclusions?

A. Yes.
Q. In what respect with regard to acute myelogenous leukemias?
A. Gun doesn't find much of an excess of leukemia in the study.
Q. So he didn't quibble with the exposures. He just doesn't think there's more leukemias than -- or an excess?
A. They -- the case control study has different numbers than the cohort study, and Gun has difficulty trying to reconcile that. They're actually part of the same research team.
Q. Well, who put Gun up to this? Exxon?
MR. SHOEBOTHAM: Objection, form.
Q. (By Mr. Lubel) Do you know?
A. Put what? Put up?
Q. What caused Gun to look into a study that was done by Glass in 2003, caused Gun to write -- to do a different study in 2004? We know some industry member put him up to it. Who was it?
MR. SHOEBOTHAM: Objection, form.
A. That's not correct.
Q. (By Mr. Lubel) Who put him up to it?
MR. SHOEBOTHAM: Objection, form.
Q. (By Mr. Lubel) Do you know?
A. Gun is part of the same university group doing the

health watch studies.

Q. I understand that but the original study that I have here -- let me see it. The 2001 version, it's about two inches thick. I'm sure you've seen. Right?

A. Yes, but you -- but there is also -- there's also a cohort paper that I think is also on the website that Gun is responsible for. That's the case control study.

Q. Were you aware that for the case control study that the project advisory group were people from Shell and Exxon?

A. I don't recall Shell, but I knew that there was one Exxon member.

Q. Well, can you explain how the 2001 version referenced a -- an excess of CLLs and how the version that we see here by Glass in 2003 doesn't make a reference to CLLs being in excess? What changed, if you know?

A. I don't know specifically.

Q. And then we've got Gun in 2004 saying almost exactly the opposite from what Glass said in 2003. Correct?

A. Not the opposite. Gun points out that there are inconsistencies between what they find in the cancer incident study and the cancer mortality study and the conclusions that one would reach from the case control study. That -- that's the level of criticism.

Q. And which one is right?

A. Don't know.

(RAABE Exhibit No. 27 was marked.)

Q. (By Mr. Lubel) And Exhibit 27?

A. Exhibit 27 is -- is three letters: One from Dr. Schnatter to Dr. Glass, one from Bernie Goldstein to Dr. Glass, both criticizing the case control study, and Dr. Glass's reply.

Q. Who does Schnatter work for?

A. Schnatter works for Exxon Biomedical Sciences.

Q. Did you know him when you worked in epidemiology?

A. Yes.

Q. Was he part of the CMA?

A. No. What -- I don't think -- I don't know. He would have been very young in his career, so I don't recall.

Q. His employer is part of the CMA? Exxon.

A. Exxon, yes.

Q. Do you know who paid Bernie Goldstein for his critique?

A. Dr. Goldstein is Dean of the School of Public Health, University of Pittsburgh, and I think he did it on his own.

Q. He's written a lot of about benzene?

A. He has lots of opinions about benzene. Correct.

Q. It goes back many years.

A. Yes.

Q. He actually had a book that he authored about benzene. Correct?

A. Sort of before my time.

Q. Mine, too.

All right. The remaining materials that you've brought with you that we have not otherwise marked as an exhibit I'm going to mark as 28 and ask you if you would just go through and tell us how you're using them for your opinions in the case.

(RAABE Exhibit No. 28 was marked.)

Q. (By Mr. Lubel) Just let you flip through at your leisure?

A. The first paper is a paper by -- actually it's a letter -- letter to the -- Dr. Hayes and an exchange. It's followed by several other papers, essentially varying criticisms of the Chinese studies.

There is a paper not included here that I did not bring with me by a gentleman by the name of Badinski (phonetic) in the same vein. But it actually discusses the problems with the Chinese studies.

Q. Which all relate to exposure assessments as I recall. Is that true?

A. Cohort -- well, a little bit more than that. Some is case ascertainment, but probably the bulk of it is exposure-related problems.

The next two documents are excerpt papers from the tox profiles of gasoline and benzene prepared by the Agency

for Toxic Substances and Disease Registry, ATSDR, in case I rely on them as part of my discussion on odor threshold.

Q. That is the odor threshold for benzene?

A. Fairly low. In a range around 1. -- 1.2 to 2.5.

Q. For all people?

A. No. Reception of odor is very subjective and that's -- and it would be used in the discussion, if the discussion ensues, about perceptions of odor.

Q. And are you relying upon the study that you just referenced on the odor threshold for benzene?

A. Yes. I would consider -- I mean, there are many others. There's a whole list of different studies, but the US ATSDR seems to think that range represents some of the better studies. So, yes, I am relying on it.

Q. And you're talking about the toxicological profile for gasoline?

A. And that odor threshold discussion is for gasoline. This one is for benzene.

Q. Okay. Thank you.

A. More for historical purposes I brought the ACGIH, the most recent ACGIH documentation on benzene, from which I may extract specific information about TLVs or vapor pressure, you know, things like that.

Also for historical perspective I brought the NIOSH 1976 criteria document as it relates to when -- when a

consensus of science believed that leukemia is potentially related to benzene.

Q. What year?

A. '74 they raised the question; '76 they felt that they -- that it was getting close.

Q. And when did the API suspect that there was a relationship between benzene and leukemia?

A. I don't know.

Q. Would 1948 surprise you?

A. Yes, as it relates to leukemia it would.

Q. It would? But if I could show you the 1948 toxicological review on benzene where the American Petroleum Institute acknowledges that there -- they suspect that benzene causes leukemia, that would -- would that change your opinion?

MR. SHOEBOTHAM: Objection, form.

A. No.

Q. (By Mr. Lubel) Why not?

A. Because there were no epidemiologic investigations in that time period that could have come to those conclusions. So, however those conclusions, if you're correctly summarizing them, came about, it would not be based on scientific evidence.

Q. Well, do you know who Phillip Drinker is?

A. I think he was a Harvard professor.

Q. Was he an epidemiologist?

A. I don't think so.

Q. Was Selocough (phonetic) an epidemiologist?
A. No.
Q. What was going on with the field of epidemiology in 1948? Was it a robust field?
A. I wasn't there.
Q. What's your hunch from, you know, going to school to become an epidemiologist, what was going on back in the '40s with that field?
A. Occupational and environmental epidemiology were just beginning in this country. They were more active in Europe but just beginning in this country.
Q. Are there any diseases that you're aware of that were related to exposures before there was epidemiology?
A. Yes.
Q. Like vinyl chloride?
A. I -- my mind was not where you were. I don't recall the vinyl chloride science issues, so I can't answer that. I was going to speak to the plastic anemia in benzene-exposed groups. There -- there was sufficient robust clinical information that some causation conclusions could be drawn in the absence of formal epidemiologic investigation. That would have been my -- my example in this context.
Q. Okay. Continue on.
A. Next document is the IRIS -- Integrated Risk -- US EPA benzene IRIS documentation. It's relevance would be that

it's a 2003 document where the US EPA concluded for a second time that the Chinese studies were unsuitable for doing dose response and risk assessment.

Q. Well, what's the difference between risk assessment and whether or not a chemical can cause a particular outcome?

A. One is an intrinsic characteristic of the material that at some dose, at some duration, it could cause a disease, a specific disease. For risk assessment purposes -- and when I use the term "dose response," they're similar issues you are attempting to characterize when that risk occurs and how large that risk is. We've know for sometime now that benzene can cause AML. It's ultimately important for public health and occupational health to know accurately the shape of that dose response, and not just at the high level but at the low level.

Q. And that's what you believe the industry-funded studies in China regarding benzene are out to finally assess once and for all?

A. Well, there's no finally in science ever. Hopefully the studies will be well conducted and can contribute new information to that, yes.

The next document is a 2004 -- only a few pages of a report of a US surgeon general entitled "The Health Consequences of Smoking." In this surgeon general report using the Hill criteria they conclude that acute leukemias, specifically acute myelogenous leukemias, are causally related

to cigarette smoking.

Q. At any dose?

A. That's why I want to read the underlying papers. The surgeon general does not remark on that.

Q. But what's your hunch? I'm not holding you to it because I understand that you want to go look at that paper.

A. Multiple pack years.

Q. Like more than 10?

A. More than 20 probably.

Q. More than 20 pack?

A. Hunch.

Q. That's a hunch. Got ya.

A. Just so we all agree.

Next paper is the -- is a paper by Robert Rinsky, et al, 1987, here for historic purposes in case we -- anyone discusses some of that.

The next paper is 1994 Paxton, there for the same reason, historical context.

The next paper is Bob Rinsky's 2002 update which he provides the most recent dose response information and the most recent cases for the Pliofilm cohort that he originally did.

Q. You can continue. Thank you.

A. The next paper is a paper by Robert Schnatter, et al. It's an analysis of the Pliofilm cohort, specifically looking

at concentration levels, not just ppm years, not just cumulative exposure. So it supports what I think a number of us believe. It's not just -- not just ppm years but some thresholds of concentration that you have to have besides just cumulative.

Q. What plant did he state?

A. He used the Pliofilm data using both Rinsky estimates and Paustenbach estimates and Crump estimates. Like I said, these are now some of the publically available data sets.

The next paper is in case it becomes a discussion, a broad discussion on petroleum exposed workers. I just brought along a paper that I did on -- the 2000 paper on what is essentially solid tumors.

Q. Is it relevant to Mr. Boren's case?

A. I don't think so.

Q. But just in case I bring it up?

A. You got it.

Q. You're ready?

A. Also brought the TLV documentation, ACGIH TLV documentation for gasoline.

Q. How is that relevant?

A. Probably not but I had it.

Q. What is the NIOSH recommended exposure level for benzene today?

A. I don't know.

Q. Would it surprise you if it's half of what the OSHA level?

A. No, it would not surprise me.

Q. Has NIOSH historically been out in front of OSHA as far as recommending reduced levels of exposure?

A. NIOSH's charge is to provide technical support for OSHA. That's all I know. I don't know what "out in front" means.

Q. Are they the investigative arm for OSHA?

A. Yes, I would think that's a fair characterization.

The next paper -- and actually the next two papers are by Otto Wong. They both relate to gasoline mortality study, essentially the same -- same study, two different variations in publication in case the issue of what are the effects of gasoline exposures are. Probably not related to Boren.

Q. Have you from anyone, including Dow's lawyer Mr. Shoebotham, been told that somehow gasoline exposures may become pertinent to this lawsuit?

A. No, not at all.

Q. I just want to make sure that -- something not out there I'm not aware of.

A. It's material I brought since, you know, it was unclear where some of the exposure discussions may drift to.

The next document is a US EPA 2002 document

looking at the Toxicological Review of Benzene (Noncancer Effects) which contains assessments of toxicity of benzene for other things including noncancer such as thrombocyte cytopenia (phonetic) or something like that.

And the last paper is well put together. It is actually three papers of various durations of Dante Picciano's work on circulating lymphocytes, and the cover page of that is a critique of that body of work.

Q. Was it a published critique?

A. No. It's a letter from Jandl who is a very famous Harvard professor in hematology as to what he thinks about it.

Q. Was Dr. Picciano's work published?

A. Yes.

Q. Do you remember which publication?

A. I believe I had it clipped here. Yes. It was published in Environmental Research. Those are all the papers I have brought with me.

Q. And how do you intend to use the cytogenetic studies that you brought, including the critique?

A. The early one? Oh, these, what we were just talking about here?

Q. Right.

A. I was -- I brought them really to -- you know, I think it's probably more appropriate to deal with the other experts, but this -- the approach of looking for circulating

markers of toxicity in benzene-exposed workers was -- was fairly active in the late '70s through the mid '80s, including several attempts -- neologic attempts to interpret those, see how they correlate with the pin points. And only if asked I would revolve around the discussion that it's no -- surveillance using circulating lymphocytes turned out to be not predictive of any adverse health outcomes and therefore has essentially been discontinued.

Q. When?

A. Probably, except for research purposes, a lot of it was stopped, because I don't think -- I don't even think OSHA endorsed it when OSHA came out with their standard.

Q. Do you have any articles that you believe are authoritative on when it stopped?

A. No, I don't.

Q. Did Mobil, when you were the Director of Epidemiology, run cytogenetic testing on their benzene-exposed workers?

A. No. After -- after Picciano and there are several other papers in literature, we had conversations with our toxicologists and felt that at this stage it was such a researchy technique that we weren't going to get into it.

Q. Before the Picciano paper came out in 1978, had Mobil done any cytogenetic testing of any of their chemical-exposed workers?

A. I don't believe so.
Q. Do you know if Dow studied any of their chemically-exposed workers cytogenetically?
A. Only what I recall the population that we looked at in Freeport.
Q. Do you know if they studied their -- the chromosomes of the Midland people?
A. I don't know.
Q. Do you know if they studied the chromosomes of their people exposed to vinyl chloride?
A. I don't know.
Q. Any other chemical?
A. Don't know.
THE REPORTER: What chemical did you say?
MR. LUBEL: Any other chemical.
THE REPORTER: Before that.
MR. LUBEL: Vinyl chloride.
Q. (By Mr. Lubel) Have you given us all the opinions that you intend to express at the trial of this case regarding Mr. Boren?
A. I think going through these papers we covered it.
Q. In other words, you're not a neurologist. Correct?
A. No, I'm not a neurologist.
Q. You're not addressing any neurological issues concerning them, are you?

A. I am not.

Q. And if I've understood you correctly today, where you have drawn the line on Mr. Boren and his AML, his particular type of AML, and why you do not believe that his benzene exposures contributed whatsoever to it is primarily based upon what you believe is new literature that suggests that translocations of 15 and 17 could not be related to benzene exposures. True?

A. I think that oversimplifies the testimony a bit. I -- it's certainly correct in that the epidemiology which is fairly recent work on -- for myocytic leukemia does not draw any -- all the papers published to date can't find occupational or environmental relationships. So yes, I'm relying on that. And -- but even besides that, everything I have seen from exposure profiles suggests that he did not have exposures sufficient to increase his risk materially.

Q. And that's where I wanted to make sure we were, because even if you and I could reach an agreement on his benzene exposures causing his particular type of AML with his chromosome abnormalities, what I'm hearing you say is that based upon the information you have regarding the alleged exposures to benzene of Mr. Boren, you don't think they're sufficient enough to have contributed to cause AML?

A. Correct.

Q. In any person?

A. As a general proposition they are probably too low.

Q. Okay. And so even if you agreed that Mr. Boren's AML could be related to benzene exposures if the exposures were high enough, you would come down and say, "Look, I've looked at the exposure information that both sides' lawyers have given me, and with that I can exclude with a certainty that Mr. Boren's benzene exposures were not a contributing factor to his AML." Is that your point?

A. I would -- if it were a 5-7, if his AML was 5-7, okay, and I had this information, I would on that specific case drill down much further to make -- to make sure that we had the exposure profiles correct and the work histories understood.

Q. How? What would you do?

A. Speculation. I don't know.

Q. But here's my point: Let's just assume that both sides agree that a particular individual's exposures are less than 200 parts per million of benzene, cumulative exposures, and let's say that you and I agree that the disease can be caused by a sufficient amount of benzene. But where you and I disagree is you say there's got to be at least 200 parts per million exposures, and your only evidence is a hundred parts per million. Are you always going to draw that line and say no way, no how did benzene contribute to causing that man's AML because he didn't have a threshold of 200 parts per million exposure?

A. Certainly at a hundred which is half of what the observed excess risk is, I would probably always say that based on the current state of the science.

Q. So you don't see AML being caused even in part for any individual if the benzene exposures don't exceed a hundred parts per million. True?

A. We're now starting to speculate all over the lot because I said the observed level is 200, and I'm not backing off of the 200.

Q. But you gave 200 -- I mean a hundred a minute ago.

A. Well, I gave you a hundred where you were trying to pin me down where I always say. And now you're trying to make it sound like I have -- I -- you know, I don't speculate because there are so many variables in play. I don't do hypotheticals like that. I just don't.

Q. So if I were to ask you if a person was exposed to 175 part per million years of benzene and they had an AML of the right subtype that you could agree could be a benzene-induced AML, would you say as a general rule that that person's 25 parts per million years short of me ever connecting up as a contributing factor their benzene exposure to their leukemia, or do you have to look at other stuff?

A. I would want to look at other stuff, but clearly the closer we are to 200, the less comfortable we'll be saying it's not.

Q. But you would agree that it may be four or five years before we have the China studies back, assuming that they're properly designed and done properly and all that stuff that you told us previously, to see if there's a threshold for benzene exposure?

MR. SHOEBOTHAM: Objection, form.

A. That's incorrect. We have a lot of science now. It would be nice to have more studies that are well done to help bolster that -- that body of science. That's what science is.

Q. (By Mr. Lubel) What study are you talking about? What studies are you saying establishes that there is no effect for benzene exposures less than 200 parts per million?

A. For AML we have the varying analyses done by Schnatter and Wong on the Pliofilm cohort. They come out of the Pliofilm cohort.

Q. Do we have anything else other than people's interpretation of the data from the Pliofilm cohort?

A. Well, we have some data in UK and we have some data, but they're very small cohorts. One of the recommendations was that you need to be merged together. We need bigger data sets to get more robust to those responses.

Q. Well, how big is the Pliofilm cohort?

A. Pretty small.

Q. Like 1200 people?

A. 1218, depending if you include St. Mary.

Q. We've got bigger cohorts that have been studied on benzene, do we not?

A. Yes. But big does -- big is not intrinsically better. It's got to be better.

Q. It's got to be big and better?

A. Correct.

Q. And although the China study done by Hayes and Yin, the National Institute of Health and the Chinese equivalent, has looked at a bigger set of people, the population exposed to benzene, you don't think that fits the better part of it?

A. Absolutely no.

Q. And although the Australian study by Glass, the case control version, reflects excesses of AMLs at what you would consider -- most people consider low level benzene exposures, that doesn't do it for you either. Correct?

A. That's also a very small study.

Q. What size?

A. The cohort from which those cases are extracted is smaller than Dow's Freeport chemical complex. And that's one single facility of Dow versus the entire country of Australia. So it's not a large study. We have refinery studies that are bigger than the whole petroleum study. But they're -- without extending this further, we -- we have discussed some other limitations, and I went into the papers.

Q. Well, you understand that I asked to take your

deposition in case you show up to trial, I -- if I'm doing a good job for my client, I need to have a pretty good understanding of what you're going to testify to. You understand generally that's what lawyers are hired to do?

A. Yes.

Q. Okay. I'm getting ready to leave here, shut this thing down. You're going to go back to work -- Pennsylvania, where you live, and I'm going to go see my little kid. Do you feel as though we together have communicated about the general substance of your major opinions concerning Mr. Boren's case?

A. Yes, I believe we have.

Q. Can you think of anything that's been missed?

A. Not -- not as I sit here, no.

Q. And the information that you're relying upon for the bases of your opinions as you sit here today, you've brought with you and we've marked as exhibits. Correct?

A. All -- all the key pieces. There are some historical and supporting documents around this, but yes, the gist of it is all there.

MR. LUBEL: Thank you for your time.

THE WITNESS: Thank you.

MR. SHOEBOTHAM: Is there a CV in there? Was there a CV?

MR. LUBEL: Yes.

MR. SHOEBOTHAM: What exhibit number is

that?

THE WITNESS: I don't --

MR. SHOEBOTHAM: It's hard to keep up.

THE REPORTER: Are we on or off?

MR. SHOEBOTHAM: We're on. I have a few questions.

THE WITNESS: I don't know. This is a list of that.

MS. RHYNE: Exhibit 15.

MR. SHOEBOTHAM: 15?

THE WITNESS: 15 would be the other way.

MR. SHOEBOTHAM: There it is.

(Time: 3:43 p.m.)

EXAMINATION

QUESTIONS BY MR. SHOEBOTHAM:

Q. Dr. Raabe, I just have a few follow-up questions for you. And I want to ask you first about Exhibit 15. Would you please identify that for the record?

A. It's my most current curriculum vitae.

Q. Okay. And what is a curriculum vitae?

A. Scientific version of a resume.

Q. Would you please describe your educational background?

A. I have a Bachelor of Arts in biology from Hofstra University and a Master of Science in computational science and

computer science from Pratt Institute, Doctor of Public Health from Columbia University specializing in epidemiology.

Q. Are you an epidemiologist?

A. Yes, sir.

Q. Do you have any affiliation with the American College of Epidemiology?

A. I'm a Fellow of the American College.

Q. And what does it mean to be a Fellow of the American College of Epidemiology?

A. It's the most senior recognition you can get from the college. The Board examines your publications and your contributions to the field of epidemiology and invites you to become a Fellow. It's like a Board certification.

Q. You received a PhD from Columbia University; is that correct?

A. Doctor of Public Health.

Q. When did you -- when did you receive your Doctor of Public Health from Columbia University?

A. '87.

Q. And what specializations did you have when you received your Doctor of Public Health from Columbia University?

A. Epidemiology, statistics, research methods, and a discipline called sociomedical science.

Q. Would you please describe your work in the field of epidemiology?

A. From 1970 through 1977, I was a senior research scientist in the State of New York, attached to Columbia University, conducting epidemiologic research, infant mortality, and disease patterns in New York City.

Beginning the end of October '87 and through a number of positions, I worked for Mobil Corporation in the Corporate Medical Department and various functions related to epidemiology and medical surveillance.

Q. Have you conducted any research concerning the chemical benzene?

A. I've conducted a number of studies relating to petroleum refinery, workers who were exposed to benzene.

Q. Are those studies listed on your curriculum vitae?

A. Yes, sir.

Q. Are they listed under "Publications"?

A. Yes.

Q. Would you please describe for the jury your publications that have involved the chemical benzene?

A. The activities that resulted in publications that have some discussion of benzene or are benzene relevant would -- would include the study of Paulsboro laboratory workers, the cancer epidemiologies of petroleum workers with meta-analysis in 1989, participation in the IARC monograph on cancer risks in petroleum refining, work on a potentially relevant scheme for classifying carcinogens with John Ashby,

working with the work group on the environmental health criteria for benzene with the World Health Organization, review of carcinogenic potential of gasoline, some benzene discussions, cell type specific leukemia in petroleum-exposed workers.

And then there are just a number of other refineries that are listed. And there's another paper on acute myla -- myeloid and mono -- mono -- getting late, I'm sorry -- AML and benzene exposure in petroleum distribution workers in the United Kingdom, paper relating -- with Dr. Bergsagel, B-E-R-G-S-A-G-E-L, regarding benzene and multiple myeloma, paper by Dr. Wong and myself on non-Hodgkins lymphoma in benzene-exposed workers, a paper by myself and Dr. Wong on leukemia on benzene, and one on multiple myeloma on benzene, and one on non-Hodgkins lymphoma on benzene.

Q. All right. Thank you. So you have a number of publications listed on your curriculum vitae that you have published in the scientific literature concerning benzene?

A. In some form or fashion or another, yes.

Q. Let me hand you Exhibit 3 to your deposition.

MR. LUBEL: Oh, I'm sorry, John. I thought you were telling me it was 3.

Q. (By Mr. Shoebotham) Dr. Raabe, Mr. Lubel asked you about Exhibit 3 earlier in the deposition. What is that grouping of scientific articles in Exhibit 3?

A. They're a number of papers that look at the -- the risk factors in epidemiology associated with acute promyelocytic leukemia or AML 3 -- in 3 that Mr. Boren has.

Q. What opinions do you hold concerning the causation of acute promyelocytic leukemia specifically with a 15:17 translocation like Mr. Boren had?

A. All the data I've reviewed specific to both APL and specific to benzene-exposed cytogenetic abnormalities indicate to me that there's no occupational -- no known occupational associations with acute promyelocytic leukemia, and any environmental are occupational exposure.

Q. The paper that's at the top of the stack marked Exhibit 3, when was that published?

A. That's the most recent paper I could find in 2003.

Q. And this paper contains in the abstract a statement -- and let me read it to you: "So far no environmental and/or occupational risk factors have been found for APL." Do you see that sentence?

A. Yes.

Q. Is this one of the papers you rely upon to support your opinion?

A. Yes.

Q. Do the papers that you marked as Exhibit 3 contain any information about the distribution of APL or promyelocytic leukemia between the sexes?

A. Yes.

Q. What do they -- what do they demonstrate?

A. Unlike other forms of AML which are heavily weighted in the male population, the promyelocytic leukemia is equally distributed amongst males and females.

Q. Does that have any significance to your opinions?

A. Yes. It supports the -- the notion described elsewhere in epidemiology that one sometimes considers occupational factors with an exposure having some influence on -- on differentially -- differential rates between men and women.

So the fact that APL is equally distributed at least supports the notion that whatever risk factors there are apply to both men and women equally.

Q. Do these papers provide any information about the distribution of promyelocytic leukemias by age?

A. Yes. It's a somewhat younger form of AML. It's not uncommon to see in the 30 to 50 range of the cases. It does go up with age, but not as more -- there is more cases at the younger ages than I've seen typically in the other forms.

Q. And do these papers that you brought with you and that have been marked as Exhibit 3 provide any information with regard to the latency period that one would see for a promyelocytic leukemia if it's induced by chemotherapy that someone might be taking at a doctor's office or hospital?

A. Yes, they do.
Q. What -- what is the latency period for a promyelocytic leukemia if it's induced by chemotherapy that a doctor might give?
A. They -- the vast majority of them appear within two to three years from -- from treatment.
MR. SHOEBOTHAM: I'll reserve further questions until time of trial. Thank you.
MR. LUBEL: I've got a few more for you.
What -- what number did we end with?
THE REPORTER: 28.
(Time: 3:53 p.m.)
(RAABE Exhibit No. 29 was marked.)

EXAMINATION

QUESTIONS BY MR. LUBEL:
Q. Can you identify Exhibit 29?
A. It's a 2001 paper by Hayes, et al.
Q. Had you seen that before?
A. Yes. I probably would have brought it with me had I thought about it.
Q. Is it authoritative in the field?
A. It's one of many published scientific papers.
Q. I'm not saying it's the only one, but it's one of many?
A. It's not authoritative. It's just a paper.

Q. Why would you have brought it with you?
A. Because I expected you'd want to have some discussion about the Chinese epidemiology. Not because it formed any basis of my opinion.
Q. Well, let me ask you: Do you agree that a scientific consensus concludes that benzene can be etiology approved related to the development of acute nonlymphocytic leukemia based upon epidemiologic studies?
A. Yes, if the exposures are of sufficient duration and in high enough dose.
Q. Do you agree that studies have confirmed that benzene causes leukemia?
A. Only -- I would agree only in benzene has been established to cause acute myelomas.
Q. You're saying there's no studies that have established benzene causes leukemia?
A. There are -- a number of the epi studies only classify leukemia. So if acute myelogenous leukemia is elevated, the whole category is elevated.
Q. Are there studies that relate benzene exposures to leukemia in general?
A. Yes.
Q. Would you agree that the data from the Pliofilm cohort are essentially uninformative at doses below 200 part per million years?

A. No.

Q. Would you agree that -- that Rinsky's most recent article reflects that there's an increase incidence of AML's exposures less than a hundred parts per million?

A. Not statistically significant.

Q. Would you agree that he writes that -- that there is an increase?

A. The SMR is elevated so that is an increase, but it is not statistically significant. So you don't know if it's a real increase or not.

Q. So when you look at the Pliofilm cohort as evidenced by the 95 percent confidence interval on the risk estimates, you wouldn't agree that exposures below 200 part per million years are consistent with increases in a risk for AML as great as six to 14 fold?

A. I would disagree. And I -- I don't know where that statement comes from.

Q. Would you agree that the National Cancer Institute and the Chinese Academy of Preventative Medicine study provides evidence that cumulative benzene exposures of less than 200 part per million years is associated with increased risk for acute nonlymphocytic leukemia such as AML?

A. I don't dispute that the Chinese study makes that statement, but the reliability and interpretability of that statement I think are very questionable.

Q. Do you agree that the study states that significant excesses of acute nonlymphocytic leukemias and MDSs were observed at average exposures of less than 25 part per million?

A. Not statistically significant. There were cases. You don't know if they're related to exposure or not.

Q. And would you agree that the article states that there's evidence of a doubled risk for acute nonlymphocytic leukemia and MDS at average exposure levels under 10 part per million for benzene?

A. I don't recall that statement, so I'd have to go back and spend some time looking at it.

Q. Would you agree that there's evidence that suggests that in -- induction of chromosomal ab -- alterations by benzene is likely to play an important role in leukemogenic -- leukemogenesis or other genotoxic effects?

A. That's what we've been talking about to some extent right here, yes.

Q. So you agree with that?

A. Generally, yes.

Q. Now, the study done in China by the National Cancer Institute from the United States and the Chinese equivalent was not funded by the chemical refinery industry, was it?

A. I don't believe it was.

Q. Would you agree that benzene is a known human carcinogen by all routes of exposure?

A. Subject -- now, I know what you're reading from. I would agree subject to dose and intensity. I mean, it's a -- it's a generic statement of its intrinsic potential.

Q. Would you agree that this statement that benzene is a known human carcinogen by all routes of exposure has been supported by evidence from human epidemiologic studies, animal data, and an improvement and understanding of mechanisms of action?

A. I would generally agree with that.

Q. Has it been clearly established and accepted that exposure to benzene causes acute nonlymphocytic leukemia and a variety of other blood-related disorders in humans?

A. Yes, at varying doses.

Q. Is it true that the findings from the National Cancer Institute's study in China would suggest that workers exposed to benzene at average levels of less than 10 parts per million are subject to a higher risk of hematologic neoplasms?

A. I don't think you can make -- draw any statements or inference from a study that you can't interpret, because you don't know what the exposure profiles are accurately.

Q. So you disagree with that statement -- what the statement suggests?

A. I -- I think that that's what the individual study may -- may state, but I disagree with the -- that suggestion can be validly interpreted at this time.

Q. Would you agree that with respect to the Pliofilm studies that the absence of earlier definitive ambient air measurements for benzene has led to many quantitative risk estimates by numerous investigators over the past several years that have differed from each other partially on the basis of differences and the assumptions made about what those earlier exposures to benzene were?

A. Yes, I agree with that.

Q. Do you agree that the second Rinsky study in 1987 included individual estimates of personal exposure that were not included in his previous study of 1981?

A. I don't recall, but my recollection is everybody has been using the estimates generated post '87. But...

Q. Would you agree that there's been some recent limited epidemiologic evidence that supports certain alternative dose measures as perhaps better than the measurement of cumulative dose?

A. That's the Schnatter paper that I gave you. I'm not sure if I agree that it's -- demonstrates that it's better, but it certainly --

Q. Suggested?

A. -- suggests that the importance of concentration. Concentration is important, which I do agree with.

Q. Would you agree that for AMML, a significantly increased risk tended to be associated with peaked -- peaked

exposures to benzene but not with cumulative exposure?

A. Let's go back to those papers.

Q. Rushton?

A. I don't recall.

Q. This analysis suggests that a person exposed to 40 part per million of benzene for one year might be at a greater risk of leukemia than a person exposed to one part per million for 40 years. What are your thoughts on that?

A. Without sticking to those specific numbers, that's essentially what we're talking about, con -- a threshold concentration as having some importance relative to cumulative dose. So that's not any different from what we've just been talking about.

Q. Were you aware that nine -- there were nine leukemia victims in the Pliofilm cohort that's been studied by these various authors?

A. There is more now in the update.

Q. Pre2002 update?

A. I don't disagree with the statement.

Q. Were you aware that out of those nine, seven had experienced latent periods of 15 years or longer?

A. I don't recall that.

Q. Would that surprise you?

A. It wouldn't surprise me or not surprise me, because you don't know which are the actual benzene-induced cases. So

you -- you can't easily make decisions on -- on -- on latency.

Q. Can a person be exposed to benzene and have a latency period of 20 or more years for which if the exposures were right and the disease was right, you could connect the two or call it a benzene-induced leukemia?

A. I don't think -- let's put it this way: All -- I would find that unusual based on what we know about secondary -- secondary AMLs, either from chemotherapeutic or even from case series where there is strong evidence that they probably are benzene-related AMLs. Those are much shorter periods of time, so I would question that.

Q. So what's the latency period for benzene-induced leukemia?

A. Well, as I just described: In general, you don't know which are which, but the two published estimates that I'm aware for benzene-induced leukemias are both around ten years.

Q. So if Dr. Irons says 15 years, he'd be wrong. Right?

A. No. I would just ask him what the source of that information was.

Q. And how do you calculate it? Is it ten years from the first exposure or ten years from the last exposure?

A. Well, ideally it would be ten years from the most meaningful where the exposure starts to become biologically relevant. So it would be closer to the early years, not to the last year.

Q. Do you agree that the Paustenbach exposure estimates for the Pliofilm cohort were based on worst-case assumptions concerning actual exposure levels?

A. I would not agree or disagree.

Q. Just don't have an opinion?

A. I don't have an opinion.

THE WITNESS: I do need to take a restroom break if you're going to be much --

MR. LUBEL: I'm almost done. Do you want to give 30 seconds? Or you can take a break.

THE WITNESS: I can go a minute or two.

MR. LUBEL: Okay.

Q. (By Mr. Lubel) Do you agree that elevations in exposure to benzene tend to suppress blood counts?

A. Read that again.

Q. Elevations in exposures to benzene tend to suppress blood counts?

A. Yes. As a general proposition that would be true if the doses were high enough.

Q. Did the Schnatter, S-C-H-N-A --

A. Schnatter.

Q. -- T-T-E-R, 1996 article and that analysis indicate that the critical concentration of benzene exposures for risk of acute myelogenous leukemia or AML is in the 20 to 25 part per million range?

A. Now, he presents three different estimates. The lowest estimate is in that range. The highest estimate, I think, is in the 50-plus range. And I think the median is around --

Q. 35 to 40.

A. Okay.

Q. Does that sound right?

A. Yeah, it could be. That's -- that's concentration, not ppm.

Q. Did we -- did I ask you what the exposures were in the Dow Chemical Ott study back in '86?

A. I don't recall, but you didn't ask.

Q. Do you know?

A. I don't recall.

MR. SHOEBOTHAM: You need to take a break?

MR. LUBEL: Go ahead. I think I'm almost --

MR. SHOEBOTHAM: Go ahead and take a break.

(RAABE Exhibit No. 13 was marked.)

(Recess from 4:09 p.m. to 4:12 p.m.)

MR. LUBEL: We're done.

MR. SHOEBOTHAM: We'll reserve further questions until time of trial.

(Signature required.)

CHANGES AND SIGNATURE

PAGE/LINE	CHANGE FROM/CHANGE TO	REASON

I, GERHARD K. RAABE, MD, have read the foregoing deposition and hereby affix my signature that same is true and correct, except as noted above.

_____

GERHARD K. RAABE, MD

THE STATE OF TEXAS)

COUNTY OF HARRIS)

Before me, _____, on this day personally appeared GERHARD K. RAABE, MD, 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 they executed the same for the purposes and consideration therein expressed.

Given under my hand and seal of office this _____ day of _____, 2004.

_____

NOTARY PUBLIC IN AND FOR

THE STATE OF TEXAS

CAUSE NO. 17958-JG01

JAMES BOREN AND GINGER BOREN	)	IN THE DISTRICT COURT OF
VS.	)	BRAZORIA COUNTY, TEXAS
THE DOW CHEMICAL COMPANY, ET AL.	)	239TH JUDICIAL DISTRICT

****

REPORTER'S CERTIFICATION
DEPOSITION OF GERHARD K. RAABE, MD
OCTOBER 6, 2004

I, Valerie B. Corrales, Certified Shorthand Reporter in and for the State of Texas, hereby certify to the following:

That the witness, GERHARD K. RAABE, MD, 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 _____ to the witness or to the attorney for the witness for examination, signature, and return to me by _____;

That the amount of time used by each party at the deposition is as follows:

Mr. Lance Lubel - 4 hours, 20 minutes
Mr. J. Robert Black - 0 hours, 0 minutes
Mr. Jonathan Shoebotham - 0 hours, 10 minutes
Ms. Katherine L. Rhyne - 0 hours, 0 minutes

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:

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I further certify that I am neither counsel for, related to, for 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've occurred.
Certified to by me this the 8th day of October, 2004.

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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 _____, Custodial Attorney;
That $_____ is the deposition officer's charges to the Plaintiffs, 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 and filed with the Clerk.
Certified to by me this _____ day of _____, 2004.

_____
Valerie B. Corrales, CSR
Texas CSR No. 6113
Expiration Date: 12-31-2005
Word for Word Reporting
Firm Registration No. 222
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