SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

WILLIAM MOLINA and ANGELINA MOLINA,

Plaintiffs,

-against- Case No. BC 367800

SHELL OIL COMPANY, et al.,

Defendants.

Friday, April 11, 2008

11:20 a.m.

TELEPHONIC DEPOSITION of the Non-party Witness, DR. JOHN WHYSNER, Ph.D., DABT, taken pursuant to agreement, held at the Marriott Hotel, 670 White Plains Road, Tarrytown, New York before a Notary Public of the State of New York.

Reported by:

Christy J. Rockower

JOB No. 86263

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D R. J O H N W H Y S N E R, Ph.D., DABT, the witness herein, having been first duly sworn by Christy J. Rockower, a Notary Public of the State of New York, was examined and testified as follows:

EXAMINATION CONDUCTED

BY MR. HANLEY:

Q Would you please state your name?

A Dr. John Whysner.

Q What is your address, please?

A 166 Farrington Avenue, Sleepy Hollow, New York 10591.

Q Good morning, Dr. Whysner.

A Morning.

Q Can you describe for me any work that you've done since we've last spoke during your deposition on April 1st?

A Well, I read over my -- the transcript of my deposition, and I read over Dr. Bennett's, the deposition or whatever you call it that was going to be used at trial. I didn't have a chance to read over his prior day deposition. And

based upon that, I also, because you'd asked him a number of very specific questions about the Hayes article and the Chinese study, I was looking into the references that -- within the Hayes study there are certain references to other articles that describe more fully certain aspects of the study, and so I obtained those and -- in other words, so in case you were asking me more detailed questions like you did with Dr. Bennett. And those are the two articles that were sent to you, the Travis article and the Dosemeci article, D-O-S-E-M-E-C-I article.

Q Okay. And you have copies of those articles there with you?

A Yes, I do.

Q Okay. And just for reference, we'll go ahead and mark those two articles as exhibits, first exhibit to the deposition.

(The Travis article and Dosemeci article were marked as Plaintiff's Exhibit No. 1 for identification.)

Q Is there any other work that

you did or articles that you reviewed?

A Well, I went back through my files in order to be able to bring them here today. I looked over some of the references that I had previously brought to my prior deposition.

Q And what materials did you review specifically?

A Well, specifically, mostly the -- those solvent case-control studies, because in many of the cases, remember I had put these colored stickys on them, and in many of the cases they were not placed in exactly where I had put them before, and so I wanted to go through them to make sure that at least that file had the -- had the markings, so that if you wanted to discuss any of those papers, I could quickly get to the relevant information.

Q Are there any particular solvent case-control studies that you reviewed?

A Well, I read -- there were 31 of them, I think, and I looked over all of them. I didn't really read them.

The only thing, like I mentioned before, that I -- I looked at in some depth was the -- was not one of those, but was a Hayes study, and also then the papers, the two papers, that have just been marked as Exhibit 1.

Q Since your last deposition in this case on April 1st, how much time have you spent working on the Molina case?

A I would say probably maybe ten hours, something like that.

Q And how much of that time was spent discussing matters with counsel?

A I would say maybe 15 minutes, maybe a little bit more.

Q Was that one conversation or multiple conversations?

A Well, there are a couple. Well, we talked a lot about scheduling and what was going to happen.

And what I had -- it wasn't clear to me whether or not I had to bring everything, and I did wind up bringing everything back again.

And I mentioned the articles

that I wanted to -- well, counsel had informed me about the Bennett deposition and that they were going to send that to me. I mean -- well, whatever you want to call it, trial testimony, and the deposition, and so they in -- that was one conversation where they informed me about that. And then after I reviewed the Bennett testimony, then I said to Miss Khan -- Mrs. Khan, that I wanted to include these two articles that are referred to in the Hayes article because they describe both the exposure assessment and also how the cases were evaluated in terms of the medical records.

Q What is the import of these two studies that we have attached as Exhibit 1 in your way of thinking?

A Well, I think that the, for example, the Travis article in particular talks about how cases were verified in terms of whether or not they were non-Hodgkin's lymphoma. And I just -- I was interested in that because you and Dr. Bennett had spent some time talking

about the degree of verification because I'd also remembered that the Mayo Clinic was involved in doing some of the verification, and I wanted to see to what extent they were able to verify cases of NHL. And it turns out that whereas there was a lot of leukemia cases that were able to be verified because it included peripheral blood slides, which, from what I gather, was more available. There were only four of the 12 NHL cases that could be verified by histopathological evidence. And that's found on page 93 of this paper, the first column, the first paragraph. And so it is -- it indicated to me that there may be some uncertainty about the actual diagnoses for the other 12 cases.

The other thing that is also, I think, of some importance, is that this is the paper where they actually talk about how many non-exposed workers they had. And also in that same page, table 1, the non-exposed workers, there were only three cases of malignant lymphoma and related disorders, which would have included

non-Hodgkin's lymphoma. And I thought that that number was quite a bit lower than one would expect.

In other words, in the Chinese studies, the reason that they compared them to non-exposed workers is that they don't have the kind of standardized mortality statistics that we have in the United States so that you can do a study and then you can compare the mortality of the group that you're studying to the general population. And so they had to have a, in essence, a comparison group. And that's why they talk about relative risks rather than SMRs.

And if the group that you're comparing, using for comparison, happens to have a lower than expected mortality rate, then that would tend to overinflate or to inflate the comparison to the group that you're interested in studying.

And so I -- you know, that's one of the things that I look for in these types of studies is to see what is the quality of the group that they are using

for comparison.

Q And in this last answer you just gave, are you referring to the Travis article?

A Yes.

And then, I guess you asked me about the articles, plural. And the Dosemeci, sorry I can't pronounce his name very well, but I wanted to see also what were the exposure levels that were involved in these studies. And I think it's clear that these were people that had reasonably high levels of exposure in terms of part per million in the atmosphere, and that can be seen on table 3 of that paper on page 408.

Q Do you agree with the findings of the Travis authors as reflected in the last sentence of the abstract where they state, "Results in our series, one of the largest to date, also indicate that a greater diversity of hematologic neoplasms is evident among benzene exposed workers than previously described"?

MS. KAHN: Objection. Overly

broad, vague and ambiguous, and calls for speculation as to what the authors had in mind.

A I mean the problem with their statement is they are not really saying anything other than the fact that there is -- there are these hematologic neoplasms in these people who are exposed, but they really don't say whether those were at a higher rate than would be expected or at a lower rate than would be expected. So I'm really a little puzzled by the way in which they describe -- made that description.

Q Do you agree or disagree with that statement of those authors?

MS. KAHN: Objection. Asked and answered.

THE WITNESS: I don't have anything to add to what I just said.

BY MR. HANLEY:

Q My question, sir, is: Do you agree or disagree with that statement?

MS. KAHN: Objection. Argumentative, asked and answered.

THE WITNESS: Well, I think

the statement is just -- all it is is saying is that there are these hematological neoplasms among benzene exposed workers, and I don't really have any disagreement with that statement because it really doesn't -- I mean that's evident that within any group of workers there would be a -- there could be a diversity of all sorts of different diseases. The crucial question is whether or not those are occurring at a greater rate than would be expected or not.

BY MR. HANLEY:

Q Isn't that the clear implication of what they are saying? Isn't that the import of that statement that there is an increase in these, in a variety or diversity of these cancers of the blood forming organs?

MS. KAHN: Objection, the question calls for speculation, it's leading and suggestive. It's overly broad.

THE WITNESS: No, I don't

think it says that or that's the implication necessarily.

BY MR. HANLEY:

Q Well, when they say that, "The results in our series, one of the largest to date, also indicates that a greater diversity of hematologic neoplasms is evident among benzene-exposed workers than previously described," what are they saying is the greater diversity of hematologic neoplasms that is evident compared to what has been previously described?

A Well, I'd have to speculate about what they actually are trying to say. I think I gave you my answer about when you tried to say, What does this imply? And I said that I don't think it necessarily implies what you had stated previously.

Q You don't think that it's explained by the context of the abstract which starts out saying that, "Although the relationship between benzene and acute non-lymphocytic leukemia, ANLL, is well established, most of the analytic cohort investigations examining the relationship

between benzene and hematologic neoplasms have evaluated only death certificates to validate diagnosis." You don't think that the context of the last sentence is to be taken from the abstract as a whole including the first sentence?

A No.

Q So is your -- is the bottom line of your testimony that you agree or disagree with the last sentence of the abstract?

MS. KAHN: Objection. Asked and answered, overly broad, vague and ambiguous, argumentative.

THE WITNESS: I don't have anything to add to what I've already said. I think you've asked that question before.

BY MR. HANLEY:

Q I have, but I'm waiting for an answer whether or not you agree or disagree with that last sentence of the abstract?

MS. KAHN: Objection. Overly broad, vague and ambiguous, argumentative, asked and answered.

THE WITNESS: I don't know any other way I can answer the question other than the way I already have.

BY MR. HANLEY:

Q Do you understand what they are saying in the last sentence? Do you believe that you understand what it is that they are saying?

A Well, I think what they're saying is that they have looked at a number of different hematological diseases in this group of people, but I don't think they have drawn any conclusion regarding it. I think that's what they are saying is that there is a -- they are looking at a more diverse group of hematological neoplasms. And don't forget that before this was published, the Rinsky cohort only really talked about leukemia. It wasn't until 2002 that, from the way I read the literature, my understanding is that Rinsky reported on their lack of increases in NHL. So I think what they are implying here is that they are looking at a wider range of hematological neoplasms than previous

authors had described in 1994. And I think that's the correct interpretation.

Q If you go to the Discussions section on page 98 of the article and look at the second paragraph of the second section, and you'll see where the author states that, quote, "Another new finding in our study is the greater diversity of hematological malignancies than usually reported with benzene exposure, perhaps due in part to the sizeable number of subjects in a variety of occupational settings."

Did I read that correctly?

A I'm missing where you are.

Q In the Discussion section on page 98 of the article.

A Okay.

Q If you look at the second paragraph in the first sentence of the second paragraph, did I read that sentence correctly?

MS. KAHN: Objection. The document speaks for itself.

THE WITNESS: Again, this says pretty much the same thing as we

were discussing before in the abstract. What these authors are talking about is that they have -- they have looked at a greater diversity of neoplasms in this particular study than has previously been studied, and I think that's a fair statement in terms of the way that they have reported the data.

BY MR. HANLEY:

Q So you would agree with that sentence?

A Well, I don't know that --

MS. KAHN: Objection.

THE WITNESS: I don't think I agree with the kind of interpretation that you're trying to imply from that sentence. I think that the sentence speaks for itself. And I think that what my interpretation -- I gave you my interpretation of what I believe it says, and I don't see how I can do anything different from that.

BY MR. HANLEY:

Q Do you agree or disagree with that sentence?

MS. KAHN: Objection. Asked and answered, argumentative.

THE WITNESS: I've already answered the question. I don't have anything to add to it.

BY MR. HANLEY:

Q Sir, do you agree or disagree with that sentence?

MS. KAHN: Objection. Asked and answered.

THE WITNESS: As I mentioned before, what I find in this sentence that I agreed with is that, yes, indeed they have examined a wider diversity of hematological malignancies. And what I see here is that they were able to do it because they had a sizeable number of subjects, because, don't forget, they looked at, I think, 74,000 benzene-exposed workers, so that allows you to be able to look at things that are less common than the leukemias that have been -- had been previously described.

BY MR. HANLEY:

Q These authors go on to publish

in subsequent papers, including the 1997 Hayes article that I was examining Dr. Bennett about, that in fact they associate high levels of exposure to benzene with an increased risk for non-Hodgkin's lymphoma; is that correct?

MS. KAHN: Objection. It's vague and ambiguous. It's not clear if by "these authors" you mean Travis et. al, or another set of authors. Also, the question is compound and overly broad.

THE WITNESS: Well, could you repeat the question, please?

BY MR. HANLEY:

Q The authors of this particular study, the Travis -- 1994 Travis article that we're reviewing include Dr. Hayes; correct?

A Yes.

Q And Dr. Hayes is one of the researchers that has published repeatedly regarding this Chinese cohort exposed to high levels of benzene occupationally; correct?

A Yes.

Q And Hayes, in subsequent studies, based on the same data described a -- in this 1994 article has gone on to conclude and published in the scientific literature, including the 1997 article that I was asking Dr. Bennett about, that exposure to benzene in the context and at the levels described in the Chinese workers is associated with an increased risk for development of non-Hodgkin's lymphoma; correct?

A Well, no, I don't agree with that, because in 2000 Hayes et. al said, and I quote, "We noted, however, that an excess of NHL was found in our study which could potentially be attributed to other exposures as the excesses were significant only in the subset of benzene exposed chemical workers."

Q Where are you reading that from, sir?

A This is one of the articles in my files which is Hayes, and that is from the Journal of Toxicological and Environmental Health, year 2000.

Q Are you saying that Hayes has published that non-Hodgkin's lymphoma is not associated with benzene exposure?

A What he is saying is that the associations they found -- don't forget, these people were exposed to a lot of different chemicals, not just benzene. And so what he's saying is that the excess that they found in their study could have been due to co-exposure to other chemicals because the excesses -- what he said, and I quote, "The excesses were significant only in the subset of benzene exposed chemical workers." And, so in the other industries, what he's saying is that these excesses weren't significant.

In order to have some degree of confidence in one's findings, I think one have to be able to see the kind of consistency among different industries because what we're looking at is benzene exposures; but, if you see a large excess as you did in the chemical workers who were exposed to lots of different chemicals and you don't see those in some of the other

industry groups. I think it leads you to the concern that this excess might be due to the other chemicals that the chemical workers were exposed to rather than to benzene.

Q Yes, those other chemicals that he's referring to are other petrochemical hydrocarbon solvents; correct?

MS. KAHN: Objection. The question is overly broad, vague, and calls for speculation.

THE WITNESS: If you would like to refer me to something that is what they are saying, I would be glad to look at it, but I don't recall that that is the case.

BY MR. HANLEY:

Q What other chemical exposures is he talking about that would -- that you have any information based on the context of the studies or the people being evaluated that would suggest that it was anything other than hydrocarbon petrochemical solvents that he's talking

about?

A If you want to give me some time to look through this, I could try to find it.

Q Yes, sir, if you can find anything that Dr. Hayes or any of his fellow authors have said in any of these studies that suggest that the other chemicals that he's referring to are anything other than petrochemical hydrocarbon solvents, please point it out to me, sir.

A Well, it would take me a while.

Q Well, do it. If he's not talking about petrochemical hydrocarbon solvents as the other chemical exposures, you show me where it says that.

A Well, you show me where he says that it is about petrochemical exposures.

Q I'll show you that all of these cohorts that he's looking at are people that are exposed to petrochemical hydrocarbon solvents, including benzene and

including other petrochemical hydrocarbon solvents. That is, from what that I see from these studies, the clear and obvious import of their findings. You show me where there's something different.

A Okay. Page 404, table 1. Chemical workers. Organic synthesizers.

Now, organic synthesizers means they are probably involved with a lot of reactive intermediates. Some of these will be epoxides. I don't know, I'm speculating a little bit about that. But as you can see, the other chemical workers are pesticide production workers. Okay.

Q Anything else?

A Well, again, I can go through and try to find some more, but it clearly says organic synthesizer and pesticide production worker, so those are not -- some of those I would propose would not be the kinds of petrochemicals that we're talking about in this case.

Q Anything else? Are you drawing the -- are you looking at the 2000 study that you referred to?

A No, this is the Dosemeci study. This is the Hayes -- this is the 1994, that's the other part of Exhibit number 1.

Q We'll talk about that in a minute. But you were talking about a statement made in this 2000 article; correct?

A Well, you asked me to find a -- some kind of documentation. And the Dosemeci study is the one that they talk most extensively about exposures, so I was looking at that particular paper because that's probably one of the better sources of information.

Q Let's go back to the article that you were quoting, published in 2000.

MS. KAHN: I believe it's 2001.

BY MR. HANLEY:

Q Was it 2001, sir?

A I think it's 2000.

Q I thought it was too. Why don't you read the name of the article and we'll attach it as Exhibit 2.

A Okay. Well, it's already part of Exhibit 10.

Q That's all right.

A It's Benzene and Lymphohematopoietic Malignancies in China. Hayes and -- it's published in Journal of Toxicology and Environmental Health, year 2000.

(Article entitled "Benzene and Lymphohematopoietic Malignancies in China" published in Journal of Toxicology and Environmental Health, year 2000, was marked as Plaintiff's Exhibit No. 2 for identification.)

Q Now, where in that article is there any suggestion that the other chemicals he's referring to are not in fact petrochemical hydrocarbon solvents?

A Well, all I can say is that they also refer to the -- or they list in their references the Dosemeci study. And what this is, this particular paper is, is more of a review article and discussion article. And so I think that on page 426 they refer to the Dosemeci article, the one

that I'm just referring to in 1994, and that's the article that then talks about the fact that these chemical workers were involved in doing -- included pesticide production workers and also organic synthesizers.

Q In that 2000 article marked here as Exhibit 2, is there any explicit reference anywhere in that article to chemicals other than hydrocarbon petrochemical solvents such as benzene and other such solvents?

A Well, they don't specify the particular chemicals that they are concerned about as causing -- may be involved in the excess -- excesses that were found in non-Hodgkin's lymphoma. But they -- all I can say is that they -- this article refers -- relies upon, as does the Hayes article by the way, to the Dosemeci article in order to -- in order to provide more information regarding the types of exposures for the various groups.

Q I'm going to attempt to ask a yes or no question. If you can answer it

yes or no, I would appreciate that. And if you need to explain your answer in any way, you're welcome to do that.

My question is: In the 2000 article marked as Exhibit 2, is there any explicit reference to any chemicals other than petrochemical hydrocarbon solvents such as benzene and other such solvents?

A Well, I don't see any reference to petrochemical solvents in that paper. You're trying to say that they are referencing petrochemical solvents, and I don't really see that. They are talking about benzene and other exposures that chemical workers could have. They haven't used the word, as far as I can tell in their statement, other petrochemicals.

Q Sir, my question was, yes or no, is there any explicit reference to any chemicals other than benzene, or any other hydrocarbon petrochemical solvents?

MS. KAHN: Objection. The question lacks foundation based upon the witness' answer. The question has been asked and answered to the best that the

witness can.

THE WITNESS: That wasn't your previous question. Your previous question, if I can have it read back.

BY MR. HANLEY:

Q Well, answer the question pending now, sir.

A Well, your question talked about the previous question. So, I can't really because it's predicated upon the fact that you said that you had already asked the same question, and I disagree with that.

Q Is there any reference to any chemical in the 2000 article other than benzene or any other petrochemical hydrocarbon solvents?

A All I can say is that they don't used the word and/or other petrochemical hydrocarbon solvents. I don't see those words in this paper.

Q Do you have an understanding of what petrochemical hydrocarbon solvents are?

A Yes.

Q What is it, and what do they include?

A Well, they include a wide variety of things. One of them, of course, being benzene. Another -- there are groups of alkanes which are things line propane. There are things like gasoline which is a mixture. There is -- there's just a huge variety of different kinds of gases, you know, propane gas is what people use in their stoves. There's methane, that's a hydrocarbon. I think that -- and there are aromatic hydrocarbons like benzene.

Q What references to any kinds of solvents are expressly made in the 2000 article?

A I don't see them using the word "solvent" in this 2000 article.

Q They do refer to benzene; correct?

A They refer to benzene, yes.

Q You'll agree with me that benzene is a petrochemical hydrocarbon solvent; correct?

A That's one of its uses, yes,

as an -- I mean it can be used as a solvent.

Q Do they refer to any other petrochemical hydrocarbon solvents expressly in this article?

A I don't see them referring to any other petrochemical hydrocarbon solvents.

Q Other than benzene; correct?

A That's correct.

Q And they don't refer to any other chemical or solvent other than, expressly, other than benzene; correct?

MS. KAHN: Objection. The document speaks for itself.

THE WITNESS: Well, I don't know in what context you're talking about. They talk about smoking, tobacco smoke, which of course includes lots of different hydrocarbons. They include paints, printing, which of course we know include a lot of different kind of solvents. But as you mentioned, this paper is only one of a whole series of papers that these authors have published,

and I think that they sort of have to be taken together because they don't redescribe a lot of things, they just refer to their previous publications which contain more complete descriptions.

Q Do you consider yourself an epidemiologist?

A Well, I would not consider myself in a sense the kind of epidemiologist who would do an epidemiological study. I mean I do a lot of review of epidemiological studies as a toxicologist, but I'm not an epidemiologist per se. Toxicology requires the understanding of different types of studies including human epidemiology studies, animal studies and invitro studies.

Q Have you had any formal training in epidemiology?

A Well, I have worked with epidemiologists pretty closely. I don't know how -- I mean Ernst Wyder at the American Health Foundation was a preeminent epidemiologist. I learned a lot from him. I co-published a paper with Jorgen Olsen,

head of the Danish Cancer Registry who is an epidemiologist. I mean certainly in medical school we had lectures about epidemiology and studies like that. So I mean -- as I said, I didn't specialize in epidemiology as my field but I do have some training and some -- and plenty of experience in terms of being involved in epidemiology studies.

Q What training have you had in epidemiology, formal training?

A Well, if working with one of the world's best epidemiologists side by side, and a couple of them, and actually working on epidemiology studies, I don't know, do you think that qualifies as formal training?

Q No, sir.

A Okay. Then I have worked with epidemiologists. I have been involved in epidemiological studies and I have reviewed epidemiological studies and I also teach my students in toxicology. One of my responsibilities is how to review an epidemiological study. And those are my

activities and that's the best answer I can give you.

Q Have you ever taken a class dedicated to the field of epidemiology or the study of epidemiology?

A No, I have not, because, as I said, I was -- I mean the -- none of my classes -- well, some of the medical school classes actually did involve looking at epidemiology studies, so in a sense I did. But I've never, for example, in schools of public health there are whole departments in epidemiology, and I have not participated in those kind of courses.

Q In other words, you've never taken a class wherein the class itself was dedicated in any way to the field of epidemiology or to the study -- or to the discipline of epidemiology; correct?

MS. KAHN: Objection. It's vague and ambiguous.

THE WITNESS: Well, like I said, I haven't because I've learned by doing and working with other epidemiologists as a toxicologist.

BY MR. HANLEY:

Q You do recognize that epidemiology is its own discipline with its own set of classes and course work and degrees including a doctorate degree in epidemiology; correct?

MS. KAHN: Objection. The question is overly broad and compound.

A Well, I know that as I mentioned there are epidemiology departments and there are degrees in epidemiology, yes.

Q Including doctorate degrees; correct?

A Yes. Usually they are part of a Doctorate of Public Health. But with the specialty in epidemiology, I don't know if there's actually a -- actually a Ph.D. in epidemiology, but there may be at some schools, I don't know.

Q Is it fair to say that you have more than 80 hours now invested into this case?

A I would say that's probably correct.

Q Is it your opinion that no matter what level of benzene exposure Mr. Molina suffered, and no matter what level of additional exposure to petrochemical hydrocarbon solvents he was exposed to, it would be your opinion that regardless of those levels, no matter how high they were, his non-Hodgkin's lymphoma was not caused by any such exposures?

MS. KAHN: The question is overly broad as phrased and compound.

A Well, I think I mentioned before we can only speak to what we have already -- what has already been studied. And so I'd have to qualify my answer by saying that studies have looked at certain exposures to benzene and other solvents. I believe those exposures have been quite high. So, given what you say, making the assumption, and I think there's good evidence for this based upon what the industrial hygienists have come up with, the fact that Mr. Molina was exposed to within the range of the exposures that have been studied in the scientific literature,

I would say the answer is yes, that within that range of studies that are available to us, which it's my conclusion which we've discussed previously that it doesn't show causal association between those benzene and the solvents in this case to non-Hodgkin's lymphoma. I can say that within those exposure levels what you said is just true, that his non-Hodgkin's lymphoma is not due to his chemical exposures.

Q That's not exactly the question I asked. My question specifically was whether or not that would be the case regardless of the level of exposure that he had?

A Well, as I said, I can only speak in terms of what we know, in terms of what we have available to us in the scientific literature. If indeed it could be documented that he was exposed to levels way above those that have ever been studied in the scientific literature, I would say that that's then an unknown. But I believe that his exposure, as they have been

estimated, fall well within what we have seen in the scientific literature. And I don't believe that his -- and that would say that his non-Hodgkin's lymphoma is not related to the exposures to those chemicals.

Q Is there any level of exposure to benzene or other petrochemical hydrocarbon solvents that a person could be exposed to that would increase their risk for development of non-Hodgkin's lymphoma?

A Not that I'm aware of. As I said, and again, that's based upon what is available in the scientific literature.

Q Are you saying that if someone has high enough level of exposure to benzene, even higher than what's been reported in scientific literature, and they had a non-Hodgkin's lymphoma, you would not know whether or not it was caused by that exposure?

A Well, I would say the best information is still that it's not caused by that exposure. However, I would say that it would be preferable to have studies

that match the exposures that we're talking about so that we can be sure that it wasn't caused.

Q Do you have an opinion whether or not benzene, in the abstract, is, in high enough dosages, is capable of causing non-Hodgkin's lymphoma or not?

A All I can say is I don't have any indication as far as I can see from the scientific literature that would indicate a causal relationship between benzene exposure and non-Hodgkin's lymphoma.

Q Are you able to categorically rule it out?

A Well, I think that calls for speculation.

Q So is the answer you are not able to categorically rule it out?

MS. KAHN: Objection. Misstates the witness' testimony.

THE WITNESS: I'm saying that I wouldn't speculate on something that I can't rely upon the scientific and medical literature for.

Q Is that to say that you cannot

categorically rule it out?

A That's the best answer I can give you.

Q Would you agree that we can categorically rule out the notion that drinking water in whatever amount is not capable of causing non-Hodgkin's lymphoma? Would you agree that as a scientist and a medical doctor and based upon your understanding of what we know about the mechanism of cancer and the affect of drinking water on the human body that regardless of whatever amount in one's life, or acutely or chronically, that you would feel comfortable categorically ruling out the notion that drinking water can cause non-Hodgkin's lymphoma?

A Well, I think it would be difficult to study because if you're talking about --

Q Sir, I'm not asking you about how you might determine that. I'm asking whether you believe you can categorically rule it out or not. If you think you can't categorically rule it out, then that may be

your answer, or do you believe that, yes, as a scientist we can categorically rule that out?

MS. KAHN: Dean, I think you interrupted the witness' answer.

MR. HANLEY: That's because I'm trying to get an answer to the question I actually asked.

MS. KAHN: Dr. Whysner, do you need to have the question read back?

THE WITNESS: No, I don't know what question is on the table right now because there were --

MR. HANLEY: I'll just ask the court reporter to read it back to you.

(The requested portion was read.)

MS. KAHN: The question is compound and overly broad.

THE WITNESS: Well, first -- okay. What I can say is I really haven't studied water and non-Hodgkin's lymphoma, and I don't know that anybody has. And I guess one can hypothesize that if you drank a whole bunch of water and since

people can get a lymphoma of their digestive tract called a MALT type lymphoma, could the irritation of drinking a lot of water over an extended period of time disrupt the lymphoid system in that area to a point where one could get a non-Hodgkin's lymphoma in that area? I think it's unlikely, but could I categorically rule that out, if somebody was indeed stressing their body in such a way that for an extended period of time, I'm not sure I can rule that out categorically.

BY MR. HANLEY:

Q Is there any degree to which you could or could not rule out benzene as a cause of non-Hodgkin's lymphoma in comparison to something like water?

A In what way in comparison to water? I mean I've given you an answer for benzene and I've given you an answer for water.

Q Yes. And my question is: Is it any closer of a call or any more difficult to categorically rule out one

over the other given what we know about the biological plausibility, for example?

MS. KAHN: Objection. It's compound, it's overbroad, it's vague and ambiguous.

THE WITNESS: I would be speculating if I had to answer that question.

BY MR. HANLEY:

Q What do you believe is the cause of Mr. Molina's non-Hodgkin's lymphoma?

A Well, I don't think we really know what the cause is of his non-Hodgkin's lymphoma.

Q Do you consider it to be idiopathic?

A Yes. And that's a medical term that's commonly used when we don't know what is the cause of a person's particular disease.

Q Do you believe that there is a cause of his cancer but it's unknown or that his cancer has in fact no cause?

MS. KAHN: Objection. Vague

and ambiguous, compound.

THE WITNESS: I don't think we really -- I don't think we really have a way of distinguishing between those two possibilities.

BY MR. HANLEY:

Q Do you believe that his cancer was spontaneous and had no cause at all, it just spontaneously developed in him for no particular reason, or do you believe that there more likely was some cause but we don't know what that is?

MS. KAHN: Objection. The question is overly broad, compound, and as to the word, "spontaneous," it's vague and ambiguous.

THE WITNESS: I think the -- obviously when somebody gets a disease there has to be some kind of disruption in the cell.

Now, whether or not -- I mean, if you want to use the term, "spontaneous," for example, if a cell is dividing and there's an error in DNA replication that takes place because

statistically there can be errors in DNA replication, and I guess you would call that a spontaneous error. I think that it's possible that people's diseases are caused by those kinds of spontaneous events, and, therefore, it's possible that Mr. Molina's non-Hodgkin's lymphoma could be caused by such a -- what you could roughly in layman's terms be called a spontaneous event even though it does result in a specific genetic alteration, but the reason for it happening is that every once in a while when there's a replication of DNA, things don't get replicated exactly properly. And that's not because of any interference with any other environmental factor, it's just a statistical probability.

Q Do you believe that can happen in a case of development of non-Hodgkin's lymphoma in the absence of any toxic exposure or environmental exposure or some outside environmental exposure to the cell that caused that genetic alteration?

MS. KAHN: Objection. The

question is overly broad, compound, vague and uncertain.

THE WITNESS: As I mentioned -- could you read back the question again. I am lost in the train of thought there.

(The requested portion was read.)

THE WITNESS: Well, I think that it can, because, like I said, there are these errors in replication, and, you know, again, it all depends on what you mean by environmental or toxic exposure.

For example, oxygen, which is something that is necessary for life, is actually a toxic substance and there's a lot of oxidative damage of DNA that goes on on a regular basis.

Now, would you call that spontaneous? I guess it depends what you mean by spontaneous. But it's been estimated that there are thousands of these events per cell per day that takes place just because we have to breathe oxygen and oxygen happens to be a very reactive chemical and it can damage DNA.

So there are all kinds of events that can take place that don't have to do with what we normally think of as some kind of an environmental exposure because there are things in the environment like oxygen which can also produce these effects.

BY MR. HANLEY:

Q Would you agree that oxygen has never been associated with any increased risk of any form of cancer?

A Well, I think that it's actually -- oxidative damage is considered in the scientific community to be one of the events that can lead to cancer in general. I mean, there's a lot of interest in antioxidants to prevent this kind of damage in order to prevent cancer. So I think that -- I think the answer is yes, I think that there is a -- there has been postulated, widely as a matter of fact, within the cancer prevention community the fact that people believe that oxygen can lead to cancer.

Q Can you point me to any

scientific or medical study that concluded that oxygen is a risk factor for developing any form of cancer?

A Well, it's -- I mean -- I think there are lots of articles in the scientific literature. I don't know that I can point you to any one right now.

When I was at the American Health Foundation we did a whole two-day meeting on the issue of antioxidants and the prevention of cancer predicated on the knowledge that oxidative damage can lead to cancer. Now, in our -- our certainty about this is based upon some -- I have to admit some circumstantial evidence. But I think the -- I think it's widely accepted in the medical and scientific community that oxidative DNA damage is potentially a cancer risk.

I didn't bring that literature with me, but it certainly is out there.

Q Exposure to oxygen in what context? Breathing?

A Yes; that's correct. Breathing every day. I mean, oxygen is an,

unfortunately, a very reactive compound and we -- it is what produces energy because it has these high energy bonds in it, but because of that it is also -- it's technically a free radical and it can produce damage.

Q And part of the human body's ability to not succumb to cancer every time there's such errors in replication due to an exposure to whatever, whether it be oxygen or asbestos in the ambient air or any other carcinogen that we're exposed to, the body has a way of dealing with that; and, that is, cells, when they get that genetic damage, more often than not if not all of the time in someone who makes it through life without developing cancer, part of the immune system is that cell dies; correct?

MS. KAHN: Objection. The question is overly broad and vague.

THE WITNESS: Well, cells that die don't cause cancer.

BY MR. HANLEY:

Q Right. That's why we don't

get cancer even though we have genetic damage that occurred to various cells in our body all of time; correct? The cells get a genetic damage, replicate or attempt to replicate, and because of that genetic damage, have a programmed cell death and -- by ap --

A Apoptosis.

Q -- apoptosis, die; correct?

A Well, yes, but that doesn't have a lot to do with the immune system. That has to do with the cell's program, self-program. And, actually, the first line in defense is DNA repair which is not an immune-mediated mechanism. It's, the body has -- and that's the reason that we don't succumb to cancer with all of these oxidative DNA damage events is because there are specific repair mechanisms that are operative that are working to repair that damage, and also there are antioxidants in the body which are -- try to prevent the oxidative damage from taking place in the first place.

Q And when someone develops a

cancer that is in fact due to an environmental exposure, whether it be a mesothelioma from asbestos or a lung cancer from smoking or an AML from benzene exposure, it is because the body has not been able to employ those normal defenses that you're describing and the cell takes that hit from exposure to the toxin, has genetic alteration, has errors in replication, and that is part of the process that ultimately develops into a cancer, and those are individuals who get such a cancer from environmental exposure; correct?

MS. KAHN: Objection. The question is overly broad and compound.

THE WITNESS: It was an -- can you make it a little bit -- can we go through that step by step in terms of what you're talking about? That was a lot of material you just covered.

BY MR. HANLEY:

Q Okay. Well, if we need to read back the question, we will. I would ask you to describe the step by step

process by which these type of cancers develop in people at a cellular level and a molecular level in the terms that we are discussing?

MS. KAHN: I'm going to object to that question to the extent that it exceeds the scope of the witness' retention and designation in this case. Lung cancer and mesothelioma are not issues that we asked Dr. Whysner to look into and are not topics about which we expect him to offer opinions at trial.

BY MR. HANLEY:

Q We're talking about general carcinogenesis. Do you understand that, sir?

A Well, yes, but I'd like to -- if I can have your question read back bit by bit so I can comment on it. To be quite honest, by the time I got to the end I couldn't remember the beginning of it.

MR. HANLEY: Okay. I'll ask the reporter to read it back.

(Record read as requested.)

THE WITNESS: So you said,

what I had been talking about. I was talking about oxidative damage. Or at least that's the way I interpret your question.

BY MR. HANLEY:

Q Well, sir, I would ask you to interpret my question in terms of what I asked which was in an individual who develops a cancer due to a particular type of environmental exposure, and I gave you three examples that I trust you would agree with, of examples where that can happen in people.

MS. KAHN: Objection. The question is overly broad, compound, vague, uncertain, argumentative and asks for opinions outside the scope of this witness' retention and expert witness designation.

THE WITNESS: Well, you mentioned those three examples, and I really -- I'm not very familiar with mesothelioma in terms of what the current thinking is the way in which cancer is produced.

And lung cancer from smoking is a huge topic. I don't think we know what the agents are within it. I mean there are so many different agents, and one of things that the American Health Foundation that I used to sit through was seminar after seminar after seminar of people trying to figure out what indeed it was within the cigarette smoke that was causing the lung cancer. I mean there's, you know, there's some good theories. There's a lot of work out there. But it's a very complicated chemical mixture, and I wouldn't want to speculate on trying to figure out what, how cigarette smoking causes lung cancer.

I have, in terms of AML, you know I have written on this, it's in the published literature. We talked about that. I have a paper out where I think that the -- my best evaluation of the scientific literature has to do with this inhibition of Topoisomerase 2, and I think that that causes certain genetic alterations which lead to acute

myelogenous leukemia. And those genetic alterations could be repaired, and sometimes they are not and sometimes they are aren't. And when they are not repaired, then that can lead to the development of acute myelogenous leukemia.

BY MR. HANLEY:

Q And it takes more than one hit from the toxin getting a single or whatever amount of genetic damage that gets replicated, but it takes more than one exposure for the cancer to develop, correct, in AML?

A Well, there's certainly a number of different genetic alterations that take place during the development of AML. I guess the presumption would be that it would require several events to take place, as well as perhaps other kinds of factors such as toxicity to the bone marrow for that to happen.

Like I say, nobody in their -- there are several theories. One of them, by the way, being oxidative damage which

has been proposed in terms of the development of AML. But without specifically knowing exactly how AML is produced, it would be difficult to provide you with an answer.

Q One of the things we know is that enough exposure to benzene over time can cause the particular cancer of the blood forming organs, AML; correct?

A If -- it can. If we know this, that people who have been exposed to large amounts of benzene have developed -- have a -- have developed AML because we know that there is a greater incidence of AML among people who are exposed to high levels of benzene.

Q And one of the reasons that it takes exposure to certain levels of benzene over time is because the cancer doesn't develop from a single exposure causing a particular genetic error in a cell that replicates; correct?

A Could I ask you just one quick question? Are we going to break pretty soon, because we have been going --

Q That's fine. If we can finish this particular area of inquiry, then I'd be happy to take a break.

A Okay.

Q So do you have my last question in mind?

A Could you read me the question, please?

MR. HANLEY: I ask the reporter to read it back.

(Record read as requested.)

THE WITNESS: I guess what I'm having a little trouble with is the last part, "that replicates." The error that a single -- would you explain or --

BY MR. HANLEY:

Q Sir, let's see if we can get on the same page. What I'm getting at is, in basic general terms, that when benzene causes an AML, it's because benzene gets to the target organ and causes genetic damage in a cell. That cell and -- that cell replicates, as in the normal course as it would, and now there are two cells with this genetic damage. In our understanding

of carcinogenesis, generally, we know that this happens, and in fact perhaps happens all the time. Maybe even every day in the human body. And very often, most of the time perhaps, or virtually all of the time perhaps in most of us if we're lucky, every time that happens those cells with the errors either repair -- repair themselves as you described, or the human body is able to repair them, or the cells die and go through this apoptosis process by which they program themselves to die. But sometimes, and in those who develop a cancer, those cells with that genetic damage or one of them does not die and continues on with that genetic damage and then continues to replicate with that particular genetic error, that itself does not mean that the person gets cancer but that particular cell with that genetic error then has another exposure to the toxin and then has further genetic error and further genetic damage, replicates, perhaps then they die, but maybe one of those cells do not die and then further has

a further exposure to the toxin like benzene, and then now has more, yet more genetic damage. And over a series of time, through a series of hits on these cells with genetic errors, and then they get increasing genetic error. And we don't know how many hits it takes or over what period of time it will take to develop, but in some people they get enough genetic errors replicated that finally this gets to a -- becomes a cell that is cancerous and has it's own uncontrolled cell growth and replication. Is that -- is that consistent with your understanding of some of our understanding of the basic mechanisms of carcinogenesis including for AML?

MS. KAHN: Objection. The question is overly broad, vague and uncertain, and it's an incomplete hypothetical.

THE WITNESS: Well, I disagree with your characterization as in certain ways. First of all, you mentioned benzene getting to the target organ, and I think we had a fairly

extensive discussion the other day that it is not benzene itself it's presumed. Benzene has to be metabolized in the liver by cytosome P450 to form certain hydroxylated metabolites. So that has to take place first, and that's, of course, going to depend upon a person's particular metabolism and so forth.

BY MR. HANLEY:

Q So benzene itself is not the toxin. It's the by-product from the liver that ends up being the toxin, but with that correction in mind, if could you then continue to answer my question.

MS. KAHN: Same objection.

THE WITNESS: Well, you know, as I said before, I think that there -- we don't really know exactly how benzene metabolites produce AML. And it would be -- it was kind of a -- your description, there was some parts of it that were -- but I mean I'd have to go through that description bit by bit to, you know, to know whether or not -- I mean, as we said, we haven't really

nailed down all of the components of the way in which benzene metabolites produce AML, and so it would be difficult for me to agree with all of that that you just said because I'm -- because in the specific case here of benzene production of AML, I think that we just don't have enough information.

BY MR. HANLEY:

Q I'm just trying to see if we can agree on the basic mechanisms, the very basic mechanism of carcinogenesis. And is not our general understanding of carcinogenesis that the toxin causing the cancer gets to a cell, causes genetic damage, it replicates, and through a series of continued genetic damage and replication of that cell over a period of time with some number, but multiple numbers, whether it's 10 or 50 or 100, that that process over time through repeated toxic exposures that leads to the development of a cancer?

MS. KAHN: Objection. The question is overly broad and compound.

BY MR. HANLEY:

Q Would you agree with that general proposition about how cancer develops?

MS. KAHN: Objection.

THE WITNESS: Well, I think it ignores the, also the epigenetic events that have to take place. You know, it's not -- and apparently at least for a lot of cancers -- and I'm not sure whether or not this applies to AML or not, but it probably does.

For a lot of cancers you also have to have an environment and the target organ that is also conducive to the development of the cancer. You know, sometimes it's a frank, you know, toxicity that takes place. So it's not necessarily just the chemical producing or the chemical's metabolites producing any kind of genetic alteration. There may also be some elements of toxicity involved which alter the environment, the general cellular environment. So it's a very complicated issue and to try to sort of take a generalized scheme and apply it

to any particular cancer is an -- I think is going to be very speculative.

BY MR. HANLEY:

Q What do you mean by "epigenetic event"?

A Epigenetic events are those that impact general expression. In other words, the genetic material is producing through the -- through messenger RNA and through protein production is -- the gene express themselves. But other things -- and genetic events can alter gene expression, but you can also alter gene expression through the interaction with hormones and other -- and chemicals. For example, estrogens.

In general, estrogens are not thought to produce genetic damage, yet it's widely believed that they are responsibile for increased risk of breast cancer, but they do that through, presumably, some kind of epigenetic mechanism where they actually alter the gene expression rather than altering the genes themselves.

Q Are you familiar with the

multi-hit theory of carcinogenesis --

MS. KAHN: Objection.

Q -- in the context of environmentally caused cancers?

MS. KAHN: Objection. Overly broad, vague and ambiguous.

THE WITNESS: And I think maybe I even mentioned this last time, that used to be thought to be -- the multi-hit theory came out, I think, in the 1950s, 1960s when we first began to understand that some chemicals could produce mutations, and, of course, that was basis of the Aims Test and so forth. But, I mean, our understanding of the process of cancer production, I think, has gotten a lot more sophisticated than the -- we've gone beyond -- used to be thought that that's all it required, just several genetic events that the chemical caused and that was the multi-hit process. But I think that -- I think that there are a lot of other things involved, like I mentioned all the epigenetic events that can take place.

Can we take a break?

MR. HANLEY: Yes.

(At this time a brief recess was held.)

BY MR. HANLEY:

Q Sir, the 1994 Dosemeci article that is attached as part of Exhibit 1, you have that in mind?

A Yes.

Q Isn't it true that the exposures that they were assessing and describing and studying in particular were the benzene exposures of those workers?

A That's what they were quantifying.

Q And that was in fact the focus of their study and the epidemiologic results that they were attempting to find was relating to these workers' benzene exposure; correct?

A That was the primary purpose, yes.

Q The 1997 Hayes article, benzene and dose related incidents of hematologic neoplasms in China, do you have that report there with you?

A Yes.

Q That study?

If you look at the abstract, and in the Results section of the abstract which begins at the bottom of the first page of the article in the abstract and the bottom of the fist column. Do you see where it says "Results: For workers historically exposed to benzene at levels -- at average levels of less than 10 parts per million, the relative risk for all hematologic neoplasms combined was 2.2." Do you see that?

A Yes, I see.

Q Do you agree with that statement?

A Well, in the sense that it -- I mean they are, in this particular statement, they are lumping together all of these different neoplasms which would include the acute myelogenous leukemia, so you can't really say what it's due to. And also I want to point out that this 10 parts per million is not 10 parts per million years. This is the actual exposure level.

And with those explanations, yes, I agree with what the statement, and that's what they found.

Q If you go down to -- skip through a couple of sentences, further on in that same abstract, now, about a third of the way down in the second column on the first page, do you see where it says, "Workers with 10 or more years of benzene exposure had a relative risk of developing non-Hodgkin's lymphoma of 4.2."?

A As I mentioned before, in terms of all their industries, that's what they found when combining all of these industries. And as I mentioned before, they later in the year 2000 were concerned that some of this might be due to chemical exposures other than benzene.

Q Do you agree with the statement that I read?

A Well, with the understanding that they weren't -- these people were exposed to more than just benzene. They found, among all of those workers, an increase relative risk for -- I mean, I

would say there's -- there's benzene exposure plus exposure to other chemicals.

Q Do you agree with the statement as stated?

A No, because --

MS. KAHN: Objection. Asked and answered.

THE WITNESS: As I said, I believe that they -- because it's an abstract statement, they didn't mention all of the other exposures that these workers had. So if I were writing this, I would say, Benzene with 10 or more years of benzene -- exposure to benzene and other chemicals involved in their industry had a relative risk of developing non-Hodgkin's lymphoma; and, which is what I mentioned that they later pointed out in their 2000 publication.

Q Are you prepared to point to anything other than that one sentence you read for me in that 2000 exposure for your argument here that they raised concerns about chemicals other than benzene or other petrochemical hydrocarbon solvents?

A Well, I think if you look at table one in this paper which is on the next page -- two pages.

Q Please refer to which paper.

A Hayes, 1997 paper. They list the relative risks for NHL -- sorry.

(Interruption.)

THE WITNESS: Sorry. They are delivering lunch. We can wait for a while.

Can we go off the record for just a second, I may have to do something here.

(Discussion was held off the record.)

THE WITNESS: The risk, the relative risk for NHL in the various industries, and so I would again say that the -- what they were referring to in their 2000 paper is actually going back to this where you see that for the chemical workers, the relative risks are much higher, twice as high just about, than any of the other industries. And for some of the industries with benzene exposure the relative risks are 1.6, 1.6.

And, actually, in the rubber industry, even though it's a 4.0, it's only involving one case of non-Hodgkin's lymphoma. So I -- so this would indicate to me that there's a wide variation depending upon the particular industry with the highest being in the people who are in the chemical industry which, as we discussed with this Dosemeci article, the people in the chemical industry were involved in pesticide production and they -- also organic synthesis reactions.

BY MR. HANLEY:

Q But my question was whether there was any statement, other than the one sentence you read to me from the 2000 article which you believe supports your argument that the chemicals at issue are being referred to are anything other than benzene or other petrochemical hydrocarbon solvents?

A Other than my interpretation of what they are referring to in the -- I mean, I mean, table one is not exactly a statement, but it is an indication of the

underlying data that has to do with that statement in their 2000 paper.

Q So the answer is no, other than that sentence and your interpretation of the table, there is no other statement that you would refer to?

A Well, I believe that there were some other statements, and I can't find them right now, that talked about other exposures of the people in other industries, but I can't really locate it right now.

So sitting right here, as I said, I think in -- I'm not sure whether it was the Hayes paper or whether it was the Dosemeci paper or whether it was a Travis paper or whether it was one of the other papers in this whole series where they discuss this issue. I believe that there was another statement about the -- the different exposure, but I can't locate it right now sitting here.

Q And you'll agree that the only chemical that is focused upon, measured and analyzed in terms of its potential

causative association with any of these cancers of the blood forming organs in all of these studies from this research of the Chinese workers is benzene?

A Well, that is the one that they quantify in these studies, and, as you mentioned, the focus of this was to determine the associations between quantified benzene exposure and non-Hodgkin's lymphoma, but obviously there were other chemicals that these people were exposed to.

Q Is that a way of saying yes, with that explanation?

A It's my way of saying what I did say, which was that this is the quantified benzene exposure, but they did not quantify the exposure to other chemicals and --

Q Let me ask my original question be read back, and I ask you, sir, for a yes or no with whatever explanation you feel necessary.

(Record read as requested.)

THE WITNESS: That is what

they focused on in these papers.

BY MR. HANLEY:

Q Is that a way of saying yes?

A Yes.

Q Thank you.

Now, going back to the 1997 Hayes article. If we continue where I last left off, that sentence continues to say, "And the development of this neoplasm was linked most strongly to exposure that had occurred at least ten years before diagnosis, i.e, distant exposure."

Do you agree with that statement?

A Well, there's a little more -- I mean, the relative risk for five to nine years in table one was 3.3, and a greater than 10 was 4.2. So there is a slightly greater risk for greater than 10 compared to less than 10, but it's not great. It's a difference between 3.3 and 4.2.

Q Is that a way of saying yes with that explanation?

A I think that's what I said.

Q So you agree with that

statement with that explanation?

A With that explanation.

Q All right. Now, if we go down a little further to the third to the last sentence of the abstract, right after where they state "Conclusions:"

A Um-hum.

Q They state, "The results of this study suggests that benzene exposure is associated with a spectrum of hematological neoplasms and related disorders in humans."

Do you agree with that statement?

A I think "suggest" is the right word. Yes, I agree with that. I don't think that they concluded that, or it's a -- it's a -- something that one can say there's a causal relationship, but there's a suggestion from this study.

Q The next sentences says, "Risks for these conditions are elevated at average benzene exposure levels of less than 10 ppm and show a tendency, although not a strong one, to rise with increasing

levels of exposure."

Do you agree with that statement?

A Again, with -- yes, I do, and with the understanding that we're not talking about ppm years, but we're talking about 10 ppm exposures which they have characterized in the paper of constant ppm. In other words, when people have been exposed to 10 ppm constantly for a given period of time. And that the dose response tendency, I agree, is not very much.

Q So you agree with that statement then?

A I think that's what I just said. That with those -- with that explanation, that I want to make sure we understand that we're talking about parts per million. Ten parts per million, of course, is ten times the current TOV in the United States.

Q And when they refer to the risk for, quote, unquote, these conditions, the conditions they are referring to are the spectrum of hematologic neoplasms and

related disorders in humans. In other words, cancers of the blood forming organs including various lymphomas and leukemias; correct?

MS. KAHN: Objection. Calls for speculation as to what they refer to and it's overly broad and compound.

THE WITNESS: Yes, I don't know that I can really -- I mean, from the abstract it's not really clear exactly what they are referring to.

BY MR. HANLEY:

Q What do you believe that they are referring to in the statement that, The results of this study suggests that benzene exposure is associated with a spectrum of hematologic neoplasms and related disorders in humans. What is the spectrum of hematologic neoplasms and related disorders in humans that they are referring to, sir?

A I don't know what "spectrum" means. A variety, but they don't specify which ones particularly, so I really couldn't say.

Q In the context of this study

and in the context of this article in particular, does that spectrum that they refer to not include AML, NHL and other cancers of the blood forming organs?

A Well --

MS. KAHN: Objection. It's overly broad, vague and ambiguous.

MR. HANLEY: No, it's not. Go ahead, Doctor.

THE WITNESS: I don't exactly know what particularly they are talking about with this spectrum. You know, I would -- I would be reading something into the paper here in their conclusions, but, you know, that's the best answer I can give you.

BY MR. HANLEY:

Q So your answer is you don't know what spectrum of hematologic neoplasms and related disorders in humans that they are referring to?

A I don't know -- for example, I don't know what they mean by "related disorders."

Q And reading this -- you've

read this entire study, I take it, many times?

A I would say I've read it. You know, recently I've read it over once and I read it a couple of times, several times in the past, but I don't know what they mean by "related disorders" for example, so it makes me a little bit curious as to what they mean by, "hematologic neoplasms and related disorders."

Q So reading this study as many times as you read it, you don't know what they are referring to when they refer to in their abstract as a spectrum of hematologic neoplasms?

MS. KAHN: Objection. Asked and answered, argumentative.

THE WITNESS: I think I've -- I think I have answered as best as I can.

BY MR. HANLEY:

Q Well, you made specific reference to the related disorders and described your nonunderstanding of what they mean by that; is that right?

A Well, if you refer me to

something where they define that, I would be happy to comment on that.

Q I'm asking for your opinion based on having read the article, sir.

A Which I've given to you.

Q Do you know what they mean when they refer to a spectrum of hematologic neoplasms?

MS. KAHN: Objection. Asked and answered.

THE WITNESS: As I said, I would be speculating if I tried to interpret what they meant by the spectrum of hematological neoplasms in this particular sentence.

BY MR. HANLEY:

Q Well, what hematologic neoplasms do the results of this study suggest that benzene exposure is associated with?

A Well, I mentioned to you the fact that I don't think it's clear, for example, that it includes NHL based upon what they have written in the year 2000 and what I see in the table regarding the

various industries and how the relative risks for NHL are so inconsistent among the various industries.

Q You would agree that their reference to a spectrum of hematologic neoplasms includes AML?

MS. KAHN: Objection. Calls for speculation. The article speaks for itself.

THE WITNESS: I can't give you a better answer than I already have. I don't know what they mean by spectrum. If they had said what that spectrum is, that would be one thing. I would presume that it includes AML, but the term is -- the wording that they are using is kind of vague.

BY MR. HANLEY:

Q Would you presume that it includes anything else?

A I don't know.

Q Would you agree that it necessarily infers more than one cancer of the blood forming organs?

MS. KAHN: Objection. Vague

and ambiguous.

THE WITNESS: Meaning in terms of suggesting something?

BY MR. HANLEY:

Q Yes, sir. In terms of what they said.

A Well, I don't know because, you know, for example, they have on table two, again, they have NHL, they have leukemia, and then they have ANLL and they have ANLL/MDS and then they have other leukemias. And I don't know exactly what they are referring to in terms of that analysis because they've obviously referred to all leukemias, but then they have referred to ANLL, and then they have referred to ANLL/MDS, so it's kind of difficult for me to figure out exactly what they are talking about here.

Q And you don't think that the spectrum of hematologic neoplasms that they refer to include all of those?

A I don't know.

MR. HANLEY: All right. I have a few more questions about this

article that I want to ask you, if you don't mind, before lunch unless you really feel like you want to take lunch right now, then we can.

THE WITNESS: No, we can go on.

BY MR. HANLEY:

Q If you go to the discussion section of the article itself on page 1068. Now, if you look at the first sentence of that discussion section where it says, quote, "In earlier reports we showed that employment in benzene exposed jobs in China, regardless of the exposure level, was linked to hematologic disease mortality and the incidence of leukemia, NHL, MDS and aplastic anemia" unquote.

Do you agree with that statement, sir?

A Well, I don't know that I have their earlier reports with me, so I don't know if I can agree with that statement.

Q You see that it refers to two studies specifically; correct?

A Refers to two.

MS. KAHN: The reference to the two earlier reports are footnotes 22 and 23.

MR. HANLEY: You know, I'm not asking you to testify, Counsel.

MS. KAHN: Just, the witness doesn't know if he has them with him. It sounded like he wanted to look at them to answer the question. So I just wanted to be helpful and tell him what they are so he can see if they are in his materials or not. He may not be able to answer your question without looking at them.

MR. HANLEY: I would certainly think if he was looking at the study he would see the footnotes for himself, Coach.

MS. KAHN: Just trying to be helpful.

BY MR. HANLEY:

Q And my question was, sir, you see the two studies to which he's referring?

A Yes, I know the question. I'm looking for the studies.

Well, I have one of them but I don't have the other one.

Q Have you read both of those studies in the past?

A I can't recall. And with the Hayes article, Environmental Health Perspectives often republishes the same thing that is in another journal. That's my recollection. And to be quite honest, I can't tell you if the reason I don't have it with me is because it's duplicative of this other paper or not, but I can't really tell you right now.

Q The two references they make to footnotes 22 and 23 in the first line of the Discussions section, in the first sentence of the Discussion section, do you know whether or not you have read either or both of those studies?

A Well, I have one of the studies with me. I have the Yen paper with me.

Q The 22 or 23?

A That's 22.

Q I take it you have read that

study?

A Yes.

Q Have you read it recently?

A No, because I focused -- I think I focused on the Hayes because the Hayes '97 and the 2000, I think, were reporting the same study. And they were more recent publications so I focused on those.

Q And the study referred to by footnote 23, is that a study that you have read?

A I believe so.

Q Do you agree that in those two reports that the investigator showed that employment in benzene exposed jobs in China, regardless of the exposure level, was linked to hematologic disease mortality and the incidence of leukemia, NHL, MDS and aplastic anemia?

MS. KAHN: Objection. Overly broad, compound, calls for speculation based upon the witness' testimony, and those documents which are not before us speak for themselves.

THE WITNESS: What I see is they reported the same thing that the Hayes -- I believe they reported the same thing that the Hayes 1980 -- I mean, excuse me, '97 article reported. I don't see any difference except they didn't need -- the non-Hodgkin's lymphoma that they reported here -- well, there's a discrepancy. There's non-Hodgkin's lymphoma data that they reported here they found a relative risk of 3.0 with a confidence limit that it included one which is probably -- wouldn't make it statistically significant. And the same -- for some reason they dropped one of cases of non-Hodgkin's lymphoma, apparently in the '97 report, because there are only 16 cases of NHL versus the 17. So, I don't -- I guess I don't understand -- and then the result is not statistic -- clearly not statistically significant in their '97 paper. So there's something different going on. They apparently must have decided that one of these cases of NHL and the exposed

workers wasn't part -- shouldn't be part of the cohort.

BY MR. HANLEY:

Q Sir, you haven't answered my question. My question was: Do you agree that those two reports -- that in those two reports they showed that employment in benzene exposed jobs in China, regardless of the exposure level, was linked to hematologic disease mortality and the incidence of leukemia, NHL, MDS, and aplastic anemia?

MS. KAHN: Objection. Calls for speculation, overly broad, compound, argumentative, asked and answered. Those documents speak for themselves.

THE WITNESS: Well, this is an introductory statement of about what they previously found. But as I said, this is -- this is reporting what this earlier paper -- well, I don't even know if it reports what the earlier paper found because the confidence limit includes one which is usually -- it has to be above one in order to be considered

statistically significant. And in the '87 article they dropped, apparently, one of the cases, and then it's no longer statistically significant. So maybe what they are saying, we showed this but it's no longer true anymore. I don't know. I would have to -- I wouldn't want to take that statement out of context.

BY MR. HANLEY:

Q Sir, are you saying that no, you do not agree with that statement?

A I don't agree with that statement in the sense that if the earlier -- well, I agree that the earlier report reported that, but the current report that they are talking about here did not report that. Because whereas -- well, it didn't even report that. Because, like I said, the relative risk includes 1.0, and I don't consider that to be a statistically significant finding, especially given the fact that when they apparently -- this later report of this study doesn't even have one of those cases, and now the lower confidence limit is -- now I lost the page

here.

Q Sir, I haven't asked for your analysis, so you're not answering my question.

A I think I did answer.

Q I simply asked you the question of whether or not you agree with these authors that the two studies that they cite show what they say they showed? Do you agree or disagree with these authors when they say that these two articles footnoted and referenced as 22 and 23 showed that employment in benzene exposed jobs in China, regardless of the exposure level, was linked to hematologic mortality and the incidence of leukemia, non-Hodgkin's lymphoma, MDS and aplastic anemia, do you agree with that statement or do you disagree with that statement?

MS. KAHN: Objection. Asked and answered.

MR. HANLEY: It's been asked. It hasn't been answered.

THE WITNESS: I guess, based upon what I have just said, I would have

to disagree with that statement because I don't believe that it shows a statistically significant result, and in their update they have dropped one of the cases which made it even less of a statistically significant result.

BY MR. HANLEY:

Q Thank you for answering my question.

That only took about 15 minutes.

The next sentence in the Discussion section says, quote, "This investigation provides evidence that benzene may cause hematologic neoplasms and related disorders at average exposures of less than 10 ppm and accumulative exposure of less than 40 ppm years."

Do you agree or disagree with that statement?

A I think the operative word here is "may." And if you look at the -- I agree -- well, let me just say that the operative word here is -- let me look at the data here for a second.

ANLL less than 40, there is an increased relative risk but the lower confidence interval is .5. So it may, but as I say it may not because this is not a statistically significant result.

Q So you would agree then with that statement as it's phrased?

MS. KAHN: Objection. Asked and answered.

THE WITNESS: Well, as it's phrased that it may, I agree the statement says that it may. But I want to point out that it's not a statistically significant result, and I want to emphasize the word "may."

BY MR. HANLEY:

Q Is that to say then that you cannot rule out that benzene causes some number, more than one, of hematologic neoplasms and related disorders at the levels that they describe?

MS. KAHN: Objection. Compound, over broad, vague and ambiguous.

THE WITNESS: Well, I don't

think you can ever prove a negative. Is that what you're trying to say?

BY MR. HANLEY:

Q Yes. I'm asking, you agree that you can't rule out that benzene causes some multiple number of hematologic neoplasms at average exposures less than 10 ppm and cumulative exposures of less than 40 ppm years?

MS. KAHN: Objection. Over broad, compound, vague and ambiguous.

THE WITNESS: Based upon this data, I don't think there's any indication one way or the other.

BY MR. HANLEY:

Q So you agree that it cannot be ruled out?

A Well, I think it's --

MS. KAHN: Objection. Argumentative, overbroad.

THE WITNESS: I think that I would have to interpret it that you can't really say. Based upon these numbers, there's not a statistically significant result and it doesn't really provide you

any information to say one way or the other.

BY MR. HANLEY:

Q If you go on a little further in that Discussion section to the next column and look at the first full sentence in the last column, do you see where it says, quote, "Risk for NHL increased with duration of exposure and was strongest among workers who had distant greater than or equal to ten years exposure" unquote.

Do you see where it says that?

A Yes, the first sentence, okay.

Q Do you agree with that statement?

MS. KAHN: Objection as speculative as to what the statement means.

(Witness perusing document.)

A Well, I have to say that the relative risks in this -- described in this program, I'm not exactly sure what they mean by risks, because risks sometimes has a -- and I know in California law I've heard it has a particular meaning. So I

wouldn't -- want to make sure that we're not talking about any kind of legal definition of risk.

What they found was that the relative risk was greater -- well, I think I mentioned this before, I don't see a big difference between the 4.2 greater than 10 and the 3.3 for five to nine years. There is a small increase, but it's not a great difference between those two for NHL.

Q Do you agree or disagree with that sentence?

A As I said, I don't agree with the way that they use the word "risk," because I think what they are talking about the relative risks that they have found in this study, I don't think that that implies that there is a true risk. But I think that this is a -- I mean I think it's just -- I think it's just not worded very well because there might be some ambiguity in the way one would interpret the word "risk."

Do you think we can take that break now?

Q This is my last question on this article is this one sentence and then I'm done. I'm just trying to understand your testimony about it.

What is ambiguous about their use of the word "risk" in that sentence?

A I think I explained that a couple of times.

Q Do you agree or disagree that their study shows that the risk for non-Hodgkin's lymphoma increased with duration of exposure?

A You know, we had this whole discussion when we were talking about the abstract because they said almost the same thing in the abstract except they used the word RR which is "relative risk," and I think we covered this already when we were talking about the abstract.

Q Defining "risk" as you deem appropriate and necessary, do you agree or disagree that the risk for non-Hodgkin's lymphoma increased with the duration of exposure in this study?

MS. KAHN: Objection. Asked

and answered.

THE WITNESS: What I think I disagree with -- I think the implication that there's a big difference between five to nine years and greater than 10, one cannot say that. I mean that's where I'm having the difficulty in terms of relative risk, because we're going from .7 to 3.3 and then to 4.2. So, I think the way I would look at this information, it's statistically significant above 10, but between five and nine it's 3.3 versus 4.2, and I don't think that -- if the implication is that there's a big dividing line between 10 versus less than 10, I'm not sure that one can conclude that from this data.

BY MR. HANLEY:

Q Then are you saying that you disagree with the statement that the risk for non-Hodgkin's lymphoma increased with the duration of exposure?

A The way that -- I think that it's fair to say that the relative risk measured in this study, what they found,

they found a greater relative risk with duration of exposure, but I think that's all one can say.

Q So you agree then that they found increased risk with increased duration of exposure?

A Yes, but like I said --

MS. KAHN: Objection. Mischaracterizes the witness' testimony, vague and ambiguous, argumentative.

THE WITNESS: As I said, they found a greater relative risk in terms of what we -- they measured relative risk for non-Hodgkin's lymphoma in the longer duration exposures.

BY MR. HANLEY:

Q All right. And I believe you started your answer by saying "yes," with that explanation that you just gave; correct?

A Well --

Q I want to make sure we're clear on our record because you were interrupted by counsel's objection. So I want to make it clear that the answer to my

last question was, yes, with the explanation that you gave following counsel's objection?

MS. KAHN: Objection to the extent that mischaracterizes what the witness said.

MR. HANLEY: Does not.

BY MR. HANLEY:

Q Go ahead, Doctor.

A What this study showed --

Q Sir, I didn't ask you -- I asked a very specific question. My question was: In response to my last substantive question your answer was, yes, interruption by counsel, then continuing with your explanation; correct?

A I don't know. I've lost the -- I've lost the train of thought here.

MR. HANLEY: Then let me ask the court reporter to read back the question that I asked about, your answer, and then my follow-up question.

MS. KAHN: For the record, I was not intending to interrupt any testimony. We're doing this deposition

by telephone. And if one person is speaking, there's no way to know if another person is speaking at the same time. So I apologize if I did interrupt an answer, that certainly was not my intent.

THE WITNESS: And I apologize, I did not give my counsel a chance to proffer her objection before I answered the question.

(Record read as requested.)

BY MR. HANLEY:

Q So, Doctor, having had the record read back to you, it's correct that what I asked you if the risk increased for non-Hodgkin's lymphoma when the duration of exposure increased in this study, your answer is yes with the explanation that you've given; correct?

A Yes, with the explanation that we're talking about relative risk in this study. We're not talking about an actual risk.

Q How do you distinguish a relative risk versus an actual risk?

A Well, I just -- I think that it's a question of whether or not one can use the results of this information and generalize. I think that the relative risk is a measurement, to me, if -- if in some context, because as I mentioned before I get involved in risk assessment procedures. The question of risk means a sort of generalizable term, and so I want to make -- I just, I just wanted to make clear the distinction between a relative measured risk in a study and something then that one can generalize about.

Q Would you agree, sir that relative risk is a term of art in epidemiology?

A Term of art. Not exactly sure what you mean by that.

Q Would you agree that relative risk is a specific epidemiologic term with a defined meaning in the field of epidemiology?

A Yes.

Q Would you agree that there is no such term "actual risk" in the field of

epidemiology?

A Actual risk?

Q Correct.

A I don't know.

Q Is there a such a thing as specifically defined term "actual risk" in epidemiology --

MS. KAHN: Objection. Vague and ambiguous.

Q -- in the way that relative risk is?

A I have not heard that term used so I really couldn't say.

Q And another term, a specific term in epidemiology is "attributable risk", would you agree with that?

A I don't know that terminology, what exactly is meant by that.

Q Would you be able to describe or define the difference between relative risk and attributable risk as those terms are understood in the field of epidemiology?

A If you would like to show me or give me a definition for what you

understand as attributable risk, I would be glad to try to explain that.

Q I'm not the expert here, sir. I'm asking you to. Are you able to distinguish and define the terms "relative risk" compared to "attributable risk" as those terms are understood and used in the field of epidemiology?

MS. KAHN: Objection. The question lacks foundation and calls for speculation based on his testimony that he's not familiar with the term "attributable risk" in the field of epidemiology or his use by you in the field of epidemiology.

THE WITNESS: I think that was in an answer that I gave to the question. So based upon that, I don't know how I could answer your question.

BY MR. HANLEY:

Q Is that to say that you don't know the answer to that, you don't know -- you would not be able to define or distinguish for us the difference between "attributable risk" and "relative risk" as

those concepts are understood in the field of epidemiology; correct?

MS. KAHN: Same objection.

THE WITNESS: Could you repeat what I said about attribute risks so that --

BY MR. HANLEY:

Q No, sir, we're not reading back your answers. My question is --

A We read back my answers several times already.

Q Sir, we don't read back testimony so that you can repeat something. We don't need it, it's already written down, sir.

My question is this: Are you able to define and distinguish the concept of "relative risk" from "attributable risk" as those terms are understood in the field of epidemiology?

MS. KAHN: Objection. The question is argumentative, it's harassing, it's asked and answered, and if the witness wants to read back testimony to refresh his recollection or

refresh anything as you have asked him to do so many times, it's perfectly okay.

THE WITNESS: I think that based upon what I've said before, as I mentioned before, I'm not familiar with the term "attributable risk", so it would be difficult for me to try to speculate how that is different from "relative risk."

BY MR. HANLEY:

Q So your answer to the question is you are not able to do that; correct?

A I think I just answered that question. I said I am not familiar with the term -- the terminology, the specific terminology sitting here. I may have read it in the past, but right now in terms of what you mean by attributable risk, I'm not able to differentiate that from relative risk.

Q All right. My last question now on this article, and that is, do you agree or disagree that in this 1997 Hayes study that the risk for non-Hodgkin's lymphoma increased?

Would you agree that in this 1997 Hayes study the authors found that the increased risk for non-Hodgkin's lymphoma was strongest among workers who had a distant exposure to the benzene?

MS. KAHN: Objection. As framed the question is vague and ambiguous and undefined.

BY MR. HANLEY:

Q Distant as in equal to or greater than ten years?

MS. KAHN: It's also overly broad.

THE WITNESS: I don't think that based upon the data that I see in this paper that one can conclude that.

BY MR. HANLEY:

Q So you disagree with their conclusion on their point?

A No, I am disagreeing with your statement just now. We've already discussed, at length, what they have said in the paper.

Q Well, you agree that that is what they said in this last sentence we

read, and -- but you disagree with what they say; is that correct?

A That's not the question you asked me. You asked me what my opinion was about the issue of duration of exposure, and I answered it. I said, what I see in this data -- we've already gone over this, you know, for 10 or 15 minutes about what they said and whether or not I agreed or disagreed with it. But you asked me my opinion. And my opinion is, based upon the information that I see in this paper in table 2, I don't think one can necessarily conclude that the break off point -- you can make a break off point of ten years.

Q And my point is, sir, that they said that it did, and you disagree with that; correct?

MS. KAHN: Objection as to what they said. The article speaks for itself.

THE WITNESS: I've answered this question so many times already, and I'm afraid that each time you're asking me to answer it, you're going to make

something out of a slight difference in which I've answered it.

All I can tell you is what I have said in the past. I've answered that question. And I just answered your question about what my interpretation of the data is.

BY MR. HANLEY:

Q Sir, this is relatively straight forward, I'm not trying to trick you or anything like that.

A Then why do you keep on asking me the same question over and over and over again?

Q Because I'm not getting an answer.

A Yes, you did.

Q You're not answering my question, sir. And if you answer my question, I can move on. But if you don't answer my question, I will not move on until I get an answer.

A You asked me --

Q I am asking you yes or no questions. So, you can give all the

explanation you want, but I'm entitled to a yes or no or I don't know, but I'm entitled to straight answer.

My question is, is a straight question: Do not the authors of this article find that the risk of non-Hodgkin's lymphoma not only increases with the duration of exposure to benzene but was strongest among the workers who had a distant exposure to that, and that's what they find, and you disagree with that; is that right?

MS. KAHN: Objection. The article speaks for itself. The question has been asked and answered. If you don't like the witness' answer, that's not his fault. It's a compound overbroad question and it has been asked and answered.

THE WITNESS: If you want to point to a specific statement that they are making in this paper, I will comment on it, because you're asking me if I agree with something that they have said. If you point to the statement, then we'll

talk about it.

BY MR. HANLEY:

Q That's fine. You recall that we read the sentence, the last sentence that we read in this Discussion section that says, "The risk for non-Hodgkin's lymphoma increased with duration of exposure and was strongest among workers who had distant exposure greater to or equal to ten years."

Do you recall that we've been discussing this sentence; correct?

A Absolutely. That's what I've talked about and I thought I answered your question about that.

Q Now, we talked at some length about the first statement that they make. And now I'm asking you in that sentence, and I'm asking you now about the second statement that they make. That the association was strongest, or the increased risk, was strongest among workers who had a distant exposure.

A Which statement are we talking about?

Q The second half of the sentence, the part that says that the increase was -- there was -- excuse me, that the risk was strongest among workers who had distance exposure.

A Which page are you on?

Q 1068.

A Okay. What line now?

Q The first full sentence of the last column of the page --

A Yes.

Q -- where they state risk for NHL increased with duration of exposure and was strongest among workers who had distant greater to or equal to ten years exposure.

My question is: Do you agree or disagree with their statement that risk was strongest among workers who had distant exposure?

MS. KAHN: Objection. Asked and answered.

THE WITNESS: The relative risk as they found here in their table, and I think -- didn't we go through this with the yes and then the whole answer

thereafter?

BY MR. HANLEY:

Q No, sir. Because that was as to the first half of the sentence and the first statement that they make that the risk increases with duration of exposure. I'm now asking you about the second half the sentence where they state that the risk was strongest among workers who had distant exposure. Because it's a compound sentence, makes two statements, so that's what I'm asking you about now.

A Well, I think I mentioned the fact that -- I don't know that you can really say that you can differentiate between ten years versus five years in this. And because they didn't do a statistical analysis between those two, the five to nine to greater than 10, so I don't think one can conclude that necessarily from this paper.

Q So then your answer is you disagree with their statement on that point; is that right?

A Well, to a certain extent. I

mean I think that the data is a little bit greater for the greater than 10, but I'm not really sure that the difference between the five to nine to the greater than 10 would be a statistically significant result.

Q Okay. Thank you. Now, this study was funded by The National Cancer Institute; correct?

A Are we -- I thought that was the last question.

Q I want to finish on this, but then I'm done with this area.

MS. KAHN: I'm going to object that the question calls for speculation as to who funded the study.

THE WITNESS: I don't think they really say who funded the study in here.

BY MR. HANLEY:

Q So you don't know?

A I don't see it in the paper so I don't know.

MR. HANLEY: Okay. Let's go off the record.

(A discussion was held off the record.)
(Thereupon, there was a luncheon recess.)
BY MR. HANLEY:
Q Sir, could you describe for me all the things that you believe are established risk factors for the development of non-Hodgkin's lymphoma?
MS. KAHN: I'm going to object that this was already covered in the first section of Dr. Whysner's deposition.
BY MR. HANLEY:
Q I'm talking about things that you believe are more likely than not basis to a reasonable degree of medical and scientific certainty are in fact, based on your understanding of the literature, established risk factors?
A Certain hereditary immuno-deficiency syndromes that have been described in the literature; certain viral infections; and drugs that induce immuno-deficiency, for example, drugs that are used in -- after somebody has had a kidney transplant in order to prevent the

rejection of the graft. Those are the ones, the main ones any way, that I believe have been found to be causally related to the development of non-Hodgkin's lymphoma.

Q And is it correct that none of those apply to Mr. Molina?

A As far as we know, that's true, yes.

Q And it's your opinion that Mr. Molina has no particular risk factors that are established to a degree of certainty for non-Hodgkin's lymphoma in his history?

MS. KAHN: Objection, to the extent that this has been asked and answered and was covered in the first session of the deposition.

THE WITNESS: Well, I think I mentioned the fact that he has things that have been studied in the literature, and I don't know if some people have considered them to be risk factors or established risk factors, but -- and those would be the obesity and the smoking history and the worker as an

agricultural worker. And we talked about this before, I don't I think that -- I think that there is evidence both ways about those particular issues and I don't think that in sum, from my opinion at least, one can truly say that there's a causal relationship.

Oh, the other one I forgot to mention was the use of non-steroidal antiinflammatory drugs. But the -- none of those, at least from my read of the literature, can truly be say -- you can conclude to a reasonable degree of medical and scientific probability that they are causally related.

BY MR. HANLEY:

Q Do you recall that when we were speaking on April 1st that I asked you to establish with your office the precise number of hours that you had billed for this case?

A Yes.

Q And did you find that out?

A Yes.

Q And what was it?

A Seventy-two and a half.
Q And the work that you've done since that day is on top of that; correct?
A Yes.
Q And how many additional hours would you estimate since then?
A I couldn't say. Probably 10 to 20, maybe more, but I haven't really sat down and calculated my work to figure everything out for the last month yet.
Q Okay. So your -- is it fair to say then that the hours that you have in the case is somewhere between 90 and 100 plus in total hours?
A Well, I think you asked me before, and you said above 80. It's certainly above 80. Above 90, it may be. I don't know about above 100.
Q Now, before you started any investigation in this case, you were already of the opinion that benzene does not cause non-Hodgkin's lymphoma; correct?
A Well, I think, as I said when I was retained in this case, which would have been in April, I had already worked on

several cases and at least at that time, pending the fact that I relooked at the information that was available, I had concluded that non-Hodgkin's lymphoma was not associated with benzene exposure.

Now, the issue of solvents was one that it had been longer because the -- I think the previous cases all were pretty much restricted to benzene exposure. So the issue of solvent exposure, other solvents, I hadn't reviewed that literature maybe for a couple of years, I can't remember exactly. And that's an, in some sense that's a larger literature than the benzene literature.

Q Is that why you needed to take so much time to review the literature and the materials in this case because of the expanded question of whether his general exposure to petrochemical hydrocarbon solvents could be considered a cause?

MS. KAHN: Objection. Vague and ambiguous and argumentative.

THE WITNESS: Well, I looked at -- I certainly looked at the solvents

as far as I could figure out that are involved in this case, and I would say that a lot of the work that I did had to do with reading Mr. Molina's deposition, co-worker depositions, looking at all of the MSDSes. There were also depositions from toxicologists for the various defendants. There was an awful lot of -- I have several boxes full of notebooks, and so I don't know that I could characterize the need for the time as being mostly due to the question of solvents and benzene exposure because there are medical records -- and I forgot about the medical records. Just a lot of information that is just specific to this case that had to be reviewed.

BY MR. HANLEY:

Q Okay. One of the things that I'm getting at is trying to understand why you would need to put in so much time to the case if you start off already having the opinion that benzene or exposure to solvents in that facility, in that Salinas Firestone plant that you so familiar with

from working on other cases, what is it that you then needed to spend all that time doing and why would you need to do it? What difference would it make what his co-workers had to say, what Mr. Molina had to say about his exposure or what his medical records would show if you already, you know that what he has is non-Hodgkin's lymphoma, if that -- isn't that enough? Don't you know enough there already, he worked at that plant and he had non-Hodgkin's lymphoma, you wouldn't really need to know much else, would you?

A Well, I think that it's important to be as familiar as I can with the circumstances of his work environment.

The issue of alternative causes, which we just talked about briefly a few minutes ago, again, that is another area which I think I previously testified to the fact that it's a rapidly evolving literature. And some of those other risk factors that we have talked about, the smoking, the potential risk factors I should say, the smoking, and the obesity,

and non-steroidal antiinflammatory drugs. There is a lot of literature coming out on those. So I had to re-review that literature to see if any of those had reached the level of causal association in terms of Mr. Molina's medical records.

And I just feel that it's necessary for me to be familiar with the medical records in any case that I'm involved in because I might be asked to describe his medical conditions, and I feel like I should be prepared to do that.

Q Is it fair to say that the reason it's important for you to be familiar with the particulars of the case is specifically to be prepared for your trial testimony?

A Trial and deposition, and as I said before, if there were any of these other risk factors that reached the level the causal relationships, I would be able to talk about that. And, obviously before I started out reviewing the medical records, I really wasn't sure that he didn't have some of those conditions which

I -- which we discussed which are clearly causally related to non-Hodgkin's lymphoma.

Q What do you consider to be the most important studies supporting your conclusion in this case that his cancer is not caused by benzene and exposure to other petrochemical hydrocarbon solvents, but, rather, is idiopathic?

MS. KAHN: Objection. Vague and ambiguous with respect to the word "important."

THE WITNESS: Well, I think the literature has to be seen in its totality. I mean with the Bradford Hill elements of looking at epidemiology studies, for example, one needs to look for consistency.

And you know, I think that it's fair to say that there is -- which we spent a lot of time talking about the Chinese study, and earlier there are several studies that don't find any indication or clear indications of any kind of associations even with non-Hodgkin's lymphoma.

We discussed the reasons why I thought that one study was not -- could be attributed to other chemical exposures, but I don't think there's any one study.

I think the Rinsky study is probably the most convincing. The one that Dr. Infante started out back when he first published in 1977, they hadn't published any information on non-Hodgkin's lymphoma, nor in '87, I don't believe. But it was in 2002 when they published the non-Hodgkin's lymphoma results, and, you know, that is a study that has good dose reconstruction data and is clearly people who have been highly exposed to benzene, and that's the study that's used by the Environmental Protection Agency to come up with use and risk assessment for the purposes of addressing the risk of benzene as it relates to acute myelogenous leukemia. So I think that the Rinsky study of the cohort analyses is probably the strongest study.

In terms of the case-control studies, there are 30-some of them. It's, you know, there are handful of them that show some kind of association with benzene, and the rest of them are in the other side of the balance of not finding association. So, that literature with the case-control studies I think has to be treated -- I wouldn't say that any one particular study there stands out as a lot stronger than the other studies.

BY MR. HANLEY:

Q When you read the videotaped trial testimony of Dr. Bennett, was there anything in particular that stood out as something that you disagreed with?

A Dr. Bennett?

Q Yes.

A I can't really recall anything. I was mostly looking for, you know, the types of information that you might be looking for. But I -- it would be hard for me to -- hard for me to -- I don't recall anything that I disagreed with. But that's not to say that there might not be

something but I just, I can't right now remember anything.

Q The Bradford Hill criteria that you referred to is a criteria for causal inference. Under epidemiologic guidelines, in other words it's an epidemiologic criteria; correct?

A Again, I don't want to use the word "criteria." Actually, Bradford Hill specifically warned against using his type of analysis as criteria. Bradford Hill, this was my understanding was that he developed these -- this way of analyzing the literature primarily with regard to looking at smoking and lung cancer. And so it also has, I think has to be seen a little bit historically in the context of that particular endeavor. But he did talk about also, you know, the Percival Pott, the chimney sweep work and so forth.

Q Those factors for consideration laid out by the Bradford Hill investigators is an epidemiologic -- those are epidemiologic conclusions and involved the use and deployment of epidemiology;

correct?

A Well, I think it's a little broader than that. First of all, it's a way of looking at a number of studies and it also includes -- I think we discussed this again at some length the other day -- the issue of -- when you asked me about biological plausibility, and there are other elements that basically say from a biological standpoint is this -- is what we're talking about, sort of coherent with what we understand biologically about the process. And the development of the -- development of the disease. And I think sort of sums it up because it says the environment and disease association or causation. And I think the primary purpose of this, because some, you know, studies, they are always -- well, there are often studies that show some association between a particular environmental exposure and a disease. And the question is, is that -- is that an association that is not causal or is that a causal association. Because there are a lot of reasons that

associations can be there for -- for reasons other than an actual causal association.

Q That's an epidemiologic question; correct?

A Well, not entirely. I mean, for example, it helps to understand something of the biology.

In one of the studies that I was involved with at Washington Occupational Health, although I became involved with them after this study was published, but I wound up doing medical surveillance on the same group of workers. A finding of triglycerides was associated with PCB levels. And it was pretty clear that that was an association, but the fact of the matter is that the -- in the original interpretation was that people had higher PCB levels had an elevation in their triglyceride levels. Well, it turns out that the reason for that was actually that the fact that people who had more fat in their blood tend to partition fat soluble chemicals into the blood more compared to

the fat tissue. So instead of the PCBs causing the elevated triglycerides, people began to realize it was the elevated triglycerides that was causing the higher level of PCBs in the blood.

So there's an example of an association that was found epidemiologically but I would say that the explanation for it not being a causal association was not based upon epidemiology but was based upon what we know about the biology of the system.

Q Would you agree that the issue of whether or not benzene or other petrochemical hydrocarbon solvents caused non-Hodgkin's lymphoma is an epidemiological one?

A Well, ultimately I think it has to be proven or disproven based upon epidemiological data. I think that there's also an underlying understanding that non-Hodgkin's lymphoma is a separate disease from acute myelogenous leukemia. So I think that both of those elements are important to realize that we have to know

whether or not benzene is causally related to non-Hodgkin's lymphoma based upon studies that have specifically looked at non-Hodgkin's lymphoma, and that goes beyond epidemiology. That has to do with our understanding of those diseases. And I think we discussed that again at some length the other day.

Q I want to be clear on your opinion that this cancer that Mr. Molina has is idiopathic.

Are you saying that -- is it your opinion that the cause of his cancer is unknown but there is some cause or are you saying that there is no cause of his cancer?

A Well, I guess I hate to quibble, but I think the problem is in the word "cause." Cause seems to imply something happening from outside of the normal process of what happens in living cells.

And, you know, one of the theories, for example, and this is not one that I'm -- I have put forward, but other

people have put forward, has to do with the cause of acute myelogenous leukemia being oxidative DNA damage. And we talked about that earlier today.

Now, the fact that oxygen is normally in the body, that it's a part of our normal processes, and if oxidative damage was -- did cause a genetic alteration -- sorry, we're being disturbed a little bit people outdoors.

But the fact that oxidative DNA damage may be involved, oxidative DNA damage is an ongoing process, it's normal in the body, it happens to all cells, and it's normally protected against, so, if that would be the reason for his non-Hodgkin's lymphoma, and I'm not saying that happens through benzene exposure, it would be hard for me to say whether there is no cause or there is a cause, because of the fact that that's a normal sort of process.

Also, we discussed the fact that just cell replication, which happens all the time in the body, there are errors.

And the fact that if there's an abnormal form of DNA developed through this process, which is a, you know, that comes about as close to spontaneous or without a cause as I can think of. But is the genetic alteration that happens because of the lack of proper replication of the DNA, that could be considered a cause. And I guess I'm -- the difficulty here is with the word "cause" I think.

There are things that happen just because of the normal replication and environment of cells that don't have anything to do with what we would consider to be sort of unusual exposures to anything. And I think those -- that may be the best explanation for his non-Hodgkin's lymphoma.

Q Do you have an opinion as to whether or not his non-Hodgkin's lymphoma was caused or contributed to by any environmental exposure, environmental toxic exposure?

A You mean -- are you talking about benzene and the solvents or are you

talking about anything else?

Q I'm talking about whether it was caused by anything else, anything at all.

A Well. All I can say is there is no indication that there is anything else, that, you know, as I mentioned, the -- I mean if there were in the next five years, if there were a whole bunch of studies that said that smoking was associated with it, we might have a different conversation. But sitting here today, I don't see anything that has been identified in his environment that could have caused his non-Hodgkin's lymphoma.

Q What is your understanding of the development of the state of the art or the historical knowledge regarding benzene causing cancers of the blood forming organs?

MS. KAHN: I object, only to the extent that this might be outside the scope of the witness' expert witness designation.

MR. HANLEY: Well, tell me if

you're not calling him to ask any of those questions and I won't have to ask them myself. If you're not calling him regarding the historical state of the art, who knew what when about --

MS. KAHN: I would ask, on the assumption that we will ask him, to express opinions on that.

THE WITNESS: Well, my -- this is really related to the development of leukemia and specifically acute myelogenous leukemia. I think that the -- I think I have in my files, for example, a document from the National Institute of Occupational Safety and Health, and I think that this probably represents the state of the art or the understanding of whether or not benzene could truly be said to cause, well, at that time it was leukemia because they weren't differentiating necessarily among different subtypes of leukemia in studies, but I think Infante's study was the study that really established the fact that benzene was associated with, at

least some form of leukemia, because the total number of leukemias as they were measuring them in that study went up which later was found to be acute myelogenous leukemia.

But in 1974 NIOSH basically said that -- I'm trying to find the particular quote. But in this particular document somewhere they say that the information regarding benzene and leukemia is -- hasn't really been established conclusively. So I think that's about the time frame, I think somewhere between '74 and '76, '77, was when there was -- people concluded that it did cause a form of leukemia. And I think that the IARC came out in 1982 with the conclusion that benzene was a human carcinogen and that AML was the neoplasm which had been specifically identified.

BY MR. HANLEY:

Q What is your understanding of when it was first reported anywhere in the scientific literature that benzene may be a cause of any kind of cancer?

A Well, I think there were studies -- and I have a couple of these here from -- in the 1939 studies, that at least reported the effects of benzene, and they discussed some cases of people who had been exposed to benzene who also had leukemia. The problem was, there wasn't any kind of epidemiological study that could say whether or not those findings of leukemia and other diseases were at increased amounts in that population or whether it was coincidental that people who were exposed to benzene, and there were a lot of people exposed to benzene, also had these diseases.

That's always the -- that's always the problem with case reports or clinical series like the ones that had been -- had been available before the Infante study.

Q When was the first epidemiologic study associating benzene with any form of cancer?

MS. KAHN: Objection. Over broad, vague and ambiguous, asked and

answered.

THE WITNESS: Well, I would say the one that was really well conducted was Infante study.

Now, there was a study of Turkish shoe workers earlier, but I don't think -- I haven't seen any real statistics as far as I know reported until after that Infante report, although the study was done ongoing and there were earlier reports, but I don't believe that there was any convincing statistically significant data presented.

BY MR. HANLEY:

Q When was the first epidemiologic study presented, what year?

A Well, that depends on what you mean by "epidemiologic study." I mean, if you include case reports -- are you including case reports in epidemiologic studies?

When I talk about epidemiologic studies, I usually mean the kind of study where either a case-control study where you have statistics or a cohort

analysis where you have statistics. I guess there are even descriptive studies where you -- statistics where you compare different population groups. I don't consider things like case reports and clinical series, in other words a doctor or a group of doctors say, We have a group of patients that we've seen who have leukemia and they have been exposed to benzene, but there is no statistical analysis presented. I guess I usually don't think of those in terms of being epidemiological studies. So, again, with that in mind, I would say that the -- there was some indication that there was work being done among the Turkish shoe workers, but the first real publication was the Infante study.

Q When was the epidemiologic study on the Turkish shoe workers first published?

A You know, I don't have that information with me because it's not something that I usually rely upon. And I think it was -- I think there were some earlier publications before 1987, but I

couldn't give you the exact details of those.

MS. KAHN: I'm sorry, Dr. Whysner, what year did you say? The phone cut out.

THE WITNESS: I didn't mean '87, sorry. Whatever I said with Infante should be 1977.

BY MR. HANLEY:

Q And when was the Turkish shoe study done, approximately, as best you can recall?

MS. KAHN: Objection, asked and answered.

THE WITNESS: I could actually -- they probably talk about that in that ATSDR talks profile. I can look that up for you if you want me to.

BY MR. HANLEY:

Q I'm just asking your best recollection of when it was?

MS. KAHN: Calls for speculation.

THE WITNESS: As I said, in terms of epidemiological evaluation,

meaning statistical analysis, I think it was after 1977. But the study was ongoing and there were reports, but, again, I believe that prior to 1977 they were just reported as a clinical series. In other words, there was no -- at least that's my best recollection is that there was no statistical analysis to show that the amount of various diseases was any greater in that group than one would expect in the general population.

BY MR. HANLEY:

Q Is 1939 the first reference in the published literature that you can find to benzene being a potential carcinogen?

MS. KAHN: Objection. Over broad, vague and ambiguous.

THE WITNESS: Well, as I said, the 1939 paper has described certain cases of leukemia among people who had been exposed. I don't know that there was any -- I can't remember exactly what the authors concluded from the study.

I believe there was some

earlier case reports going back. I think there was one even before the turn of the century that had talked about this, but it might have been just a single case, I can't remember exactly.

BY MR. HANLEY:

Q Between 1939 and 1997, how many references in a scientific published literature were there to benzene being a potential carcinogen in case reports and the like?

MS. KAHN: The question is overbroad, compound. It's vague and ambiguous.

THE WITNESS: I don't know that I can give you a number.

BY MR. HANLEY:

Q Could you give me something approximate?

MS. KAHN: Objection. Calls for speculation.

THE WITNESS: You're including case-control studies in that, too, I presume?

BY MR. HANLEY:

Q Well, I'm talking about from 1939 until the first case-control study was published.

MS. KAHN: The question calls for speculation in light of the witness' testimony.

BY MR. HANLEY:

Q Can you tell me when the first case-control study was published?

A Again, a number of questions going. The first question was from 1939 to 1997.

Q No, 1977. Let me start over.

A I think you said '97, that's why I was --

Q Let me start over.

Can you tell me when the very first case-control study relating to benzene as a potential carcinogen was published?

MS. KAHN: I object. The question is vague and ambiguous.

(Witness perusing documents.)

BY MR. HANLEY:

Q Are you doing something to

evaluate --

A Yes, I am looking through my file here, case-control studies, to tell you which one is the earliest date.

Q Well, do you know whether or not it predates the 1977 Infante article?

A Well, one of my -- I got 1988. That's -- I don't see any that I have that predate the Infante article.

Q Do you know whether or not any epidemiologic study regarding benzene as a potential cause of cancer was published before Infante's 1977, do you know whether or not that's the case?

MS. KAHN: Can, I please hear the question again.

(The requested portion was read.)

Q Let me start over. Do you know of any epidemiologic study of any kind that was related to the issue of whether or not benzene was a cause of cancer that was published before Infante's 1977 article, do you know whether or not any such study was published?

MS. KAHN: Objection.

Compound, overbroad, vague and ambiguous.
BY MR. HANLEY:
Q As you sit here today, sir, and you are looking through your materials --
A Well, I'm looking through this NIOSH 1974 document because I figured that they would have reviewed studies, and I might be able to answer your question at least up to 1974. Okay, epidemiologic studies.
(Witness perusing documents.)
Q I note that we've been waiting some number of minutes for you to look through your materials. Is it fair to say, sir, that without looking back at the literature, you don't know whether or not there were any such epidemiological studies?
A Well, again, as I mentioned before, when I think of epidemiologic studies, I'm thinking about studies that I have actual statistical analyses, and it's my recollection that there weren't any studies before 1977 that were able to say

that with any kind of reasonable -- what I would consider to be reasonable degree of medical and scientific probability, i.e., statistical analysis, that there was a association between benzene exposure, and in this case it was all leukemias. And that's the earliest study that I can recollect that which I would call a true epidemiologic study as opposed to that '77. As opposed to case reports or just discussions of clinical findings among people exposed to benzene.

Q Okay. I would --

MS. KAHN: For the record, I just want to state the witness was looking through his materials for less than a minute according to my watch, and if there are materials that he wants to look at in order to give a complete answer of a question, I'm going to advise him he has a right to do that.

BY MR. HANLEY:

Q Sir, my question was a little broader than that. It was whether or not there were any case control or any type of

epidemiologic investigation into the issue of whether or not benzene was a carcinogen, regardless of whether you find them persuasive or conclusive or determinative on the issue. Were there any such publications in the scientific literature prior to 1977, if you know, as you sit here today, without taking any great length of time to investigate it as you sit here.

MS. KAHN: Objection. Argumentative, asked and answered, overbroad, vague and ambiguous.

THE WITNESS: What's a great amount of time?

BY MR. HANLEY:

Q Do you know the answer to the question as you sit here right now?

MS. KAHN: Objection. Argumentative.

(Witness perusing documents.)

THE WITNESS: Well, you talked about, I mean, you mentioned -- I'm trying to get the wording down of what you wanted. You said case control, which is a certain kind of study I guess

you know. That would be a study of people who have leukemia.

BY MR. HANLEY:

Q Yes, sir, or any other kind of epidemiologic investigation. Do you know of any sort of epidemiologic investigation published in the scientific literature relating to the issue of benzene as a carcinogen prior to 1977?

MS. KAHN: Please, don't interrupt the witness' answer. Your new question is argumentative. Argumentative, overbroad, compound, vague and ambiguous.

BY MR. HANLEY:

Q Go ahead, sir, if you please.

A Okay. So we discussed those article from 1939. So would you include -- I have to ask you the question, would you include -- is that within your meaning of epidemiological studies, because I have given those to you previously?

Q Do you consider the 1939 article an epidemiologic study?

A I think it's a case -- a

series of cases. I would not consider it to be what I -- I think that there -- I mean what I consider to be an epidemiological study would be one that includes statistical analysis. I think I already defined that a couple of times.

Q Is there any study in a scientific literature published prior to 1977 you that would consider to be an epidemiologic investigation into the issue of benzene as a carcinogen?

A If you include case studies and clinical series as within the rubric of epidemiologic studies, then there are studies, and I have mentioned a couple of those.

Q Are those -- my question was anything that you considered to be an epidemiologic study. Are you saying that you consider those to be epidemiologic studies?

MS. KAHN: Objection. Argumentative. Mischaracterizes the witness' testimony. The question is asked and answered.

MR. HANLEY: It's asked. It's not answered.

THE WITNESS: I don't think I can answer it any better that than.

BY MR. HANLEY:

Q Sir, do you consider those -- is there any study in the scientific published literature prior to 1977 that you, sir, Dr. Whysner, consider to be an epidemiologic investigation into the issue of whether or not benzene causes any form of cancer?

MS. KAHN: Objection. Argumentative, asked and answered, vague and ambiguous.

THE WITNESS: All I can say is this, is that if we consider -- and I'm just trying to define the term epidemiologic right now.

Q I've asked, sir --

A You said --

Q I asked what you considered to be an epidemiologic study, sir. You defined it, what you considered to be an epidemiologic study, and then you tell me

if you consider there to be any such study before 1977, an epidemiologic investigation regardless of whether it was determinative, regardless of whether you feel it compels a particular result or not. Was there any such investigation made, published in the literature before 1977, it's a simple question?

MS. KAHN: Argumentative, asked and answered, vague and ambiguous.

THE WITNESS: Can I define what I'm calling an epidemiologic study --

BY MR. HANLEY:

Q Please.

A -- as part of the answer which I think I have done.

See, my -- the way I define an epidemiologic study is one that includes a statistical analysis. And the Infante study is the first one that I have seen that includes a statistical analysis.

Q All right. Thank you.

Now, how many references in the scientific literature of any other

sort, case studies -- excuse me, case reports or series of cases or any other references in the literature are there, approximately, to your best recollection as you sit here today, prior to 1977?

A Okay.

(Witness perusing documents.)

THE WITNESS: So there's a study by Vigliani, V-I-G-L-I-A-N-I. And then there's the Turkish study by Aksoy, A-K-S-O-Y, published in 1974. There was a case -- well, there's a case of benzene poisoning. There were reports, as I mentioned, by Vigliani, Goguel, G-O-G-U-E-L. Mallory, M-A-L-L-O-R-Y, 1939. And we discussed the two other 1939 papers which I have in my file. And those are the -- those are the ones that I can -- ones that I can recall that described people who had leukemia and also had benzene exposure and I can consider them neither case reports or clinical series.

BY MR. HANLEY:

Q And is that all of them that

you're aware of in the scientific literature?

A That's all I can recall right now.

Q Thank you.

Now, what work have you done in your professional career outside of litigation where you have been called upon to determine the cause of someone's cancer, if at all?

A You mean in terms of clinical practice?

I've never, you know, worked as a -- as a clinician in the field of cancer, if that's what you're getting at.

Q So is it fair to say, outside of litigation, you have never been called upon or involved yourself in the process of trying to determine what any given individual's cancer may or may not have been caused by; is that correct?

A Well, probably there is some instances when I was doing my pediatric -- you mean why it was caused rather than it being diagnosed?

Q Correct. I'm not talking about diagnosis and treatment, I'm talking about etiology of someone's cancer.

A All my work in this area has been as a researcher and not as a practicing clinician. So in terms of research, I've been involved in studies of groups or in terms of, you know, doing experimental research, but I haven't in terms of causation, but I haven't in terms of a clinical analysis, I haven't done that.

Q So you haven't done it ever with respect to an individual; is that correct?

A You mean as a practicing physician?

Q In any context outside of litigation?

A Well, not that I can recall right now, no.

Q All right. Now, as to generally whether a particular toxin of any sort is a cause or not a cause of any particular kind of cancer, what work have

you done in that regard outside of the context of consulting and litigation?

A Well, in terms of, you know, some of my work had to do with various chemicals which I've published in the scientific literature that have to do with -- I can give you an example.

The question came up of whether phenol barbital caused liver cancer in humans, and in that context I was not only involved in doing experimental work in animals looking at genetic -- possible genetic damage caused by phenol barbital but also involved in an epidemiology study where we looked at whether or not people who had been exposed to phenol barbital for long periods of time -- they were being treated as epileptics -- whether there was an indication that there was a higher, greater amount of liver cancer among those individuals.

Paradoxically, we did find that there was an increase in non-Hodgkin's lymphoma among these people, but it's a single finding and I don't know that I

would put a lot of -- we did find an association, but I don't put a lot of credence in that one association.

I've done a lot of work in a chemical called acrylonitrile, which, remember we were talking about oxidative DNA damage which this chemical produces brain tumors in rats, and it's pretty potent in doing that. And we found that in conjunction with the doses that produce those brain tumors in rats, there was an increase in oxidative DNA damage in the brain of those rats, and, therefore, that finding might be causally related to the increase in somehow acrylonitrile enhanced oxidative DNA damage in rats and, therefore, produced drain tumors.

I've done work on PCBs. I've done work in chlordane. I have done work on an antioxidant that causes forestomach tumors, but it's a widely used antioxidant called butylated hydroxyanisole in terms of forestomach tumor production in animals to see if that was at all relevant in human cancer.

And of course we talked about my work which was -- it was a little bit of an experimental work, but most of my work on benzene had to do with looking at the results of other people's genotoxicity test to see whether or not they fit within a certain framework of a mechanism that we could say looked like it was responsible for the genotoxicity of benzene. Actually, benzene metabolites as we discussed.

Q You described six different areas in which outside of litigation you have been involved in work relating to the determining whether or not a given chemical is a cause of cancer, is that right, those six areas?

A Well, I mean I don't know if you include my work with the International Agency for Research on Cancer where I was involved in working groups that produced the IARC monographs. And I was involved in working on those for, I think we probably did somewhere between 40 and 50 chemicals to determine what -- whether or not they were causative of human -- could be

predicted to be causative of human cancer. But that was not my own experimental work. That was, you know, that was participating in the working groups.

Q In all of these areas that you've described, the six areas that you you've done experimental work, were epidemiologists involved in any of that work?

MS. KAHN: Objection. Vague and ambiguous.

THE WITNESS: I'm trying to remember. I mean obviously the one -- you're just talking about the experimental work now?

BY MR. HANLEY:

Q You described the phenol vitatal (sic).

A Phenol barbital.

Q Phenol barbital as the cause of liver cancer. You discussed the acrylonitrile, the PCBs, the chlordane, the antioxidant, the butylated something.

A BHA.

Q BHA. And the work relating to

the benzene metabolite. Did your work in any of those areas involve the assistance of any epidemiologists?

A Well, I say we published a paper with Jorgen Olsen who is the head of the Danish Cancer Registry, at least was, I don't know if he is anymore. But that was an epidemiology study on phenol barbital.

The -- I published papers with epidemiologists regarding certain issues. Well, this isn't of those six chemicals that had the experimental work, although I have to admit that a lot of my ideas for doing the experimental work came out of the results of epidemiology studies.

The work that I did -- I did some other work where we looked at certain issues. And in those cases I worked with epidemiologists analyzing other chemicals, additional chemicals. The one name that comes to mind is Peter Boyle who is now the head of the International Agency For the Research on Cancer. Another name is an epidemiologist named Demetrius Tricolopolous who is at Harvard. And there

are probably others, but I just can't recall right now, where I've worked with them looking at epidemiological studies and coming to conclusions regarding a particular issue.

Q In these studies or investigations where you have worked with epidemiologists, what is the role that you have performed in the investigation compared to the role performed by the epidemiologists that you were working with?

A Well, my primary role was -- my specialty is in the area of mechanism and cancer mechanism in order to determine whether or not findings in experimental animals can be extrapolated to humans, and that has to do with comparative analysis of animal and biological systems. However, often I get involved in doing analysis of the epidemiology studies as well because, for example, in the IARC working groups, one of the four subgroups is the epidemiology group. But even though they come out with a recommendation regarding certain findings, it's still up to a vote

of all the members of the working group in terms of whether or not, for example, something has been found to be a human carcinogen. And in order to vote as a member of the working group, you have to do your own independent analysis of the literature in order to figure out whether or not that is indeed a human carcinogen. So even though my primary role was as a cancer mechanism person, I'm still required to make a decision about epidemiology studies as to whether or not they do show that something is a human carcinogen.

Q And with respect to what sorts or forms of cancer do you consider yourself to be an expert in the mechanisms by which those cancers develop?

A In general, you know, I would say, yes, there's some cancers I'm more familiar with than others.

Q Which forms of cancer do you consider yourself to be the expert in the mechanisms by which those cancers develop?

A Well, I'm certain we spent a fair amount of time with acute myelogenous

leukemia and benzene, and, therefore, the underlying mechanism by which benzene produces those effects. I've looked at issues of relating to benzene toxicity in terms of, you know, the mechanism by which it could produce aplastic anemia or myelodysplastic syndrome. I think I'm -- so I'm generally familiar with hematological and lymphatic cancers because of that interest of mine.

I would consider myself an expert in brain cancer development.

I've done work with breast cancer development.

I've done work with gastrointestinal development. And you know, there are probably some others that I can't recall right as we're sitting here right now.

Q What work have you done determining whether any given toxins are a cause of cancers of any blood forming organs?

A I think I just answered that about benzene and acute myelogenous

leukemia.

Now, when you say, "work," I guess you're talking about experimental work or literature reviews or --

Q No. I'm talking about any experimental work.

A Well, I did some work on benzene and DNA binding in terms of benzene mechanism.

Q Anything else?

A In terms of experimental studies?

Q Any published research relating to the issue of determining whether any given toxins can cause cancers of the blood forming organs?

MS. KAHN: Well, that's a different question.

THE WITNESS: I've published a fair number of papers. I'm probably going to forget some. But, I think benzene has been the one that I've looked at the most.

BY MR. HANLEY:

Q Would you agree that

Mr. Molina was exposed to benzene and other petrochemical hydrocarbon solvents in his work at the Firestone Salinas plant both by inhalation and dermal absorption?

MS. KAHN: Objection. Overbroad, compound, vague and ambiguous.

THE WITNESS: I guess I don't know the answer -- I mean I haven't really looked into his exposure. I know what other people have, in this case, have -- have developed. And that was a -- I'd have to look at the studies right now, but I think it's somewhere between four and 11 ppm years of exposure to benzene. And I don't have any reason to disagree with those, but I haven't been asked specifically to look into that issue.

BY MR. HANLEY:

Q I'm just asking you as a general qualitative, and not necessarily a quantitative analysis, but qualitatively, having reviewed his deposition testimony, his description of his work, his coworkers testimony, the conclusions drawn by

Dr. Reiss and Dr. Nicas, would you agree with me that that Mr. Molina was indeed exposed to benzene and other petrochemical hydrocarbon solvents in his work at the Firestone Salina plant?

MS. KAHN: Objection. Overly broad, compound, vague and ambiguous.

THE WITNESS: Well, I think that there is indication that he was exposed to benzene and these other solvents, but how much, I really couldn't say.

BY MR. HANLEY:

Q Now, you would agree that smoking cigarettes can cause lung cancer?

A Yes.

Q And would you agree that it is the totality of the chemicals in the cigarette smoke that is properly associated with an increased risk of lung cancer from smoking as opposed to the individual chemicals in the smoke?

MS. KAHN: Objection. Overbroad, vague and ambiguous.

THE WITNESS: I think I

mentioned before that I don't think we really understand which or how many of the chemicals in cigarette smoking is responsible for causing lung cancer. So that would be a difficult question for me to answer, and I've never really -- I mean, like I said, I used to go to seminars at the American Health Foundation because there was a lot of research going on to try to figure out those answers, but I really don't have an opinion about that right now.

BY MR. HANLEY:

Q Would you agree that it is the totality of the chemicals in cigarette smoke that are properly considered to be the carcinogen rather than any of the particular individual chemicals themselves?

MS. KAHN: Objection. Asked and answered, lack of foundation, calls for speculation. Beyond the scope of the witness' assignment and beyond the scope of his designation in the case.

THE WITNESS: Sorry, I just don't know the answer to that. It's not

an area that I'm -- that I've really kept up with the literature on.

BY MR. HANLEY:

Q Do you recall my questions of Dr. Bennett relating to the Shell study that he was involved with?

A I remember there being some mention about it, but I don't remember your questions.

Q Have you ever read that study that was referred to?

A I don't recall.

Q Would you agree that if you are comparing a would be exposure cohort to a control group in an epidemiologic study that it is important to have data relating to the exposure that you are attempting to evaluate?

MS. KAHN: Objection. It's an incomplete hypothetical, the question is overbroad, compound, vague and ambiguous.

THE WITNESS: Sorry, do you mean quantitative information or qualitative information?

BY MR. HANLEY:

Q Either one.

MS. KAHN: Same objection.

THE WITNESS: Well, all I can say is I think that -- I think that it's probably better to have quantitative information in terms of being able to define the parameters of the study in terms of what you're actually -- you're actually looking at. But, you know, I don't -- for example, we talked a bit previously about some of these older -- some of the epidemiology studies, and, for example, the original Infante study. I don't think -- I'm looking at it right now. I was looking to see whether or not they had actually some quantitative exposure information in that study. I think they did have some information. I don't know that it was -- and certainly to they did dose reconstructions later in terms of their study.

BY MR. HANLEY:

Q Are you done with your answer?

A I'm just looking here.

(Witness perusing document.)
I think that there are certainly studies in the literature where there hasn't been quantitative information that -- some of these studies in my file here on benzene exposures, these case-control studies for example, I don't think they have quantitative information because they're just asking people whether or not they have been exposed to benzene. And yet, you know, there are plenty of these studies published in the scientific literature, so I don't think it's an absolute requirement to publish an epidemiology study.

Q Would you agree that it's important to have in your would be exposure group some information as to whether or not the people were actually exposed to the chemical being evaluated?

MS. KAHN: Objection. Overbroad, vague and ambiguous.

THE WITNESS: Well, it's probably better if you do. I don't know that it's -- as I mentioned, a number of

these studies that we've got in our files here, and I think that Dr. Infante had his files and Dr. Weisenburger had in his files, are studies where -- and they said they relied upon them, are studies where there actually wasn't verification. I mean it was based upon people self-reports of whether or not they had been exposed to benzene. I think those -- I think studies where there is quantitative information about benzene may be better studies but not necessarily so because there may be other difficulties with those studies.

BY MR. HANLEY:

Q Would you agree that if you have a would be exposure cohort that has non-exposed people mixed into it that you will dilute the findings in terms of establishing whether or not there's an association between the chemical being evaluated and the disease being looked for?

MS. KAHN: Objection. Incomplete hypothetical, overly broad, vague and ambiguous.

THE WITNESS: Well, I think I would have to actually be -- in order to give you an opinion on something like that, I would have to actually be looking at a particular study to see, you know, what the number of people, how that was determined that they weren't exposed. It sort of depends upon the study. I mean my guess is that -- well, I know that people in cohort studies have a wide range of exposures, and I don't know any -- many epidemiology studies or maybe any epidemiology studies where there isn't a fairly wide range of exposure levels including some people who don't -- probably don't have any exposures. So I think it's -- so I think it's the nature of doing an epidemiology study that you can't be certain that everybody within that so-called exposed cohort has truly been exposed to the chemical.

BY MR. HANLEY:

Q Would you agree as a general proposition that to the extent that you have non-exposed people in a would be

exposed cohort that you will necessarily dilute your findings relating to causation?

MS. KAHN: Objection. Overly broad, vague and ambiguous.

THE WITNESS: Well, I think you have to predicate that on the fact that the thing you're studying is truly caused by that particular chemical. I mean, obviously, if it's something that isn't caused by that exposure, then it wouldn't make any difference. But I would say that if you do have a -- if you are studying something that truly is related, causally related to a particular chemical exposure, and you had a significant number of people -- and I don't know what that would be -- who were not exposed, it could dilute the findings and decrease the SMRs of the study or the relative risks I guess, since you're talking about a control group. But I don't know whether it would -- it would all depend on the details within that study about whether or not it would significantly do so.

MR. HANLEY: Okay. All right. Thank you, sir. I have been on the record with you four hours now, and we'll send you a check for that time. And those are all of my questions at this time.

MS. KAHN: You also owe Dr. Whysner an hour from April 1st.

MR. HANLEY: Yes. That check should have gone out by now. Have you gotten that?

THE WITNESS: Yes, I got that and I got your check increasing the amounts from 450 to 550.

MR. HANLEY: All right. So I'll send you now 550 times four.

THE WITNESS: Okay.

MR. HANLEY: Thank you, sir.

THE WITNESS: Okay.

MS. KAHN: Thank you, Madam Reporter.

THE REPORTER: Ms. Kahn, I wanted to ask you on the record, you wanted to order a copy of today's transcript?

MS. KAHN: Yes, I do. It's fine with me if you send the original of the transcript -- actually, you probably should send it to my office so I can send it to the witness to make corrections, if that's all right with Mr. Hanley, and then I will retain custody and control of that. And if changes are made, I'll notify Mr. Hanley of those changes within a reasonable period of time or let him know that no changes were made. And if no changes are made, a certified copy will be used for any admissible purpose, if changes were made, those changes need to get taken into account in using any copy of the transcript.

MR. HANLEY: I'm fine with that agreement, Ruth, as long as we understand that any such changes will need to be timely under the California code, and that if any such changes are made, I would be immediately notified. And by immediately I mean with 24 hours of those changes being submitted to the record court reporter.

MS. KAHN: I'm not sure what "timely" means in terms of the California code.

MR. HANLEY: Well, the time within which someone has to review and make corrections to their deposition is controlled by the code, as I recall it.

MS. KAHN: I think it's like 30 days, which I'm not sure has any applicability here since trial is set for Tuesday, but, you know, and I don't know how quickly the transcript is going to be ready. I don't know if you're expediting it or not. But --

MR. HANLEY: All that being as it may, whatever the time limitations may be, they are, you will choose to make any changes, you know, at whatever point you choose to do that. And my only request with that is that I get the immediate advisement of those changes as opposed to, you know, delaying my being advised of that.

MS. KAHN: Of course. We'll let you know within 24 hours of our

receipt of changes that the changes have been made and what they are.

MR. HANLEY: Very good.

THE WITNESS: Let me just say something. I have two changes from the previous testimony just -- should I just wait until I get today's testimony to incorporate those or send them all at once?

MR. HANLEY: No, why don't you state what the changes are on the record now, if you don't mind.

THE WITNESS: On page 74 line 16, instead of it being CLL, should be lymphoma. And the answer was -- the answer was, "the type of NHL that we talked about, the small cell" -- and then it says "CLL represents less than 10 percent," it should be, "small cell lymphoma."

MR. HANLEY: Okay.

THE WITNESS: And the other change is page 92, line 15, and it should -- instead of being pulmonary, it should be hepatic. So the sentence, instead of

reading, "I can't remember if it's directly off the aorta, but it comes eventually from the aorta into the" and it should be "hepatic artery" instead of the "pulmonary artery."

MR. HANLEY: Okay. Thank you, sir.

With that I think we can go off the record and conclude the deposition.

(Time noted: 4:00 p.m.)

I, DR. JOHN WHYSNER, Ph.D., DABT, do hereby declare under penalty of perjury that I have read the foregoing transcript; that I have made any corrections as appear noted, in ink, initialed by me, or attached hereto; that my testimony as contained herein, as corrected, is true and correct.

EXECUTED this_____day of_____, 20___, at_____, _____.

(City) (State)

_____

DR. JOHN WHYSNER, Ph.D., DABT

I N D E X

STATE OF NEW YORK)
) Ss:
COUNTY OF PUTNAM)

I, Christy J. Rockower, a Shorthand Reporter and Notary Public within and for the State of New York, do hereby certify:

That the witness whose examination is hereinbefore set forth, was duly sworn by me, and that the transcript of said examination is a true record of the testimony given by the witness.

I further certify that I am not related to any of the parties to this action by blood or marriage and that I am in no way interested in the outcome of this matter.

IN WITNESS WHEREOF, I have hereunto set my hand this 18th day of April, 2008.

_____

Christy J. Rockower