IN THE COURT OF COMMON PLEAS
PHILADELPHIA COUNTY, PENNSYLVANIA
CIVIL DIVISION

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EUGENE SALMIERI, et al. : APRIL TERM, 2006
:
vs. :
:
CRC INDUSTRIES, INC., :
et al. : NO. 02439

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SEPTEMBER 9, 2008

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ORAL DEPOSITION OF ROBERT R. MONSON, M.D., taken pursuant to notice, was held at the Hyatt Harborside Hotel, 101 Harborside Drive, Boston, MA 02128, commencing at 9:38 a.m., before Kimberly S. Gordon, a Registered Professional Reporter, Certified Court Reporter and Notary Public.

* * *

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I N D E X

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Testimony of: Robert R. Monson, M.D.

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E X H I B I T S

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(It is hereby stipulated and agreed by and between counsel for the respective parties that sealing, filing and certification are waived and that all objections, except as to the form of the question, be reserved until the time of trial.)

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ROBERT R. MONSON, M.D., after having been first duly sworn, was examined and testified as follows:

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EXAMINATION

- - -

BY MR. DuPONT:

Q. Good morning, Dr. Monson. Did you bring a copy of your file with you here today?

A. Yes.

Q. I see there's a box over on the side of the room. Is that it or --

A. No, it's here.

Q. Is there any organization to your file here what you've just handed me?

A. The beginning stuff is things, plaintiff witnesses and a couple of odds-and-ends of papers and some e-mails between Mr. Woolf and me. And then there's my report in this case, the papers that upon which the report relies. And then there's a later report, I'm sorry, another report on NHL which I added because Mr. Woolf asked me to because of the recent deposition of one of your witnesses.

Q. Can you identify for me what the recent report on NHL is that you're referring to?

A. It's at the very bottom. It was not done for this case.

Q. And that clipped document, the first page is entitled Evaluation of Paper by Steinmaus C et al. Meta-analysis of Benzene Exposure and Non-Hodgkin Lymphoma, --

A. Yes.

Q. -- colon, Biases Could Mask an Important Association. Can you tell me what this document is?

A. It's a review of the paper that was written by Steinmaus, et al., as well as the paper by Martin Smith that preceded it.

Q. And who performed the review of the paper?

A. I did.

Q. When did you perform this review of the Steinmaus and Smith papers?

A. The Smith one I did probably a year ago whenever it came -- it came out in 2007. The Steinmaus one I did earlier this year.

Q. Why was it that you performed a review of the Steinmaus paper earlier this year?

A. Mr. Woolf asked me to.

Q. So your review of the Steinmaus paper, was that in conjunction with the benzene litigation?

A. It was in junction with the subject NHL and benzene. It wasn't related to any case or anything.

MR. DuPONT: Let's go ahead and mark that Evaluation of Paper by Steinmaus and mark that as Exhibit Monson-A.

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(Whereupon the document was marked, for identification purposes, as Monson-A.)

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THE WITNESS: The usual question that comes up, are you going to keep these exhibits and what do I get back? How does that work?

BY MR. DuPONT:

Q. The exhibits will go with the court reporter. The court reporter will make copies. The originals are maintained by our office.

A. The papers as well? Scientific papers?

Q. We can discuss that.

A. I just want to get it back. I don't care how it's done but I don't want to have to go to the library and get all the papers again.

Q. I understand. Can you explain to me what it is you did specifically with respect to the Steinmaus paper, how you evaluated it, what your methodology was?

A. I read it. I did the Smith one first, and I read it and evaluated it and looked at the original upon which I relied. And I did a

similar thing with the Steinmaus paper and did some similar corrections to the data or re-analysis of the data that Steinmaus did and then I summarized what I did and compared it to what Steinmaus did.

Q. I am going to hand Exhibit Monson-A back to you. Were you asked to perform this review of the Steinmaus and the Smith papers on behalf of any defendant to the benzene litigation?

A. No.

Q. Any petroleum companies?

A. No.

Q. What were your conclusions with respect to the Steinmaus paper?

A. My conclusion states, quote, the weight of the evidence from a review of the epidemiologic literature comprising over 40 published papers shows that there's no basis for suggesting that benzene is associated with the occurrence of non-Hodgkin lymphoma. I call it non-Hodgkin without the S.

Q. So you went through and looked at the same studies that the Steinmaus article

looked at?

A. At the what study?

Q. That the Steinmaus article looked at.

A. Did you say same?

Q. Is that correct?

A. Yes.

Q. And you provided a separate analysis than Steinmaus, et al. did?

A. Yes. He did an extensive statistical analysis. I didn't repeat that. But in terms of summarizing the data, looking at the papers, taking numbers from the papers that relate any association between benzene and NHL.

Q. Did you analyze any of the data in the studies cited by the Steinmaus paper for Healthy Worker Effect or any other influence that the methodologies in those studies could have had on their findings of risk?

A. Yes.

Q. And what did you do?

A. The first issue was which data Steinmaus abstracted from the papers, and I looked at that and sometimes I agreed with what

he did and other times I disagreed. He adjusted relative risks based on all cause MR as well as all cancer SMR, and I did the same thing for all cancer SMR, if that's clear. I did the same adjustment that he did or methodologically the same. And then I compared his paper to Smith's paper in terms of overlap or non-overlap as well as papers I had looked at that either of them may not have.

Q. Now, you come to a different conclusion on the causal relationship between NHL and benzene exposure based on the review of the same literature than does Steinmaus?

A. I couldn't state what Steinmaus' conclusion was with respect to causality.

Q. You come to a different conclusion with respect to the risk of contracting NHL from benzene exposure than does Steinmaus?

A. I'd have to look at Steinmaus' conclusions to make sure I can answer that correctly.

Q. Why don't you take a look at that?

A. I don't have his paper here. It would be somewhere here.

I may not have Steinmaus' paper with me. It may just be an oversight. I'm sure one of you has it. I don't see it, I'm sorry. Do you have it?

Q. We'll get to that in a moment.

A. Okay. I just added this report at the last minute, and I obviously just took my report, my evaluation.

Q. Is it your understanding that CLL is a form of non-Hodgkin lymphoma?

A. In recent years, that's the way the classification has moved, yes.

Q. Do you have with you a list of your previous testimonies, cases in which you've given depositions or --

A. No.

Q. Do you have that information at your office?

A. Yes.

Q. Doctor, because we have a court reporter here today, I notice that you are predicting what my question is going to be and at

some points beginning the answer before I finish my response. It makes her job a little difficult if we're speaking over each other. So, if you could allow me to finish my question before you begin your response, I would appreciate that. In turn, I will try to allow you to finish your response before beginning my next question. Is that okay?

A. Yes.

Q. If at any point in time I ask you a question that you do not understand, would you please let me know and I'll do my best to rephrase or re-ask the question?

A. Yes.

Q. Doctor, who have you been hired to testify on behalf of in this case?

A. I've been hired by Mr. Woolf. And frankly, until today, I wasn't sure on whose behalf he was, he and Mr. Scott are representing. I asked them this morning and I've already forgotten, so Mr. Woolf can tell you what it is.

Q. Are you testifying on behalf of Sunoco in this case?

MR. WOOLF: Do you want me to

answer for him?

THE WITNESS: I don't know.

MR. WOOLF: If you really don't know who I represent, I'll be more than glad to restate it for the record. But the witness is here on behalf of Exxon, Sunoco and I believe Chevron.

THE WITNESS: This hasn't been discussed.

BY MR. DuPONT:

Q. How many times have you given a deposition in a case where it's been alleged that somebody sustained a disease or an injury from exposure to benzene?

A. I would estimate 15 to 20.

Q. And on those occasions, have you ever testified on behalf of an individual who had alleged that they contracted an illness?

A. No.

Q. Were all your depositions given on behalf of companies that either manufactured or sold products that contained benzene?

A. When you say all, do you mean all benzene or do you mean --

Q. Sure. Of those 15 to 20 depositions where you've testified in benzene litigation, we'll talk about those cases, --

A. Yes.

Q. -- were you always testifying on behalf of defendants to the benzene litigation?

A. You asked me that question before, didn't you?

Q. I'm asking it a different way. I want to make sure I understand.

A. I don't understand the difference.

Q. My question is: In all of your 15 to 20 cases in which you've given depositions, they've been on behalf of companies that made or sold benzene-containing products?

A. I would say products alleged to contain benzene.

Q. Are you able to recall the names of any of those 15 to 20 cases, the names of the plaintiffs?

A. No, I don't remember that.

Q. Do you have a list at your office or somewhere compiled all those cases that you've

given testimony in?

A. Yes.

Q. You did not bring that with you today?

A. No.

Q. Could you provide that to Mr. Woolf so he can give it to us, please?

A. Yes.

Q. Thank you. Dr. Monson, what is your rate for testifying here today?

A. 8,000 a day.

Q. And what is your rate for, either on an hourly basis or however you work it, for preparing your report in this case and reviewing the file?

A. 600 an hour. The daily rate is prorated, of course. It's not a flat rate.

Q. Have you testified in litigation other than allegations of benzene exposure?

A. Yes.

Q. What other areas of litigation have you testified in?

A. Are you talking now about trial testimony or depositions?

Q. Let me be a little broader. Have you been hired by any companies to testify or prepare reports on their behalf in other areas of litigation besides benzene?

A. Yes.

Q. Which other areas of litigation?

A. I hesitate only because if you say prepare a report and there's no deposition I consider that to be confidential. But if you're talking depositions or trials, then I can just go ahead and tell you what I remember.

Q. Tell me what you remember.

A. I remember welding; Bendectin, which is a drug taken by women during pregnancy; various radiation cases. There's been several environmental cases; several have been for the plaintiff, on behalf of the plaintiff. There's undoubtedly others but that's what I remember.

Q. Have you ever testified on behalf of a plaintiff in a welding case?

A. No.

Q. The second area was Bendectin that you identified. Did you ever testify on behalf of a plaintiff in Bendectin litigation?

A. No.
Q. How about radiation, have you ever testified on behalf of a plaintiff in a radiation exposure case?
A. Yes.
Q. Did you testify on behalf of defendants as well?
A. In other cases, yes.
Q. The fourth area was environmental litigation. Can you explain for me what you mean by environmental?
A. Yes, one was I testified on behalf of the plaintiff for a sick building issue in a courthouse in Chicago where the ventilation system was improperly installed and the air was bad. There was a situation in Alabama or Georgia where creosotes soaked into the ground and was found to be in a ditch surrounding a church. I testified for the defense in terms of the allegation was multiple diseases were caused by this tarring-like substance.
Q. Any others that you can remember?
A. Not right now, no. There may be others but I don't remember them.

Q. When did you first begin to testify in litigation?

A. The first one was in fact another one it was 1989 thereabouts -- or was it 1979? Sorry, I got to think about my timing here. Anyway, it was emergency temporary ban of 245 T, which is a pesticide made by Dow, and Dow was suing the government. They had the emergency temporary ban overturned, and I testified on behalf of EPA saying that I agreed that there was epidemiologic evidence that warranted concern. I'm not sure about whether it was '79 or '89, but let's say it was 1980.

Q. And when did you first begin testifying in benzene exposure cases?

A. Maybe 15 years ago, 15 or 20 years ago. I can't be precise.

Q. How much income do you derive in a year, start with 2007, the last full year, from testifying in litigation?

A. That would include preparing reports?

Q. Yes.

A. Not testifying?

Q. Correct.
A. It varies from year to year. I would say in the last decade the lowest was 30,000, the highest was 150,000. The mean would probably be around 80,000.
Q. And do I understand you've testified on behalf of Mr. Woolf's firm before?
A. Yes.
Q. Approximately how many times?
A. Again, depositions and trials, there's been two trials, perhaps 10 or 15 depositions.
Q. And have all of the trials and depositions that you testified in on behalf of Mr. Woolf's firm, have all of those involved benzene exposure, allegations of benzene exposure?
A. To the best of my recollection, yes.
Q. And would I be correct in that all those cases you never came to the conclusion that the plaintiff had contracted an illness as a result of exposure to benzene?
A. My testimony related to the

epidemiologic literature rather than to the specific individual.

Q. And in any of those cases on which you've testified on behalf of Mr. Woolf's firm, did you ever come to the conclusion that the epidemiological literature supported a causal connection between exposure to benzene and the injury that the plaintiff alleged they sustained as a result of benzene exposure?

A. No.

Q. Can you tell me how much you have charged for your work in this case, the Salmieri case to date?

A. I received one payment I think around 6,000. And a second bill is in the mail, again, for about 6,000.

Q. And what did the second bill include, up until what date, what portion of your work?

A. Probably through July 31st.

Q. Have any of your opinions ever been excluded or have you ever been prevented from testifying in a case?

A. No.

Q. Let me take another look at your file here. On the top here, you have an article or actually a printout from the Internet called "What is Non-Hodgkin Lymphoma?". Can you tell me what that document is and why you have it in your file?

A. The main reason I looked it up was to determine what percent of NHL is either CLL or small lymphocytic leukemia, and the statement in this document is these related diseases account for about one out of four lymphomas.

Q. One out of four lymphomas or one out of four non-Hodgkin lymphomas?

A. It says lymphomas.

Q. Is it your understanding that CLL and SLL are forms of B cell non-Hodgkin lymphoma?

A. Yes.

Q. Have you ever sought to determine what percentage of non-Hodgkin lymphomas CLLs and SLLs make up?

A. That's what I think the one-in-four refers to. My guess is that this one out of four lymphomas really means one out of

four NHLs. I think the ACS probably didn't write this as clear as they might have.

Q. When you say ACS, you're referring to the American Cancer Society?

A. Yes.

Q. Have you looked beyond the document that you have in front of you from the American Cancer Society?

A. I had it and I was trying to find it, but it may have been in one of these papers that I reviewed.

MR. DuPONT: Let me mark that as Monson-B, please.

- - -

(Whereupon the document was marked, for identification purposes, as Monson-B.)

- - -

BY MR. DuPONT:

Q. Then the next thing you have in your file are some communications with Mr. Woolf concerning what, this case?

A. Right. This is really a cover sheet for what files, just an e-mail saying, not an e-mail but a letter that says enclosed are --

Q. I see. We'll put that aside.
You're referring to the May 29, 2008 e-mail from Ms. JoAnn --
A. It's a bit out of order. This goes first. These are two e-mails from, I'm sorry, one e-mail from Lou and one memorandum from me about this case.
Q. I'm going to mark your memorandum as Monson-C.

- - -

(Whereupon the document was marked, for identification purposes, as Monson-C.)

- - -

BY MR. DuPONT:
Q. In the last paragraph of your memorandum, and this is dated November 21, 2007 from yourself to Lou Woolf, the last paragraph you refer to a report of September 10, I believe. Do you see that?
A. Yes.
Q. What report is that that you're referring to?
A. I don't remember. It was a report unrelated to this case that I had done,

and I had all the papers in a pile somewhere. It probably had to do with AML but I can't be sure. But I just looked through those papers as sort of preliminary, to get some preliminary impression of what information was available on CLL.

Q. The report would have been from September 10, 2007?

A. Yes.

Q. And was that a report that you prepared in the context of benzene litigation?

A. Yes.

Q. Do you have a copy of that report in your office?

A. Yes.

Q. Is that a report that you can give to Mr. Woolf so that he can provide it to us, please?

A. Again, I think it depends on whether it was done for a specific case which was not subject to deposition. Therefore, I believe it was confidential.

Q. I'm sure your counsel in this case can sort that out for you. But was this a report that was served on a party to --

A. I don't remember what report it was. I looked at it last night and realized I didn't know which report I was referring to.

Q. In any of your other work on behalf of Mr. Woolf's firm and benzene cases, have you analyzed the literature as it relates to CLL?

A. No.

Q. In any of your other work on behalf of Mr. Woolf's firm, have you analyzed the literature as it relates to benzene and NHL?

A. Yes.

Q. How many other cases?

A. I gave you an answer of 10 to 15. That would be the number.

Q. I'm sorry, perhaps I wasn't clear. In how many other occasions on behalf of Mr. Woolf's have you analyzed the literature on non-Hodgkin lymphoma and benzene?

A. Two or three. I mean, these were done not with respect to any litigation. They were done by request.

Q. And did you formulate reports in those cases involving non-Hodgkin lymphoma?

A. Yes.
Q. And do you recall what your conclusions were in any of those reports?
A. You can look at my report here and that would be my conclusions. Basically, there's no evidence, there's no consistent evidence for an association between exposure to benzene or benzene-containing products and NHL.
Q. When you say I can look at your report here, are you referring to the report that you issued in this case, the Salmieri case?
A. No, the Steinmaus and the Smith thing that I gave you before which is unrelated to this case.
Q. Have you testified on behalf of any other law firms representing defendants in benzene litigation besides Mr. Woolf's firm?
A. Yes.
Q. How many others?
A. I can recall two at the moment.
Q. And who are they?
A. One was a paint case and I'm not sure who the defendant was. But the lawyer escapes me; he was from Philadelphia. So,

anyway, the allegation was that there was benzene in paint. And I don't recall the illness.

And the other attorney was in Chicago and it was an AML case. And the product I don't recall because there were multiple defendants and it wasn't clear, with multiple products, some were distributors, some were manufacturers, some were users. It was very confusing legally.

Q. In any of your prior work for companies that are alleged to have sold or manufactured benzene-containing products, did you look at gasoline literature specifically?

A. Yes.

Q. How many other cases?

A. One or two is what I recall.

Q. Do you recall the names of those cases?

A. No.

Q. Did you issue reports in those cases?

A. Yes.

Q. And did you testify?

A. There were depositions, I

believe. Usually, there's a deposition somewhere along the line.

Q. Do you remember when those depositions were given?

A. No. It would be in my list of depositions, within the last ten years.

Q. We'll mark as the next exhibit, Monson-D, the January 14, 2008 e-mail from Mr. Woolf to yourself.

- - -

(Whereupon the document was marked, for identification purposes, as Monson-D.)

- - -

BY MR. DuPONT:

Q. In the PS line of this e-mail, Mr. Woolf asks you and it says: I would appreciate it if you would do it for CLL specifically but also with regard to the small cell NHL issue.

And he's referring to his request that you do a detailed report with regard to the relationship between gasoline on the one hand and CLL and benzene and CLL. Take a look at that.

Did you in fact do a review of

the literature and issue a report as it relates to small cell NHL?

A. I looked to see if I could find anything and didn't.

Q. When you say small cell NHL, is that small cell lymphocytic leukemia?

A. It's what it's called. You called it SLL. That's small lymphocytic lymphoma. Is that what SLL stands for?

Q. What's your understanding of what you were asked to do with respect to small cell NHL?

A. Small cell lymphoma, yes. It's called small cell -- no, it's called small lymphocytic, I'm sorry. The terminology is very confusing and this has just come along in the last few years. I believe it's called small lymphoma. Is that the correct term?

Q. Is that your understanding of it?

A. It's the same disease except for location as CLL. There's CLL which is classically a disease of chronic lymphatic leukemia. And then there's this type of NHL which is called small lymphocytic lymphoma.

That's what it is.

Q. Those are both types of B cell non-Hodgkin lymphomas?

A. They're two different forms of the same disease is my understanding. I'm not a hematologist. I wouldn't want to. I'm not an expert in that area.

Q. And you would agree with me that both CLL and SLL fall within the rubric of B cell non-Hodgkin lymphoma?

A. To the best of my recollection. I'm not an expert. I believe I just read it there in the American Cancer Society document.

Q. Have you spoken with anyone from ExxonMobil or Sunoco or Texaco regarding this case?

A. No.

Q. Have you ever spoken with Dr. Schnatter?

A. I may have met him many years ago. I don't recall.

Q. Did you review any depositions given by Dr. Schnatter?

A. No.

Q. There's a paper in here by Aaron Blair, and that's part of your file. Can you tell us why it is you reviewed that paper?

A. The title is Chemical Exposures and Risk of Chronic Lymphocytic Leukaemia, spelled the British way.

Q. And when in your analysis in this case did you review that? Did you review that as part of preparing your report?

A. I was trying to think -- I didn't include it in my report and that was either because when I first wrote the report it hadn't been published -- it was published on-line in October 24, 2007, and I'm not sure exactly when I came across it. But on the other hand, it's a review. It's not a specific study. But it has to do with chemicals and CLL.

MR. DuPONT: I'm going to go ahead and mark that as E.

- - -

(Whereupon the document was marked, for identification purposes, as Monson-E.)

- - -

BY MR. DuPONT:

Q. The next section of your file I'm going to hand you appears to be your report and the studies that you relied upon?

A. Yes.

Q. Are those studies that you've cited to in your report all the studies you intend to rely upon in support of your opinions in this case if you were to testify at trial?

A. No.

Q. Can you tell me what other articles you rely upon in support of your opinions?

A. If new articles come along, I would include them. And if there are articles I've missed that are pointed out to me, I may or may not consider them.

Q. Up until this point in time, can you tell me all the articles you intend to rely upon in support of your opinions?

A. Yes.

Q. And which are those?

A. I'm sorry?

Q. Which are those? Which articles do you intend to rely upon that have been

published up until this point in time?

A. The ones here. As I say, there may be others that I missed, and if I find out about them, I may add them. I have no plans to do that, but you never know.

Q. I want to get to your methodology here in a little bit. But I'd like to know --

MR. DuPONT: First of all, Exhibit-F, we'll make that his report along with the studies that he's relied upon.

- - -

(Whereupon the document was marked, for identification purposes, as Monson-F.)

- - -

BY MR. DuPONT:

Q. How did you perform your literature review in order to come to your opinion in this case?

A. I usually do a PubMed search of either the disease or the exposure or both. And then I read the bibliography of the papers to see if there are other papers that have not turned up in PubMed.

Q. You say that that's how you usually perform your search?

A. Yes.

Q. I want to know how you did that specifically as part of your methodology in this case.

A. That's what I did. I mean, also, my initial review was of papers I had for other cases on AML which I had obtained through PubMed.

Q. And how did you come to the decision on which papers to include or which papers to rely upon in support of your opinions?

A. Basically, I looked at studies and saw if they had any information on CLL and benzene or benzene-containing products.

Q. And did you then include all papers that had information on CLL and exposure to benzene or benzene-containing products within your report?

A. There are some papers that were not studies but were reviews or opinion pieces I did not rely upon.

Q. Did you include all papers that you felt were studies as opposed to reviews or

opinion pieces in your report?

A. Some papers are updates of earlier papers and I include only the latest paper on a given group of people.

Q. Other than situations where you have an update of a previous study and you exclude the prior study, have you included all studies that relate to CLL and exposure to benzene or benzene-containing product?

A. Yes, all the ones that I've identified.

Q. Am I correct that you did not review the literature and report on that as it relates to non-Hodgkin's lymphoma in your report?

A. That's correct.

Q. And why is that?

A. I wasn't asked to. Other than the small lymphocytic request that we've already discussed, I found no papers.

Q. Are you familiar with Dr. Schnatter's 1996 article concerning benzene exposures in the Canadian petroleum distribution workers?

A. I don't see that I relied upon

it. I know that Schnatter has written a number of papers and I know that he's written papers with other people, so I don't have -- I don't see that I relied upon it. But if you show it to me, I may be able to say yes, I've seen that and go from there.

MR. DuPONT: Why don't we take a five-minute break here?

- - -

(Whereupon there was a recess in the proceedings from 10:31 to 10:39.)

- - -

BY MR. DuPONT:

Q. So the first part of your method in this case is that you go through, you perform a literature search using MedLine?

A. PubMed.

Q. And then you select those cases that you believe deal with CLL and exposure to benzene or products containing benzene, correct?

A. Yes.

Q. And then once you have that group of documents you divided them up into studies which dealt with exposure to, likely exposure to

gasoline and CLL and exposure to benzene and CLL?

A. Yes.

Q. How is it that you came to the conclusion whether or not a study dealt with persons likely exposed to gasoline?

A. Either it was about either "gasoline" was in the title or the word "gasoline" was used. Petroleum marketing and distribution, it's my understanding that at least some of those people are exposed to gasoline. Then there are people who work in service stations, people who work as vehicle mechanics. That's about it.

Q. So you looked either at the title of the study or the body of the study to see if there was mention of gasoline or a job category such as petroleum distribution, vehicle mechanic, gas station attendant that would indicate to you that there was exposure to gasoline?

A. Possible exposure.

Q. How come you did not include the 2003 Glass article --

A. That's under benzene.

Q. Let me finish. How come you did

not include the 2003 Glass article among the Studies of Persons With Likely Exposure to Gasoline?

A. Because it's a study of benzene.

Q. Do you have an understanding as to whether or not the workers studied by Glass in the 2003 article were exposed to gasoline?

A. Again, all I say in my, on Page 7 is that they worked in the petroleum industry and they were -- information on benzene was obtained.

Q. Do you know whether or not the workers that were involved in the Glass study of 2003 were exposed to gasoline?

A. I don't know.

Q. If they were exposed to gasoline, would you then include them within your listing under Roman Numeral II of Studies of Persons With Likely Exposure to Gasoline?

A. I'd have to look at the paper and decide exactly where it should go. I only include it in the paper once. And benzene is a more specific chemical than gasoline is and I tend to group specific chemicals together separate from mixtures of chemicals.

Q. So, if there's a study that deals with both gasoline exposure and benzene exposure, you put that study in the benzene exposure column as opposed to the gasoline column?

A. Yes.

Q. Why is that?

A. As I say, I think it's something that's specific that bonds together. It's a chemical, whereas gasoline is a product which is a mixture of a number of chemicals.

Q. Which include benzene, correct?

A. Which may include benzene.

Q. Do you know if gasoline is made without benzene?

A. No, I don't know that. I'm just saying that. You were asking a general question and I was giving you a general answer.

Q. So, if Dr. Glass had published that subjects of her nested case-control study had exposure to gasoline, you would not include that within your list of Studies of Persons With Likely Exposure to Gasoline?

A. If I had not done benzene as well, then I would have included it. But since I

did both benzene and gasoline, I included it under benzene.

Q. I am going to hand you Dr. Schnatter's 1993 article entitled Retrospective Mortality Study Among Canadian Petroleum Marketing and Distribution Workers. I'll ask you is that a study that you reviewed in the course of coming to your opinions in this case?

A. I'm certainly aware of the study and I assume I reviewed it. I can't tell you I did for sure. Do you want to point out anything specific?

Q. You have no recollection of reviewing this study in the context of preparing your report in this case?

A. I would say that that's correct. It's likely I did review it but I don't recall specifically excluding it or reviewing it. I certainly read it in other contexts.

Q. And Dr. Schnatter in that study finds a significantly elevated mortality due to leukemia among tank truck drivers and the SMR is 3.35. Do you see that?

A. Yes.

Q. Would you include that that study, specifically a finding of SMR of 3.35, supports a conclusion that gasoline distribution workers are at an elevated risk for contracting leukemia?

A. Yes, leukemia as a general category, not CLL.

Q. A finding of elevated risk for leukemia generally among gasoline distribution workers, is that relevant to CLL?

A. No.

Q. Why not?

A. If there's no information on CLL, it's not relevant. From a cursory look here, I don't see any information on CLL.

Q. What about lymphocytic leukemias, if there's a finding of elevated risk for lymphocytic leukemias in general, would that be relevant to whether there's a risk for contracting CLL from exposure to gasoline or benzene?

A. It may or may not be because it could refer to acute. It could refer to chronic. I believe I included one or two papers that did

just have lymphocytic.

Q. How about the 2002 Guenel study, if you turn to Page 7 of your report?

A. Okay.

Q. Why did you place that underneath the Roman Numeral III, Studies of Persons Exposed to Benzene, as opposed to Studies of Persons With Likely Exposure to Gasoline?

A. It says gas. It doesn't say gasoline.

Q. So what do you understand gas to be that they're referring to?

A. Natural gas.

Q. Do you have that report with you?

A. Yes.

Q. If you look at Page 89 under Benzene Exposure Assessment?

A. Yes.

Q. If you look at the first sentence under Benzene Exposure Assessment, it says:

"Use of solvents" for cleaning or degreasing materials and "exposure to gasoline" from motor vehicles were the two work tasks that may have entailed exposure to benzene. Do you

see that?

A. Yes.

Q. Do you agree with me that the gas that's being discussed in Guenel is gasoline and not natural gas?

A. No.

Q. No?

A. If you look further in the article, examples of occupations with such exposures include plumbers of the gas distribution network. And that's not clear.

Q. He also discusses mechanics, correct?

A. In thermic or hydraulic electricity production plants. And then Group 3 up above is coal gasification which leads me to believe that the gas is gas produced from coal, which is essentially natural gas.

Q. What about the second exposed work task?

A. I'm sorry, where are you looking?

Q. If you look at the very bottom of Page 89 on the right-hand column, they discuss benzene exposure may also have occurred from

"exposure to gasoline" identified as the second exposed work task. Motor vehicle mechanics working in garages represented the main occupation with such an exposure. Do you see that?

A. No, I'm sorry. Just say again where it is.

Q. Sure, the very bottom of Page 89 on the right-hand column.

A. Yes. Okay, fine. So they may have been exposed to gasoline. But I believe the term "gas" refers to natural gas.

Q. And if you continue further down, it also says: In addition, gasoline may have been used as a solvent in garages, even though this practice is proscribed.

A. Yes.

Q. Did you see anywhere in this case that Mr. Salmieri used gasoline as a solvent?

A. I know nothing about the case.

Q. Would you now agree with me that the subjects of the study Guenel 2002 had exposure to gasoline?

A. Yes.

Q. Based on that, would you include the Guenel study within the list of cases under Studies of Persons With Likely Exposure to Gasoline?

A. Not in the article -- not the report as I wrote it because benzene is the primary exposure. Had I done a study only of gasoline workers, then I would have included that.

Q. But the benzene exposure comes from gasoline, correct?

The authors aren't concluding that the individuals are having exposure to pure benzene in this case, are they?

A. It says use of solvents for cleaning or degreasing materials that may have entailed exposure to benzene.

Q. Correct. Use of solvents and use of gasoline, correct?

A. So use of solvents containing benzene was widely used, so it's hard to say whether there was more exposure from solvents or from gasoline.

Q. Do you know what the authors

concluded regarding the benzene content of the solvents versus the gasoline being used?

A. On Page 89, they talk about the standard which was less than 1 percent in 1969 and less than 2 percent weight in 1986.

Q. That's actually .2 percent weight, correct?

A. Yes.

Q. That's with respect to solvents, right?

A. Yes.

Q. And then the standard referred to with respect to gasoline is 5 percent?

A. Yes.

Q. And then the author gives a ratio for exposure to gasoline versus use of solvents on Page 90. Do you see that?

A. Yes, I'm trying to figure that out.

Q. Am I correct in that the authors assign a ratio of two-to-one for exposure to gasoline versus use of solvent pre-1970 and then a ratio of ten or .4 to .04 for gasoline versus use of solvent after 1985?

A. This is an index of benzene exposure intensity.

Q. Correct.

A. It doesn't refer to the ratio of use of solvents to the ratio of use of gasoline.

Q. Now, you've published some studies with respect to the incidence of leukemia amongst rubber workers?

A. Yes.

Q. Am I correct in some of those studies you found where there was an increased risk of leukemia amongst rubber workers that some of those workers used gasoline as a solvent in the context of the work?

A. They used white gas, which is I guess a form of gasoline.

Q. Actually, you characterize it as high test gasoline?

A. If you say so. You'd have to point that out to me.

Q. I'm going to hand you your article Cancer Mortality and Morbidity Among Rubber Workers and point your attention to Page 1035.

MR. WOOLF: What's the date?

MR. DuPONT: It's 1978.

BY MR. DuPONT:

Q. I'll apologize, I'm going to look over your shoulder because I don't have another copy with me. But you write here in 1978 that the most common solvents used in this company were fine petroleum solvents, principally high test gasoline and varnish makers naphtha. Is that correct?

A. Yes.

Q. You did not include that study within your review of literature in your report in this case, did you?

A. No.

Q. And that study shows an elevated risk of leukemia among those workers who used gasoline as a solvent?

A. Yes. There's no information on CLL.

Q. There's 19 cases lymphatic leukemia?

A. There's 21 mortality deaths and 27 cases.

Q. Did that study in 1978 find 19 workers who were diagnosed with lymphatic leukemia?

A. No, found 27.

Q. 27 with lymphatic leukemia?

A. Yes.

Q. And then it also found workers with lymphosarcoma?

A. Yes.

Q. Do you know where the information is today on which those diagnoses were based on? In other words, are there still records that can tell us what forms of lymphatic leukemia those 27 workers had?

A. No, I don't know.

Q. You state in your paper that you hesitate to attribute the excess of leukemia solely to benzene. Do you see that?

A. Yes.

Q. If you can turn to Page 7 of your report, please?

A. Go ahead.

Q. At the bottom, you have your discussion of the Glass 2003 study?

A. Right.

MR. WOOLF: While you're doing that, let me see Monson '78.

MR. DuPONT: Sure.

BY MR. DuPONT:

Q. Now, your principal comment with respect to the Glass study is that it has a potential flaw and that the cases were identified by self-report rather as part of the standard system of follow-up. Is that correct?

A. Yes.

Q. And am I correct in that your concern with that is that cases of CLL may have been omitted because they were not reported?

A. No. It's more that cases of CLL may have self-identified if they were concerned about benzene exposure.

Q. Have you reviewed Dr. Glass' response to critiques of the 2003 article and the use of self-reporting?

A. I believe so. There's two or three subsequent articles dealing with the exposure assessment, and the self-reporting, I don't have it in my mind.

Q. Are you familiar with the Institute of Occupational Medicine?

A. Can you say more about it?

Q. Sure. In July of 2005, Institute of Occupational Medicine authored a study entitled A Review of the Data Quality and Comparability of Case-Control Studies With Low Level Exposure to Benzene in the Petroleum Industry. The principal author was B.G. Miller.

A. Can I see it?

Q. Sure.

A. No, I haven't seen this.

Q. Do you agree with Dr. Glass' response to the criticism and the use of self-reporting that essentially it does not affect the validity of the study because there's an over 90 percent participation rate of the members of the cohort?

A. Can I see that, please?

Q. Sure.

A. I don't see the 90 percent. I suspect it's in here somewhere. Was it on this page?

Q. Were you aware that there was an

over 90 percent participation rate?

A. I know I've read that paper. That's all I can say.

Q. So you're not aware that there was an over 90 percent participation rate?

A. If I had read the paper, I undoubtedly read that statement. I don't recall that statement.

Q. If there was an over 90 percent participation rate in that cohort examined by Dr. Glass in 2003, would you agree with me that that would eliminate any concern with respect to any selection bias or reporting bias?

A. It wouldn't eliminate anything because that's an overall percentage. It's not specific for people with CLL or for benzene exposure.

Q. If you had no information to suggest that there was a different percentage of reporting rate or inclusion rate for CLLs --

A. It doesn't eliminate it. It's better to have a 90 percent inclusion rate than a 50 percent inclusion rate.

Q. Would you agree with me that it

would eliminate any cause for concern with respect to the reporting bias?

A. It wouldn't eliminate it. It would minimize it.

Q. If Dr. Schnatter wrote that there were no obvious design flaws in the 2003 Schnatter study, would you agree with him?

A. I'm sorry?

Q. If Dr. Schnatter concluded that there were no obvious design flaws in Deborah Glass' 2003 study, would you agree with his conclusion?

A. I wouldn't disagree with it. I haven't read that. The main concern about the health watch study in general is comparing Glass to Gun, overall excess of CLL in the entire cohort. And when you do a case-control evaluation of benzene which goes beyond the basic data in the follow-up study, you find this excess. So it's an ambiguous study.

Q. The Glass 2003 is a nested case-control?

A. Within the Gun cohort.

Q. Correct.

A. In addition, she identifies other cases that are not included in it.

Q. So, with Gun and Glass, in certain respects, you're looking at different cohort members?

A. The Gun study is the cohort. The Glass study includes Gun plus other people. And it's not clear to me that they belong to different cohorts.

Is Gun a mortality or -- Gun has cancer incidence as well so it's -- there's ambiguity into how those studies were done because they were done by different people at different institutions.

Q. So you can't necessarily relate the findings of one to the other, can you?

A. They should be consistent with each other and they're not, and that raises a general concern of what's going on and I can't answer that question.

Q. But the Gun study doesn't invalidate the findings of Glass' 2003 study, does it?

A. It's not consistent with it.

Q. But it does not invalidate the findings, does it?

A. Not by itself. But one has to present both sets of findings in attempting to evaluate that study in general.

Q. You mentioned that you used a weight of the evidence approach in your report. Can you describe that for me what your weight of the evidence --

A. Can you point out where I say that?

Q. Sure. Do you in fact use a weight of the evidence approach?

A. That term is a bit loaded and I -- okay. It's on Page 2. The term "weight of the evidence" is used in describing or in discussing the Bradford-Hill criteria.

Q. So do you use a weight of the evidence approach in coming to your opinions in your report?

A. I would say that that is part of what I do, yes.

Q. Could you describe for me what your methodology is for your weight of the

evidence approach?

A. If you look at the summarization of the papers, just start out on Page 10 which contains Studies of Persons With Likely Exposure to Gasoline, one way to summarize the weight of the evidence is to summarize the data and that would be under or in the line titled Sums.

Another way to describe the weight of the evidence is to compare the distribution of observed over expected ratios, whether they're less than .9, .9 to 1.1 or greater than 1.1. So those are two different ways of summarizing the weight of the evidence.

Q. Your weight of the evidence approach is limited to looking at epidemiology studies. Is that correct?

A. Yes.

Q. And specifically it's limited to those epidemiology studies which you felt were properly included within either benzene-exposed populations and likely exposure to gasoline as it related to CLL?

A. Yes. I include all leukemia as well.

Q. So you include all leukemia?
A. In this particular review, I included all leukemia as well as CLL.
Q. So findings relating to all leukemia in your review are relevant to CLL?
A. No.
Q. But you included them?
A. I included them for comparison purposes.
Q. On Page 10, you have your summary of the data and you have a table for the Studies of Persons With Likely Exposure to Gasoline, correct?
A. Yes.
Q. And there you look at six studies; Schwartz, Wong, Hunting, Lynge, Milham and Sorahan?
A. Yes.
Q. And you provide the observed and expected numbers of leukemia in one column and then CLL in another and then what the ratios are?
A. Yes.
Q. And then you provide the sums of all the observed, all the expected and all the

ratios?

A. It's not the sum of the ratios. It's the ratio of the sums.

Q. Again, in that chart, you did not include, on Page 10, you did not include the Guenel or the Glass study?

A. No.

Q. And you did not include Schnatter's 1996 study?

A. No.

Q. And then on the next page you do the same thing for what you've chosen to be Studies of Populations Exposed to Benzene?

A. Yes.

Q. And there you also did not include the Glass study. Is that correct?

A. Yes.

Q. And you did not include Dr. Schnatter's 1996 paper either?

A. Right.

Q. Are you familiar with Wendy Huebner's 2004 study looking at Baton Rouge and Baytown Mobil refineries?

A. Yes.

Q. I'm correct, am I not, that refinery workers are exposed to gasoline?

A. I would say there's potential exposure. I don't know the nature of refinery workers in general.

Q. Wouldn't that be important for you to know in order to assign a study such as Dr. Huebner's study to benzene exposure or likely gasoline exposure category?

A. Yes.

Q. And am I correct that you did not include Dr. Huebner's 2004 study in your review and your report, did you?

A. No.

Q. You did not do that?

A. I don't see it.

Q. How come you didn't include her study?

A. I'd have to look at it and see.

Q. While you're doing that, can I have your copy of the Glass study, please?

A. (Witness complies.)

Q. Thank you, Doctor.

A. This is included in a group of

petroleum workers that I did not review because there was little or no information on specific exposures.

Q. That study found an SMR of 2.42 for chronic lymphocytic leukemia within the Baton Rouge finding, didn't it?

A. I believe so, yes.

Q. Would you agree with me that refinery workers are a population of workers that are exposed to gasoline and benzene?

A. As I say, I don't know that. I notice that it says during the war there was use of or production of high something, aviation gasoline. But it doesn't say how many people were exposed or how long this was done. And the paper doesn't talk about gasoline beyond that that I can see.

Q. So gasoline exposure was one of the exposures that the authors considered?

A. They mentioned it. I can't say that they considered it.

Q. Doctor, if you look to Page 12 of your report in the Conclusions section?

A. Go on.

Q. Under Studies of Persons Exposed to Benzene, second paragraph, you say there is no consistency among the 12 studies of persons exposed to benzene that there is a positive association between benzene and CLL. Do you see that?

A. Yes.

Q. Which of the 12 studies are you referring to?

A. The inverse studies would be Decoufle -- I'm sorry. 1983 Tsai, Rinsky, Collins, Ji.

Q. You say six show a positive association. Those would be Linos, Arp -- which ones are they that show positive association, the six?

A. Linos, Arp, Bernard, Malone, Guenel, Sorahan.

Q. Again, you're not including Glass?

A. The problem with Glass is that this is -- I didn't include Gun either. I agree that Glass shows a positive association on an internal analysis. But the external analysis

shows no excess. And so it's an ambiguous situation that by itself provides little information to base an opinion. I don't deny the existence of the Glass study nor its conclusions.

Q. You don't consider them in your analysis of the --

A. It's not a study of a population. It's a case-control study within a cohort.

Q. That makes it a nested case-control study, right?

A. It's more than that. That's the problem. To some extent, it's nested but then it goes beyond that. It's a combination of a nested case-control and a non-population-based case-control study.

Q. Certainly, in 2003, what were the expected number of CLLs for Glass' 2003 study? There's 11 observed, correct? How many expected?

A. I should have the Gun paper to put it in context.

MR. WOOLF: I have a copy of the Gun article with me if you want to see it in conjunction with what you're looking at.

THE WITNESS: All right.

BY MR. DuPONT:

Q. My question is is with respect to the Glass 2003 study. Do you know what the expected number of CLLs were?

A. The observed number was 11.

Q. Correct.

A. You said expected? And then the analysis was internal by cumulative lifetime benzene exposure. That's Table 6 and that's the relevant data in this article. So that under four cumulative lifetime -- under four PPM years by definition the relative risk is 1; between 4 and 8 is 2.8; and above 8 it's 4.5. It doesn't break it down by the numbers.

Q. So your answer is no, you don't know what the expected number of CLLs was?

A. No, because it isn't -- it's 11 in this particular analysis. Observed is 11 and expected is 11. And within that, she looks at the distribution but doesn't say how many observed cases there are in the three categories. And the intervals are extremely wide, so it's quite possible that the observed number in the

higher categories is 1 each or something like that.

Q. I respectfully move to strike your response. It was not a response.

My question was: Do you know what the expected number of CLLs was?

A. No, except as I said in this table the observed number and the expected number are the same. That's what I responded.

Q. Turn to Page 9 of your report. You provide your analysis of the 2007 ATSDR toxicological profile for benzene?

A. I'm sorry, let me just -- is this mine or yours?

Q. It's yours.

A. Did you write on it?

Q. I did not.

A. I'm sorry, go back to your question.

Q. Sure. Turn to Page 9 of your report.

A. Okay.

Q. At the bottom of the page, you provide your analysis of the ATSDR's

toxicological profile for benzene in 2007?

A. I provide a statement from the ATSDR report.

Q. You state that CLL is not discussed, correct?

A. Yes.

Q. Had the ATSDR found that there was suggestive evidence of association between benzene and CLL, would that have influenced your consideration?

A. I would have looked to see why they made that statement.

Q. Would a finding by the ATSDR that there was suggestive evidence that benzene causes CLL, would that influence your weight of the evidence analysis towards a more consistent finding of elevated risk for CLL from benzene exposure?

A. You're giving me a hypothetical and I can't answer that.

Q. Do you give credence to the fact that the ATSDR or other government agencies find suggestive evidence of the link between CLL exposure and benzene -- I misspoke. I'll

rephrase the question.

Is a conclusion of an agency of the federal government such as the ATSDR that there's suggestive evidence or causal association between benzene exposure and CLL, is that important in your consideration or weight of the evidence?

A. It's relevant but then I have to look at what the basis for that conclusion is.

Q. What would you want to look at?

A. I'd want to look at why they make that statement, which there should be a reference to the scientific literature.

MR. WOOLF: Counsel, since you've asked him about ATSDR, if you have a reference from the ATSDR that there is a suggestive basis that benzene causes CLL, I'd appreciate it if you would show it to the witness so he might comment on it.

BY MR. DuPONT:

Q. Would your answer be the same if the EPA concluded that there was suggestive evidence that exposure to benzene causes CLL?

A. Yes.

Q. So you would find that relevant and important to your analysis, correct?

A. I didn't say important. I said I would consider it as relevant to look at.

Q. If the EPA concluded that there's suggestive evidence of a causal association between CLL and benzene exposure, would that influence your decision that the weight of the evidence is stronger that benzene exposure causes CLL?

A. Not by itself. I'd have to look at the basis for that conclusion.

Q. Doctor, are you familiar with the Shanghai studies?

A. Are these the Chinese benzene studies?

Q. Right, being done by your clients.

A. No.

Q. You're not familiar with them?

A. No. My clients?

Q. ExxonMobil, Chevron, Texaco.

A. No. You're not talking about the NCI studies is what I'm asking?

Q. No, the ones that are being conducted by the American Petroleum Institute and some of the oil companies.

A. No, I'm not.

Q. So you wouldn't know whether those studies made any findings with respect to CLL?

A. No.

Q. Do you agree that the epidemiological findings as they relate to NHL are applicable to CLL?

A. No.

Q. If Dr. Schnatter testified that the epidemiological findings as they relate to B cell NHLs are applicable to CLL, would you disagree with him?

A. I don't know enough about the types of NHL to make a comment.

Q. Would you disagree with him? Would you think he's wrong?

MR. SCOTT: Object to form.

THE WITNESS: No, I can't make a comment.

BY MR. DuPONT:

Q. Doctor, you accept that benzene causes leukemia, correct?

A. I accept that at certain levels it causes myelogenous leukemia.

Q. And if you accept that benzene causes, as you say, acute myelogenous leukemia at certain levels, is what's the determinant for you is then the amount of exposure that the person sustains in order to link the benzene exposure to AML, correct?

A. In essence, yes.

Q. So all you're really looking at is the dose of benzene that the person intakes into their body, correct?

A. Specifically a measure that is used is parts per million years, which is an exposure measure.

Q. Correct.

A. Not a dose measure.

Q. And what's important is how much benzene exposure there is, correct, in your analysis?

A. How much there is for how long.

Q. No matter where that benzene

exposure comes from as long as you have that amount of benzene exposure for what you say is the correct length of period of time?

A. Can I just get a cup of coffee since I see it over there? Then I'll answer your question.

Q. I'll get you the cup of coffee, if you can answer the question?

A. With a little bit of milk. I was distracted by seeing the coffee, I'm sorry.

- - -

(Whereupon the court reporter read back the pertinent testimony.)

- - -

THE WITNESS: Yes.

BY MR. DuPONT:

Q. So, in your analysis, it's relevant not where the exposure to benzene comes from; what's important is the amount of exposure?

A. Yes.

Q. Doctor, the IARC, their monograph on gasoline was published in 1989. Is that correct?

A. Yes.

Q. So that predates Dr. Schnatter's 1996 study?
A. Yes.
Q. And it predates Guenel's study in 2002, correct?
A. Yes.
Q. Would you agree with me that a lot has happened with respect to benzene research since 1989?
A. Yes.
Q. Would you also agree with me a lot has happened with respect to gasoline research since 1989?
A. A lot is -- I'm not sure I would agree with that gasoline per se.
Q. I guess what I'm getting at is: Isn't the 1989 IARC monograph on gasoline a little outdated?
A. Yes.
Q. The risk in contracting leukemia from benzene exposure, is that supralinear at low doses?
A. At low doses? You have to define low.

Q. Is the risk in contracting leukemia supralinear at any point?

A. I have no information to that effect.

Q. Do you agree that benzene is a carcinogen by all routes of exposure?

A. Are you talking about human or animal?

Q. Human.

A. I don't know that. The only information that is in the epidemiologic literature to the best of my knowledge is inhalation.

Q. Now, are you familiar with how the EPA performs its weight of the analysis?

A. No.

Q. Strike that. That was a poorly phrased question.

Do you know how the EPA performs its weight of the evidence analysis as it relates to benzene?

A. No.

Q. Do you know how epidemiology studies rank in comparison to things such as

mechanistic studies or chromosomal studies or bioassays in the weight of analysis approach?

A. For EPA?

Q. Correct.

A. No.

Q. Why is it that epidemiologists use nested case-control studies as opposed to cohort studies?

A. Ordinarily, when one does a cohort study, one has only limited information on exposure either work area or substances. So, if they find an excess or if there's a disease that is of interest, they then go back and collect further exposure information on the cases of disease of interest and the comparison group.

Q. So would it then be an advantage of the nested case-control study that you have better information concerning exposure?

A. Theoretical advantage.

Q. Is it also an advantage of the nested case-control study that you have a better understanding of what the comparison population is?

A. I don't understand that question.

Q. When you have a nested case-control study, you're taking cases within a larger cohort and comparing them to the cohort as a total?

A. No, you're comparing them to a subset of the cohort.

Q. And the subset that you're comparing them to, is that generally better defined in other forms of cohort studies?

A. I still don't understand -- better defined with respect to what?

Q. With respect to what the comparison population is. Are you able as an epidemiologist to assure yourself or have a more better understanding of what the comparison population is?

A. You're talking about two different things. In the cohort study, usually the comparison population is external.

Q. Correct.

A. Either another company, another area within the same company or general population statistics.

Q. Correct.

A. In a case-control, a nested case-control study, the comparison group -- I call them a group rather than a population because they're selected on some basis from the overall non-case population.

Q. And because the comparison group in a nested case-control study is internal as opposed to external in the cohort study, you have a better defined comparison group for nested case-control studies?

A. See, that's what I have trouble with. You're saying "better defined". With respect to what?

Q. You have a more certain understanding of what exactly the comparison group is?

A. I don't think that that follows.

Q. You're familiar with the Bradford-Hill factors?

A. Yes. The term usually uses criteria but I wouldn't object to your using factors.

Q. In order to use the Bradford-Hill factors, you don't necessarily have to satisfy

all the different factors in order to come to a conclusion concerning causation, do you?

A. No.

Q. So they're not really criteria; they're just different things you want to look at?

A. Yes. The word "criteria" is used but it doesn't mean it's the best word.

Q. Sometimes, for example, the biological plausibility of a causal association will be stronger than say the consistency of the evidence?

A. You better rephrase that, please.

Q. Are there circumstances wherein reviewing the Bradford-Hill factors the biological plausibility of the causal association is stronger or more relevant than the consistency of the evidence?

A. You mean epidemiologic evidence?

Q. Yes.

A. Thank you. Would you repeat the question?

- - -

(Whereupon the court reporter read back

the pertinent testimony.)

- - -

THE WITNESS: I'm aware of one situation, and there may be a second, where IARC has considered mechanistic factors, which are biologic factors, if you will, in animals, to be sufficient to judge that the substance is a carcinogen in humans. That's ethylene oxide. There's very little human evidence on ethylene oxide.

BY MR. DuPONT:

Q. So where there is little human evidence, looking at the biological plausibility is more important?

A. There has in situations where that is correct. In general, it's not the usual way of making judgments.

I believe the reason that they came to that conclusion is they understand the mechanism that causes ethylene oxide to cause cancer in animals and they understand that the mechanism is the same in humans.

Q. You'd agree with me epidemiology

studies cannot rule out causal association?

A. Whether an association is causal or not is a matter of judgment. No study can either rule out or rule in the judgment of the causal association.

Q. Then it also would be true that epidemiology studies cannot rule out an elevated risk as a result of exposure of a particular chemical? In other words, if you have a negative finding in an epidemiology study, that doesn't mean that there's no risk to exposure to that agent, does it?

A. Single study, no. But if you got 100 studies which shows an elevated risk, I would say that's sufficient evidence to come to a judgment that there is no elevated risk.

Q. Are you familiar with the terms Type 1 error and Type 2 error as they relate to biases in epidemiology?

A. You say bias and I'm trying to think what I learned in statistics. I wouldn't call them biases. I would call them what statisticians call them, which is errors. And I'm familiar with the terms.

Q. The term Type 1 error, does that often refer to a finding that there is an association when none is actually present due to chance?

A. I'm sorry, I never remember which is one or two. One of them is that way and one is the other way. One is failure to find an association when in fact there is one.

Q. When you design an epidemiology study as an epidemiologist, do you design the study so it's more likely you're going to have an error of not finding an association than you would of actually finding association when it's actually by chance? In other words, do you try and design the study to avoid what I'll term the Type 1 error?

A. In most of the studies I've done, I haven't considered Type 1 or Type 2 errors because I study everyone available in a given population, so there's no issue as to is it too large, too small, whatever. You study everybody.

Q. When an epidemiologist sits down and designs a cohort study or other type of epidemiology study, is it the general practice to

design the study so that you're avoiding the error of finding a risk by chance?

A. That's done much more in clinical trials, how big a clinical trial should one do based on what one already knows.

In epidemiology, if you know nothing, you can't do a power calculation. And if you have a population of limited size, either you make a judgment is this population large enough to study. And in the absence of any information, there's no answer other than yes, it's large enough to study.

Q. In your analysis in this case, in your report as part of your methodology, do you make any kind of accounting for any biases that were present in studies that you reviewed?

A. Only to the extent if I felt the study was totally wrong, bad that I would not include it.

Q. For example, did you consider whether in any of your studies that you've listed in the report there is a misclassification of exposure or misclassification of disease?

A. No.

Q. Did you consider the Healthy Worker Effect in analyzing any of the studies that you relied upon?

A. On this report, no.

Q. Essentially, what you've done is a small meta-analysis of the CLL and leukemia literature?

A. No. I did a description.

Q. A description?

A. A meta-analysis makes a number of assumptions with respect to the papers that are being reviewed, and they wouldn't be met by a review of the literature. Meta-analysis, again, are much more relevant to clinical trials.

MR. DuPONT: Let's take a five-minute break.

- - -

(Whereupon there was a recess in the proceedings from 12:11 to 12:24.)

- - -

BY MR. DuPONT:

Q. Doctor, I have before me what's marked as Monson-A. It's your Evaluation of Papers by Steinmaus and then also your Evaluation

of Martin Smith's Paper on NHL. And the Steinmaus evaluation, if I'm correct, is dated July 7, 2008 at the bottom of the paper and then on your signature line it's also dated July 7, 2008. And the analysis of Martin Smith's paper is dated June 30, 2008.

When did you start the project that culminated in these two evaluations?

A. The Martin Smith one would have been sometime in 2007. I just, I put the date on when I printed it out. The Steinmaus one, again, was just after it came out, probably the beginning of this year.

Q. How much were you paid for your work to analyze the Steinmaus and Smith papers?

A. I really don't have any recollection. I can make a guess but I don't really know. Ten to 15,000 I'd say.

Q. Did you submit an invoice to Mr. Woolf for that?

A. Yes.

Q. You included in your evaluations discussion of Glass' 2003 article, correct?

A. If you say so, yes.

Q. Which study design or data collection biases did you analyze in your report in this case with respect to the literature that you cited?

A. Are you talking about Smith or Steinmaus?

Q. I'm sorry, I wasn't clear. Let's talk about Steinmaus first.

A. Why don't you give me your copy? Can you repeat the question, please?

- - -

(Whereupon the court reporter read back the pertinent testimony.)

- - -

THE WITNESS: First, I compared my reading of the literature to Steinmaus and either agreed or disagreed with his abstracting of data from the literature. So I did that for case-control studies of benzene exposure and NHL; cohort studies of benzene exposure and NHL; cohort studies of refinery work and NHL.

Next, I adjusted the relative risks that's similar to what Steinmaus

did in Tables 3 and 4 based on the all cancer SMR. And then I compared papers that Steinmaus either referenced, cited, or not referenced in Smith. Smith's paper had a lot of papers that he cited in his tables but didn't give references, whereas if I say referenced that means that Smith gave a reference with a number.

And there were some papers in Steinmaus but not in Smith, and there were some papers in Smith that were not in Steinmaus.

BY MR. DuPONT:

Q. And where do you provide your analysis of any design bias in the studies or any data collection bias in the studies?

A. I don't think I mentioned design bias per se. On Pages 4 through 6, I note where I agreed with what Steinmaus took out of the paper in terms of numbers and then when I disagreed.

Q. But I want to talk about design biases specifically for these various studies.

Did you look at and try and ascertain whether there were any design biases for any of these studies?

A. If I did it, it was implicit, not explicit.

Q. What do you mean by that?

A. I didn't go into it with that term in my mind.

Q. What types of design biases are there that you would be looking for?

A. I don't know. I didn't do it. I haven't thought about it. I don't want to make up something on the spur of the moment.

Q. So you didn't look at this literature that you cited in your analysis of Steinmaus for any design biases?

A. When I go through literature, the first thing I do is, as I said before, if I feel it's a paper that is of no value, I exclude it as a design bias. But I don't keep a list of such papers. Otherwise, if the paper is of reasonable scientific merit, I include it. There still may be design biases in it but I don't discuss them when I summarize the literature.

Q. So is there any way for me to know which studies you excluded because of a design bias as opposed to for some other reason?

A. No, I wouldn't say there were a lot.

Q. Is there any way for me to know which ones you excluded?

A. No.

Q. Did you find design biases in any of the studies that you included in your evaluation of Steinmaus?

A. As I say, I didn't look for design bias.

Q. How about data collection biases, did you evaluate any of the studies that you analyzed with respect to the Steinmaus report for data collection biases?

A. Not by the authors.

Q. What do you mean by that?

A. I didn't look to see whether an author, whether there was data collection bias in the paper itself.

Q. You mean the Steinmaus paper itself?

A. No, the paper that Steinmaus cited.

Q. What are the relevant design biases that an epidemiologist would consider in evaluating literature?

A. As I said, I haven't considered that at all in this report and I don't want to make things up on the spur of the moment.

Q. I understand that. Putting this report aside, generally, what are the various design biases that an epidemiologist would want to consider when analyzing the literature?

A. Mainly, the most common one is studies where an author selects people who have both exposure and the disease and then mills a study around it which guarantees an association.

Q. And what's the term for that type of bias?

A. Selection bias.

Q. Any other forms of design biases?

A. There's observation bias where either the disease is misclassified because of knowledge of the exposure or the exposure is misclassified because of knowledge of the

disease. Then there is a term "confounding" which is not a design bias but which is a characteristic of all scientific studies.

Q. Any other design biases besides selection?

A. Those are the three that I use.

Q. How about data collection biases?

A. I included that in what I was just saying.

Q. So data collection bias is a part of --

A. Design bias would be selection bias, data collection bias, and there would be observation bias.

Q. What about Healthy Worker Effect, where does that fall?

A. I call it confounded. Other people call it selection bias, and I disagree with them.

Q. Why do you call it confounding?

A. Because it's a characteristic of the individuals in the population and there is a potential for having knowledge of both health status and disease outcome and, therefore, you

have the ability to control for it.

Q. Did you do anything to determine how classification of disease may have influenced the findings?

A. No.

Q. Did you do anything to determine how classification of exposure in the various articles cited and referenced by Steinmaus influenced the findings?

A. No.

Q. If you go to Page 12 of the evaluation of Steinmaus that you've done?

A. All right.

Q. Under Title B, Evaluation of Papers Cited by Steinmaus, and then No. 1, Citations in Table 2, you say you disagree with eight of the authors' citations from these 16 papers. You say two of the papers include solvents other than benzene.

A. Right.

Q. Why do you disagree with their use of those studies that include solvents, as you characterize them, to be other than benzene?

A. It doesn't say that it's benzene

that's in the paper. One says all solvents.

Q. Did you do anything to determine whether or not benzene was in the solvents that the papers were referring to?

A. No.

Q. Did you do anything to determine that benzene was not in the solvents the paper was referring to?

A. No. I read the paper and if it said -- I read the paper. And if it didn't say this is exposure to benzene, I wouldn't include it.

Q. So am I correct in that what you're doing here in your evaluation of Steinmaus is you're evaluating the paper and either agreeing or disagreeing with the authors on, A, whether they chose to include a particular study or, B, how they characterized the study?

A. Yes, which includes abstracting data from the study.

Q. Lymphosarcoma, by the way, that's a form of NHL?

A. Yes.

MR. DuPONT: Why don't we take a

break for lunch?

As a matter of housekeeping, Doctor, this is the last portion of your file that hasn't been marked and I'll mark it as Exhibit-G. This is the May 29, 2008 e-mail from Ms. JoAnn Niedens on behalf of Louis C. Woolf to yourself enclosing the report of Paul Roda, report of Dr. Robert Laumbach. And then there are two articles, the Mehlman 2004 article from European Journal of Oncology, and Dr. Peter Infante's article Benzene: An Historical Perspective on the American and European Occupational Setting.

BY MR. DuPONT:

Q. Do these two articles go with Exhibit-G in that they're articles cited by Dr. Roda and Dr. Laumbach?

A. Yes.

Q. Then we'll group those with Exhibit-G. So then do we have the entirety of your file here?

A. Yes.

- - -

(Whereupon the document was marked, for identification purposes, as Monson-G.)

- - -

BY MR. DuPONT:

Q. Do you have invoices that you prepared for your work to date?

A. Once it's paid, I throw it away. I know I have at least one because it hasn't been paid.

Q. If I could ask you to provide that to your counsel and he can give me a copy of that, I would appreciate it?

A. Just to be clear, which invoices are you talking about?

Q. That's a good point, your invoice for your work on the Salmieri case.

A. Fine.

Q. I'd also like invoices for your work in preparing your evaluation of the paper by Steinmaus and Smith that we've marked as Monson-A.

A. Is that acceptable to Mr. Woolf?

MR. WOOLF: He'll just ask you

and then I'll take care of whether it's acceptable or not. I'll let you know.

THE WITNESS: Fine.

BY MR. DuPONT:

Q. And additionally, with respect to your requests to prepare this evaluation of Steinmaus and Smith, were there any written communications that set forth for you what it was that Mr. Woolf wanted?

A. If there were, I don't have them.

Q. If there are any, if I could ask you to provide those to your counsel as well? I'd appreciate it.

A. Fine.

- - -

(Whereupon there was a recess in the proceedings from 12:45 to 1:39.)

- - -

BY MR. DuPONT:

Q. Could you turn to your Summaries and Conclusions starting on Page 12 with respect to the Steinmaus?

A. Okay.

Q. First of all, do you agree that

the study power of an epidemiology study is higher when the relative risks are higher?

A. Power is generally used to -- when it's used in an epi study, it's used to decide what theoretical relative risk could be observed so that it's statistically significant. It's a predictive thing, not a descriptive thing. Do you understand the difference?

Q. Right. Go ahead.

A. So that the way one states it is that, if in fact there is an association in the data irrespective of causality, we're just talking about data now, if there is in fact a relative risk greater than one, if the relative risk is higher, it is more likely to be found to be statistically significant than if the relative risk is lower. I'm talking about relative risk above one now.

Q. Right.

A. So, in that sense, statisticians say something like you said that the study has more or only has the power to detect a relative risk of whatever, say five. It doesn't have enough power to detect one of three, even though

they may observe a relative risk of three.

Q. But, generally, the higher your relative risk gets, there's a corollary that the higher the power the study is?

A. Again, I want to be careful about prediction versus description. The confidence interval is the description of the data, whereas the power is something that's used in designing studies.

Q. Right.

A. It's not used in interpreting studies.

Q. Independent of classifying whether it's descriptive or interpretative --

A. Predictive. Interpretative is entirely different.

Q. Independent of the classifications, would you agree with me that when you see studies with higher relative risks that studies also have a higher power?

A. I don't use that type of statement. That's a description of a study that's already been done. Power is used before the study is done.

Q. Regardless of whether you use that type of statement, is that true --

A. If I don't use it, it's irrelevant. I don't comment on that situation.

Q. How can you say it's irrelevant just because you don't comment on it?

A. Because I think it's incorrect to say that.

Q. So you think it's incorrect that when you have a higher relative risk there's usually a corollary that the study power is higher as well?

A. You stated that differently than you did before. See, I don't use the term "power" hardly at all. I don't want to go beyond that. I don't want to make something up.

Q. What do you use instead of "power"?

A. Confidence interval.

Q. Would it then be true that the higher relative risks are found in a study the narrower confidence intervals are?

A. No. Confidence intervals has to do with both the relative risk and the size of

the study.

Q. Do you agree with the proposition that higher relative risks are less likely to be due to confounding factors?

A. I've written that, so I agree with it in general. It's not an absolute statement.

Q. Do you agree that studies where you have an internal comparison, such as nested case-control studies, that those types of studies are not subject to the Healthy Worker Effect?

A. In general, yes.

Q. Turning to your paper under Summaries and Conclusions on Page 12, you say that the authors' methods do not accord with methods recommended by other authors. In other words, Steinmaus, their methods don't accord with methods recommended by other authors. Do you see that at the bottom of the second paragraph under Comment?

A. Yes.

Q. Which authors are you referring to? Which other authors are you referring to?

A. Bottom of Page 2, Checkoway, et

al.; top of Page 3, Breslow and Dade and Greenberg, et al., as well as Checkoway again.

Q. How are the Steinmaus methods different than those recommended by Checkoway and Breslow?

A. The specific issue had to do with how one measures exposure and all the papers -- I shouldn't say all. Many of the papers use different methods.

You can use peak exposure. You can use duration of exposure. You can use average exposure. You can use length of work. Anyway, there's different ways to describe exposure which industrial hygienists use. But the standard way and the one that these authors recommend is use of what I said, PPM years, which is in essence cumulative exposure. And that's what I used.

Q. So one of your disagreements with Steinmaus is that they do not use or they do not solely use cumulative exposures?

A. That's right. They seem to pick the one that they like the best.

Q. And you believe that Checkoway

and Breslow in their articles recommend the use of cumulative exposures?

A. Yes.

Q. Then you say that the rules used by Steinmaus are arbitrary. Do you see that under Results on Page 12 of your report?

A. Yes.

Q. Which rules do you believe Steinmaus used that are arbitrary?

A. There's, as I said, there's a fair amount of, fair number of papers that Steinmaus either uses that Smith doesn't or vice versa and it's not clear why. So, if it's not clear, it's arbitrary as far as I'm concerned.

Q. So, because you don't understand why they selected, Steinmaus selected to use certain papers that Smith either did or did not use, that's what you believe is arbitrary about the rules?

A. Yes. Some of them clearly were incorrect in Smith. Steinmaus was correct in not including them for a variety of reasons. But there are others that it wasn't clear why they were in Steinmaus, and that's on Page 11.

Q. So which are the papers that you believe it's not clear why Steinmaus decided to use them?

A. I can't. I don't have that listed and I don't remember.

Q. Besides selecting papers to use without clear reasons for doing so, are there any other rules used by Steinmaus that you believe are arbitrary?

A. There were two things I pointed out, one is the one-sided P value, but that doesn't enter into any of what I did so we'll just let it go. I don't believe in one-sided P values.

The other thing is the Healthy Worker Effect. I would say it's generally accepted that the Healthy Worker Effect for cancer is less than that for all causes and that's just safer -- for example, if the Healthy Worker Effect for all causes is .8, then for cancer it would be .9. It's just to illustrate what I mean.

Q. Okay.

A. So that one should really correct

for it comparing it to the SMR for all cancer rather than for all causes.

Q. And why is that? What's your basis --

A. Because if you got a cancer, you should compare it to other cancers. Most of the Healthy Worker Effect is due to non-cancers which can be detected at the time people start working, whereas cancer cannot be predicted at that time.

Q. Isn't the Healthy Worker Effect the result of a judgment of a person when they first start to work?

A. The Healthy Worker Effect is an observation that studies of workers compared to general population shows a lower than expected mortality. And that is because of selection at the time people search for it. But it's different for different causes.

Q. Do you have a greater power or greater ability to determine the Healthy Worker Effect by looking at all causes as opposed to cancers because there are more cases of all causes than there are of cancers?

A. No. All causes includes diseases

that, as I said, people tend to develop before they're say age 20, which is sort of an average year of starting work; chronic respiratory disease, chronic GI disease, chronic cardiac disease. Many chronic diseases that develop in younger people lead to the Healthy Worker Effect.

Q. I guess my question is: If you're looking at two populations, you're looking at cancers and you're looking at all causes of death?

A. Those aren't two populations. Those are two types of outcome.

Q. Right. If you're looking at a group of workers and trying to determine whether or not there's a Healthy Worker Effect, those workers who died of cancer, there's going to be less of those workers dying of cancer than there are going to be of workers dying of all causes, correct?

A. Yes.

Q. So that you have a greater power to determine the Healthy Worker Effect by looking at all causes?

A. You don't use it in that context.

You use the confidence interval. You'd have a narrower confidence interval for all causes than for cancer, but you'd have a higher relative risk that's closer to 1 for cancer than for all causes. So they work in opposite directions, so I can't answer the question.

Q. You also say the data selected by Steinmaus is arbitrary?

A. That's where I'm talking about the various measures of exposure to benzene. In a paper where there are different ways of doing it, he tends to pick the one that gives the highest relative risk.

Q. Have you reviewed any literature that suggests that peak exposures and other models of benzene exposure are more important in the causation analysis --

A. I'm aware that such literature exists, and I'm sure I've read snip-its of it.

Q. Do you disagree?

A. I'm not convinced that there's a strong case that can be made for it.

Q. But do you disagree that that's true?

A. I don't disagree and I don't agree. I'm aware that some people hold that view, and I believe that it differs for different chemicals or physical agents.

Q. By the way, have you ever testified in tobacco litigation?

A. No.

Q. Have you done any work in the context of tobacco litigation?

A. I wrote a report once for a tobacco lawyer. He didn't like it.

Q. Going back to Page 12, under Title B, Evaluation of Papers Cited by Steinmaus, et al., Subpart 1, Citations of Table 2, we discussed the two papers that includes solvents other than benzene. Which two papers are those you're referring to?

A. Sorry, I lost my place.

Q. Page 12.

A. No, but I refer to one of the tables.

Q. I'm sorry.

A. Citations in Table 3, so it's Page 5 -- that's not correct. I'm sorry, which

question did you just ask me?

Q. There's Citations in Table 2.

A. Okay, 2. You say two of the papers includes solvents other than benzene.

Q. My question is which papers are you referring to?

A. Yes, Dryver and Franceschi.

Q. What are the solvents that are included in Dryver?

A. Aromatic, all aromatic.

Q. Aromatic solvents containing benzene. Is that correct?

A. Benzene is an aromatic solvent.

Q. Do aromatic solvents contain benzene?

A. Some do and some don't. If the classification is all aromatic solvents, then benzene would be included.

Q. Do you recall how Dryver defines aromatic solvents?

A. No.

Q. Incidentally, did Dryver find an increased risk of NHL from exposure to gasoline?

A. I don't know, unless it's in my

other report. It's not in this report. On NHL, I don't know.

Q. Look at Table 2 of the Dryver study. I point your attention to the category for Gasoline Greater Than Five Years Versus Less Than Five Years. You find an OR of 1.92.

A. Okay.

Q. Is that correct?

A. Yes.

Q. And that's statistically significant?

A. Yes.

Q. Why do you disagree with the selection of Franceschi?

A. Again, it says all solvents.

Q. What type of solvents were being used in the Franceschi study?

A. I don't know.

Q. Which study from this Table 2 do you say was used in a misleading way?

A. Is there only one?

Q. I believe you say in your commentary that there was one.

A. I say two.

Q. Do you? Then I apologize.

A. Fritschi 2005, she reported a relative risk of 0.3 based on two cases with substantial exposure. But if you look at all exposure, you get a relative risk of 1.1. So the substantial group is so small to be meaningless.

And your other one that's misleading is Glass. There's no dose response relationship, and when you do a post hoc division of PPM years, you usually cut the line where the data leads you to and that's not appropriate.

Again, if you use all 20 cases, the odds ratio is .9. If you use only two cases, it's 1.5. It's a similar criticism to Fritschi.

Q. You say that there are cases that are incorrectly cited out of this Table 2.

A. Fabbro-Peray, I say not final analysis. I don't know specifically. I think that probably corrected for confounding or something.

Schnatter, it's hard for me to decipher exactly what I meant. But it didn't change relative risk very much, .9 to .8. Even that, that's a guess.

And in Seidler, there were actually 12 cases and not 11. There's one other case that was sort of in another table. Again, it doesn't change the relative risk substantially.

Q. So, in Seidler, there are 12 cases because there's one case reported in a separate table?

A. It's either in a separate table or a separate section, yes. There is the 11 but then there's one somewhere else and you just missed it.

Q. Anything else?

A. No.

Q. Then you have a discussion of Citations in Table 3 and you say you disagree with two of the citations. Those would be the Bloeman 2004 and Wong 1987?

A. Right.

Q. Why do you disagree with Bloeman?

A. It's highly selected data of 15-years lag. The Bloeman citation is based on two cases with a 15-year lag with exposure greater than 28 PPM years, which gives a relative

risk of 2.2. But if you use all three cases and no selection in terms of follow-up, you get a relative risk of .7. So major differences based on very small numbers.

Q. Why do you disagree with Wong?

A. It's based on only one case. And Steinmaus states the relative risk is 1.5, but in Table 11 on Wong, the relative risk is 0.7. And I can't go beyond that. That's what I wrote.

Q. You say in Citations in Table 4 you disagree with eight of the citations. The first is Collingwood 1996. That's at the Paulsboro, New Jersey refinery?

A. Right. Again, there's only two cases and you can pick and choose. Steinmaus used Table 7 and I used Table 5, which I assume I thought was a more relevant table.

Q. Why is that? Why did you use 5 instead of 7?

A. I don't know. It's the same number of cases and it doesn't change the relative risk that much. But I didn't go through these papers at all in preparation for this, so I don't know.

Q. You disagree with the Dagg 1992 El Segundo and Richmond studies?

A. Right, superceded my Satin.

Q. So, since Satin later published on the same cohorts, --

A. Right.

Q. -- you disagree with their use?

A. Right.

Q. You disagree with the use of the Gun 2006?

A. Only because they used mortality and not incidence.

Q. Why is that?

A. Incidence, if you have it, it's better information because it includes non-fatal cases.

Q. Next is Thomas 1982.

A. Right. This is one of a whole bunch of studies that were superceded by later papers.

Q. You say disagree, data likely included in Divine and Satin?

A. Right.

Q. What makes you think it was

likely included?

A. There's other papers that, there's at least two other papers that I didn't put in because I don't think Steinmaus did. But they were members of the OCAW, Oil, Chemical and Atomic Workers. They were case-control studies. And they were in companies that were later, in which cohort studies were later conducted during the same time period.

Q. What information do you have to show you that the Thomas data was included in Divine and Satin?

A. As I say, I believe they belong to the union in the plants that were studied by Satin and by Divine.

Q. And then next is Tsai 1993. Is that the same reason? You say you disagreed because it did not include the Wilmington plant.

A. Yes. I guess Martinez is one plant and Wilmington is another. And the paper includes both plants, but Steinmaus only picked the one where there was slight excess.

Q. And then the last two that you disagree with is Wong 2001a and Wong 2001b. And

A related to Beaumont and B to Torrance.

A. Here, Steinmaus chose to put in people who had been exposed for 30 or more years which gave higher relative risk than those. I tried to include all data in these tables.

Q. When you say three citations from this table are outdated, are you referring to the two Dagg and the Thomas 1982 which you believe were superceded?

A. I think that's right, yes.

Q. When you say four are cited in an arbitrary and misleading manner, which ones are you referring to?

A. What page are you on now?

Q. Still under discussion of Citations in Table 4.

A. So you're up here?

Q. Page 13.

A. Yes, I see. So Collingwood would be arbitrary, Gun and the two Wong papers.

Q. One of the main things you focus on is there's six papers on NHL not included by Steinmaus that were apparently included in Smith, and you point to the appendix on the Smith

evaluation to identify which papers those were. Which appendix are you referring to?

A. It would be Appendix A of this report. I only have -- did I get this from you or I got this from Lou? You should have the appendix. It starts with A. It would be Page A1 or A16; I'm not sure.

Q. Let me hand back Exhibit-A to you and ask you to refer me which appendix you're referring to.

A. That was a separate report that I included in the appendix to this report because the whole thing is very confusing.

Q. Which of the six studies that you say were not included within Steinmaus that were included within Smith?

A. I can just read these.

Q. What page are you reading from?

A. On Table 8.

What I'm questioning is you say that these were included in Smith and I just want to make sure that that's correct. Where do you read that?

Q. If you look to the last page of

your report or your summary of Steinmaus above where you give conclusions, --

A. Right.

Q. -- under Comment, it says of special interest are the six papers cited by Smith but not mentioned by Steinmaus.

A. Okay.

MR. WOOLF: If you guys will look at Page 11, it's talking about the same six studies.

MR. DuPONT: Okay.

THE WITNESS: I guess that's it then, okay, because there's another table with six studies and that's on Page 8 and they're the same six studies. Okay, so we're clear on that.

MR. WOOLF: No, they're not.

THE WITNESS: They're not?

MR. WOOLF: No. If you'll turn to Page 11?

THE WITNESS: I'm turning to Page 8 and Page 5. They're the same.

MR. WOOLF: Yes.

THE WITNESS: Thank you.

BY MR. DuPONT:

Q. So the six studies listed on Page 5 and the six studies listed on Page 8 are the studies you claim were omitted by --

A. That's what I'm uncertain about.

MR. WOOLF: Dr. Monson, if you look at Page 11 of the critique and refresh your recollection?

THE WITNESS: Thank you. That's the different six studies. Thank you very much.

All right, so these would be the papers that are not included in Steinmaus but are included in Smith on Page 11.

BY MR. DuPONT:

Q. And why do you think they should have been included in Steinmaus or not?

A. I didn't say I think they should. I'm saying they weren't even though they were included in Smith. And since Smith is the co-author, obviously they were working together. I don't know why Steinmaus didn't include them.

Q. Why do you critique the fact that Steinmaus didn't include them?

A. Steinmaus?
Q. It is Steinmaus.
A. Then I spelled it wrong. Because I think they're relevant papers.
Q. Do you disagree with the ATSDR's evaluation that there's suggestive evidence for benzene causing NHL?
A. Did I put that in my report?
Q. I believe you did.
A. I wouldn't have done it in my CLL report, and I don't think it's in this report either.
Q. Turn to Page 9 of your report in this case, Salmieri. You quote the ATSDR's 2007 toxicological profile for benzene, Page 223, including the quote that there is suggestive evidence of associations between benzene and non-Hodgkin's lymphoma and multiple myeloma. Do you disagree with that statement?
A. I agree the ATSDR stated that.
Q. Do you agree with their position?
A. I wouldn't go so far as to say suggestive evidence. I believe that's an overstatement. I would agree that there are

papers that show positive associations. But the weight of the evidence does not support anything beyond saying that some studies show a positive association, some show a negative association and there's no consistency among the studies.

Q. So the ATSDR has it wrong but you're right?

A. They're not wrong. I'm saying the word "suggestive" is too strong to me.

Q. If you were to rank it on a scale of one to ten, what does the term "suggestive" mean with ten being the highest and one being the lowest?

A. I don't have any number in mind.

Q. But suggestive, you used the word "suggestive" causation is strong?

A. I don't quantitate a substantive, a descriptive term.

Q. Nonetheless, you believe it's a strong description of the causal association?

A. I'm sorry?

Q. To say that there's suggestive evidence, you believe that --

A. I didn't say it's strong. I said

it's stronger than I would say it. I would say it's weak. Suggestive means weak to me. But I would make an even weaker statement.

Q. Are you familiar with Lewis' 2003 study where he found a relative risk of 1.81 for CLL?

A. I'm familiar with several studies by Lewis but I don't have any recollection of the specific ones, if you can show it to me.

Q. I will show you the cover page with the citation.

MR. WOOLF: What's the year of that?

MR. DuPONT: 2003, Lou. It's Mortality and Cancer Morbidity in Cohort of Canadian Petroleum Workers.

THE WITNESS: I've seen it but I don't know of any specifics on it.

BY MR. DuPONT:

Q. Are you able to tell me why you didn't include it within your review?

A. It's a petroleum study and I didn't include petroleum studies per se.

MR. WOOLF: What was that, Lewis?

MR. DuPONT: 2003.

THE WITNESS: Occupational and Environmental Medicine, OEM.

MR. DuPONT: That's all I have.

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(Witness excused.)

(Deposition concluded at approximately 2:27 p.m.)

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INSTRUCTIONS TO THE WITNESS

Please read your deposition over carefully and make any necessary corrections. You should state the reason in the appropriate space on the errata sheet for any corrections that are made.

After doing so, please sign the errata sheet and date it.

You are signing same subject to the changes you have noted on the errata sheet, which will be attached to your deposition.

It is imperative that you return the original errata sheet to the deposing attorney within thirty (30) days of receipt of the deposition transcript by you. If you fail to do so, the deposition transcript may be deemed to be accurate and may be used in court.

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ACKNOWLEDGMENT OF DEPONENT

I,_____, do hereby certify that I have read the foregoing pages, 1 - 125 , and that the same is a correct transcription of the answers given by me to the questions therein propounded, except for the corrections or changes in form or substance, if any, noted in the attached Errata Sheet.

_____

DATE SIGNATURE

Subscribed and sworn
to before me this
day of , 20 .

My commission expires:

Notary Public

CERTIFICATE

I HEREBY CERTIFY that the witness was duly sworn by me and that the deposition is a true record of the testimony given by the witness.

Kimberly S. Gordon, a
Registered Professional Reporter,
Certified Court Reporter
and Notary Public
Dated: SEPTEMBER 22, 2008

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LAWYER'S NOTES

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