SUPERIOR COURT OF THE STATE OF CALIFORNIA

FOR THE COUNTY OF LOS ANGELES

WILLIAM MOLINA and ANGELINA MOLINA,

Plaintiffs,

v. Case No. BC 367800

SHELL OIL COMPANY, et al.,

Defendants.

DEPOSITION OF JOHN WHYSNER, M.D., Ph.D., D.A.B.T.

Washington, D.C.

Tuesday, April 1, 2008

9:15 a.m.

REPORTED BY:

JOAN V. CAIN

JOB NO. 85149

Deposition of JOHN WHYSNER, M.D., Ph.D., D.A.B.T., held at the law offices of:

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Pursuant to Notice, before Joan V. Cain, Certified Court Reporter and Notary Public in and for the District of Columbia.

A P P E A R A N C E S

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C O N T E N T S

E X H I B I T S

(Attached to the Transcript.)

E X H I B I T S C O N T I N U E D

(Attached to the Transcript.)

P R O C E E D I N G S

JOHN WHYSNER, M.D., Ph.D., D.A.B.T.

having been duly sworn, was examined and did testify as follows:

EXAMINATION BY COUNSEL FOR PLAINTIFFS

BY MR. HANLEY:

Q Good morning, sir. Could you state your name, please.

A John Whysner.

Q And can you describe for me what work you've done in the case and what opinions you've formed?

MS. KAHN: Objection, overbroad. Compound.

THE WITNESS: You mean by this work, materials I reviewed? Is that what you're talking about?

BY MR. HANLEY:

Q Sure. We're here today in the Molina matter; is that right?

A Yes.

Q What work have you done in this case and what opinions have you formed?

MS. KAHN: Objection, compound.

THE WITNESS: Well, in terms of the work that I've done, I have reviewed -- I think you have a list of the materials that were sent to me.

MS. KAHN: I do. Let the record reflect I am handing a typed list to Mr. Hanley.

THE WITNESS: I asked that this be prepared so that I could show you the materials that I reviewed.

BY MR. HANLEY:

Q This was prepared by the Steptoe office?

A This was the materials that they had sent to me, yes.

Q And you ask them to prepare such a list?

A Yes.

Q When did you ask them to do that?

A When?

Q Yes.

A A few days ago.

MS. KAHN: In lieu of bringing 11 boxes or so of material with him from New York to Washington.

MR. HANLEY: Okay. We'll mark that as Exhibit 1.

(Whysner Deposition Exhibit 1 was marked for identification and was attached to the deposition transcript.)

BY MR. HANLEY:

Q So what work did you do in this case, sir?

A So I reviewed these materials that are on

the list, which include certain MSDSs, records of the amount of materials and chemicals that were used that were provided by the defendants in the case, certain depositions of the plaintiff, co-workers, and certain -- some of the treating physicians, the medical records, and I did bring the medical records with me, and so I reviewed those.

And then I reviewed the medical and scientific literature concerning the issue of whether or not NHL has been associated or could be causally related to exposures to either benzene or to the -- the solvents that have been -- are at issue in this case. That was the work that I did. And then I was asked to form opinions about whether or not the benzene or the solvents in this case could cause NHL and in particular could have caused Mr. Molina's NHL.

I also reviewed my literature on -- that I've accumulated over the years on just in general the things that -- studies that have been done relating various either medical conditions or medications or other types of exposures to NHL.

Q Is that everything that you've done?

A As far as I can recall, yes.

Q That last item that you just itemized for me, were you -- sorry -- by saying you reviewed your

literature, would you explain that again to me? I didn't quite understand it.

A Okay. Well, as is true for some of the -- these solvents that we've talked about and for benzene, there are a whole -- a great number of things that have been studied in relationship to whether or not they are associated with NHL, and so I'll give you an example. Obesity, there are studies that say that obesity -- increased body mass index in particular is associated with NHL, and there's some studies that say that it isn't. Same is true with smoking. Same is true with exposures to herbicides. The same is true with occupations like working in the agricultural industry, the use of nonsteroidal anti-inflammatory drugs. And -- and I would say that some of these have a certain number of positive studies and then there are negative studies as well in -- in these regards.

There are also things that -- and I would classify them as possibly or unlikely, either one, to be related to -- to NHL. I don't think there's any absolute -- like for benzene or for these solvents, there's no causal relationship that could be concluded. But there are some things that are, like HIV infection, immunodeficiency syndromes, and certain drugs that cause immunodeficiency and -- and I think

the studies on hepatitis C infection are getting to the point where one could say that there appears to be a causal relationship.

So there's this whole literature that -- that looks at all these various factors, and -- and I reviewed that literature as well.

Q Did everything you've described for me this morning that you've done for this case, was it done specifically in -- with respect to this particular lawsuit?

A Yes.

Q And how much time have you spent reviewing all the materials that you've described and working on this case?

A I don't have an exact number, but I would say it comprises -- I would say my best estimate would be somewhere between 50 and 70 hours, something like that.

Q And, again, that's for this particular case, Mr. Molina's case?

A Yes.

Q Have you kept track of your time systematically so that you can bill Steptoe & Johnson for it?

A My office does that, yes. They bill Steptoe

& Johnson for it.

Q How do you record your time with your office and track your time? What's your system for doing that?

A I have time sheets that I submit to them on a monthly basis and then they submit an invoice.

Q When you say them, you mean your office?

A Yes, Washington Occupational Health Associates.

Q So you have time sheets for all of your time so far?

A Well, Washington Occupational Health does, the office does.

Q And who owns that business?

A Kenneth Chase.

Q Who else?

A Nobody.

Q Are you an employee?

A Yes.

Q Do you get a direct benefit for the time that you bill in a case like that --

MS. KAHN: Objection.

BY MR. HANLEY:

Q -- that's beyond your salary or however you're compensated?

MS. KAHN: Objection, vague and ambiguous.
THE WITNESS: Could you be more specific what you're asking about?
BY MR. HANLEY:
Q Sure. The 50 to 70 hours that you've billed so far, was that at a rate of 450 an hour?
A Five hundred -- my office bills $550 an hour.
Q Has that changed recently from 450 or sometime in the past?
A No. At least for the -- well, I don't remember. I think it changed two years ago. It used to be 500 and there was an error in the expert disclosure, and I transmitted that. It was put down as 450 and it wasn't, and I think there's been an amended -- amendment to this.
MS. KAHN: That's correct. About a week after the expert disclosure was served on February 25, 2008 our office served an errata concerning expert witness rates for three of the witnesses. I have a copy of it here.
MR. HANLEY: Okay.
BY MR. HANLEY:
Q All right. So your office has billed 550 an hour for these 50 to 70 hours you've billed so far?

A Like I say, I don't know exactly what the numbers are, but I'd say probably in that ballpark.
Q Your office has records of exactly what that number is, right, how many hours so far?
A Yes.
Q Would you be able to call at the lunch hour and ask them for the exact number of hours?
A If my -- if it's agreed to, yes, I could do that.
Q You mean if it's agreed to by whom?
A By Ms. Kahn.
Q Why would she have to agree to it?
MS. KAHN: I have no objection to your making a phone call at lunch if somebody's available and they can provide that information.
BY MR. HANLEY:
Q Why would Steptoe & Johnson have to agree to it?
A Well, I didn't see it in the notice of my deposition. Often when things like that are asked for, they're in the notice, so I don't know the legal -- legalities of the thing, and that's outside my scope.
Q At any rate, will you do that for me, call at the lunch hour and find out exactly what the number

of hours is so far?

A I will try.

Q Who will you talk to doing that?

A John Jackson.

Q And who's John Jackson?

A He's the head of the accounting department.

Q How are you compensated by Washington Occupational Health Association?

A I receive a salary and I also receive a -- I'm also a consultant, paid as a consultant on a 1099 form.

Q And describe that. Paid for as a consultant on a 1099 by whom and for what kind of work?

A Well, for the same type of work. I used to be employed by the American Health Foundation until 2002, and so I was just acting as a consultant to Washington Occupational Health, and the -- so I would be paid as a consultant. When I -- when I stopped being a -- an employee of American Health Foundation, then at some time after that I wanted to be employed by somebody for health benefits and so on and so forth. So we made an arrangement whereby my compensation would be in line with my previous consulting agreement, but I'd receive part of it as an employee.

Also, I'm asked by Washington Occupational Health to participate in some of the management duties in the company, be involved in a minor degree on other kinds of projects that I don't even bill for in terms of administrative matters and so forth. So I have -- so it was mutually beneficial for me to become also an employee of the company.

Q What is your compensation per year?

MS. KAHN: Objection. Invades the witness's right to privacy. You can find out what percentage of his income is from litigation matters. You can find out a lot of things related to compensation, but his total compensation is a matter of his privacy.

MR. HANLEY: That's not true.

MS. KAHN: File a Motion to Compel.

BY MR. HANLEY:

Q What's your compensation from Washington Occupational Health Association?

MS. KAHN: Same objections.

THE WITNESS: I don't know.

BY MR. HANLEY:

Q What do you mean you don't know?

A Well, I can't remember the number.

Q How much did you make last year?

MS. KAHN: Objection. Invades the witness's

right to privacy. He has a number of responsibilities and duties that has nothing to do with this case, nothing --

MR. HANLEY: You know, why don't you just state your legal objections.

MS. KAHN: I am.

MR. HANLEY: No, you're not.

MS. KAHN: Don't interrupt me.

MR. HANLEY: No, these are speaking objections and they're going on and they're wasting my time that I'm paying $550 an hour for and I would appreciate it if you would make your legal objection and let me move on with the deposition.

MS. KAHN: And you wasted our time when you were half an hour late.

MR. HANLEY: I was not half an hour late. I was 10 minutes late. And I apologized. We started an hour early at your request. And I was 10 minutes late. I'm sorry about that. Don't turn 10 minutes into a half an hour and don't interrupt me and obstruct me when I'm trying to conduct a deposition.

MS. KAHN: I object to your question. It invades the witness's right to privacy. It's overbroad. It's beyond the scope of his retention. It's none of your business.

MR. HANLEY: You're not Chelsea Clinton. This is my business.

MS. KAHN: It is not. It invades the witness's right to privacy.

BY MR. HANLEY:

Q How much did you make last year, sir?

MS. KAHN: Same objection.

THE WITNESS: I can't recall.

BY MR. HANLEY:

Q You can't remember how much money you made last year?

MS. KAHN: Argumentative.

BY MR. HANLEY:

Q Seriously, you can't remember how much money you made last year?

MS. KAHN: Argumentative.

THE WITNESS: I think I've answered the question.

BY MR. HANLEY:

Q Well, give me an approximation of how much money you made last year?

MS. KAHN: Objection. Invades the witness's right to privacy. Irrelevant to the issues in this lawsuit. Not reasonably calculated to lead to the discovery of admissible evidence. Not relevant to the

issue of bias or any issue on which this witness has been retained as an expert.

MR. HANLEY: Overruled.

BY MR. HANLEY:

Q Give me a ballpark of how much you made last year?

A I can't recall.

MS. KAHN: Same objections.

BY MR. HANLEY:

Q Was it over a million dollars?

MS. KAHN: Same objections.

THE WITNESS: I can't recall.

BY MR. HANLEY:

Q Was it over $5 million?

MS. KAHN: Same objections.

BY MR. HANLEY:

Q Go ahead. Was it over $5 million?

A No.

Q Was it less than a million dollars?

A Yes.

MS. KAHN: Objection. Invades the witness's right to privacy. Beyond the scope of the deposition notice. Beyond the scope of the witness's designated testimony. Irrelevant. Overbroad and not reasonably calculated to lead to the discovery of admissible

evidence.
BY MR. HANLEY:
Q Can you explain to me your thinking and memory in terms of telling me you couldn't recall whether it was over a million, but you do recall that it was under a million?
A That's just what I recall, but I can't recall more specifically.
Q Do you remember more now about what your income was last year to remember that, in fact, now you think it was under a million; whereas before when I asked you about 90 seconds ago, you couldn't remember if it was over a million?
MS. KAHN: Objection, argumentative. Not reasonably calculated to lead to the discovery of admissible evidence. Irrelevant.
THE WITNESS: I corrected my -- what I said. I remember that it was under.
BY MR. HANLEY:
Q Can you give me your -- now that you seem to have more of a memory, can you give me your best recollection or your best estimate of approximately how much it was?
MS. KAHN: Same objections.
THE WITNESS: Other than that, no.

BY MR. HANLEY:

Q So explain to me how your compensation works vis-a-vis the $550 an hour that you bill to Steptoe & Johnson or your office does for work like this?

A As I said, some of it comes -- well, some of it comes from my salary and some of it comes from a consulting agreement that I have.

Q Okay. And what's the consulting agreement? What is the consulting agreement?

A The consulting agreement is that -- I'm trying to remember exactly what it is now, but my compensation is the equivalent of -- it's a little bit over $300 an hour.

Q Does your work that you've done in this lawsuit translate into you getting paid as a consultant from Washington Occupational?

A Yeah, at that rate.

Q So, in other words, for every 550 you bill to Steptoe & Johnson, you're paid 300 of that; is that right?

A A little bit over 300. I can't remember exactly, but that's about right.

Q Okay. Is it a little over as in less than $325?

A I'm trying to remember. I can tell you it's

point -- whatever .62 is of 550.

Q So you've paid 62 percent of the money you do for your outside consulting work?

A Correct, for the consulting work with Washington Occupational Health, yes.

Q Then the other 38 percent goes to Washington Occupational?

A Yes.

Q What opinions have you formed in this case after having reviewed the materials that you did?

A Well, I was asked about two opinions, which I mentioned previously. One is whether or not either benzene or the solvents that are in this case are capable of causing NHL, and my opinion is to a reasonable degree of medical and scientific probability that they do not, and the other is whether or not Mr. Molina's exposure to either -- well, the solvents that are in this case would have contributed to his development of NHL, and my opinion is that they do not.

Q Have you formed any other opinions in this case?

A Well, to the extent there are -- there are other things in the literature that have been associated with the development of NHL such as -- I'd

say obesity is probably the one that is -- that Mr. Molina has that is -- had probably the most positive evidence. I can't -- I can't attribute his NHL to obesity either, but there are a number of other factors that have been studied and there are some positive associations in some studies, and Mr. Molina has a history of some of these other either medical conditions or use of nonsteroidal anti-inflammatory drugs, his smoking history, his obesity, anti-inflammatory drugs, and -- oh, his -- his history as a farm laborer. And, as I said, those have been associated, some studies, with -- with the development of NHL.

Q Were you just looking at some notes of some sort?

A I have a -- my review of his medical records (indicating).

Q That's a document you prepared?

A Yes.

Q Is that everything, your complete set of notes?

A On the medical records, yeah, and I have the medical records here.

Q And do you have other notes that you've prepared in the case?

A I don't believe so. These are the only ones. No. Those are the only ones.

Q So other than this one page that we'll mark as Exhibit 2 right now of your notes on the medical records, have you written anything down, other than time sheets you described already, taken any notes, produced any reports, anything like that?

A No.

Q Okay. So let me mark these notes as Exhibit 2. Are these done by your own hand typing in?

A Correct.

Q Okay.

(Whysner Deposition Exhibit 2 was marked for identification and was attached to the deposition transcript.)

BY MR. HANLEY:

Q And when did you prepare those?

A When I reviewed the medical records, which would have been -- I think most -- see, I started working on this case in April of last year, and I can't recall exactly when I got notebooks of the medical records, but I would say probably a few months after that.

Q So you've been working on the case for approximately a year almost?

A Yes.
Q What percent of your overall income comes from litigation work such as this?
A Of my income?
Q Yeah.
A I would say it's -- well, it's greater than 50 percent. It's probably -- right now it's probably somewhere between 50 and 75 percent.
Q That's a pretty big range. Can you describe your thinking or how it is that you kind of come to that range or whether you think it might be closer to one or the other, but just kind of what your thought process is on how you give that range?
A Well, because I do other consulting projects as well, but some of -- a lot of which don't have anything to do with litigation, and -- and it varies from time to time what I'm working on. So that's why it's sort of in that range.
Q What other kind of consulting work do you do that's not legally oriented?
A Well, I do some work on Superfund sites, which is oriented -- and I've been doing that for a long time, you know, work where -- or even other kinds of cleanup sites that aren't federal Superfund sites where I participate in the process of the review of

the literature and providing an opinion on whether or not certain levels of contaminants in the environment would pose a risk. In the past I've done things -- things like permitting of power plants. I've worked for, you know, putting together informational literature for certain types of issues. So there's a fairly wide range.

And my company does a lot of medical surveillance programs, and I help participate in -- in those. When they, for example, run into a toxicology issue, I provide some consulting for those kinds of projects.

Q When you do work relating -- excuse me.

When you do your consulting work that relates to Superfund sites or permitting power plants, who typically are the people you're consulting to?

A Well, they could be another consulting firm. They could be a power company. In the case of the medical surveillance work, most of our clients are the federal government, our biggest client is the FBI, and we do fitness -- you know, fitness exams for the FBI and the Army Corps of Engineers.

Q Does the work that you do relating to Superfund sites tend to be for companies involved in -- petrochemical companies or people who have put

some of the materials in the ground or wherever it is?

MS. KAHN: Objection, overbroad. Compound. Calls for a legal conclusion. Lack of foundation. Vague and ambiguous.

BY MR. HANLEY:

Q Go ahead, sir.

A Well, the one that I'm involved in right now actually has to do with the railroads, a railroad yard that primarily involves Amtrak.

Q So your work is for Amtrak?

A Ultimately, yes. The consulting firm is working for Amtrak.

Q What other Superfund sites have you worked on? How many others? Let me ask you that question.

A Well, I worked on the Paoli site. That was back in the '80s. That was a large one. The Bedford Harbor -- New Bedford Harbor.

Q Who ultimately were you working for in that?

A For Paoli. That was three railroad companies. That would have been Amtrak, Penn Central, and SEPTA, which is the Southeastern Pennsylvania Transit Authority.

Q And on these Superfund sites where railroad companies are involved, what kinds of chemicals or agents are involved for being considered for

remediation or whatever's going on with the Superfund site?

A These are primarily PCBs, polychlorinated biphenyls.

Q And what has your -- what does your work involve relating to the PCBs?

A Well, that's -- you mean related -- I've done research on PCBs and, you know, laboratory research when I was at the American Health Foundation, and I've also been involved in these Superfund sites in terms of trying to translate that information into whether or not PCBs pose a human health risk.

Q So you're consulted by the railroad companies to give an opinion regarding whether the PCBs that are in whatever state and whatever location that they are, pose a human health risk?

A Right. As part of the -- there's a process called an RI/FS, remedial investigation/feasibility study, and part of that is something called a baseline risk assessment, and my role in that is usually to -- to deal with the outcome of that risk assessment process and to review the medical literature regarding PCBs and to put the risk assessment issues into perspective.

Q Are you a risk assessment professional in

that regard?

A I would say in a limited sense. You know, I usually don't get involved in cranking out the numbers, the risk assessment numbers. That has become a process which is -- you know, involves today using a lot of computer programs and so forth in order to do that kind of work. What I primarily do is to take the outcome of that and use my knowledge as a toxicologist and the medical and scientific literature to then -- as I said, to discuss those results in terms of -- of the -- what would be called moving the risk assessment into a risk management situation in terms of what -- what needs -- what needs or does not need to be done in terms of remediation.

Q So am I -- do I -- I'll start over.

Is that to say then that you are not someone who is qualified to determine quantifiable risk assessment from those toxins?

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

BY MR. HANLEY:

Q Or are you someone who is in a position to quantify risk from a given toxic agent?

MS. KAHN: It's overbroad. It's vague. It's ambiguous.

THE WITNESS: I -- I would say it probably depends on the specific situation.

BY MR. HANLEY:

Q Can you give me a specific situation where you believe that you are qualified to do a quantitative risk assessment and a situation where in contrast you are not qualified to do a quantitative risk assessment?

A Well, I mean I can -- it depends on the complexity of the modeling that's required in order to come up with the number. There are some things like air dispersion modelling, for example, that I wouldn't even attempt to do. There are certain areas such as, you know, dispersion and sedimentation and understanding the levels of fugitive emissions from certain places that -- that are just -- you know, I don't do that. You know, it's the mathematical models and computer programs are something I've just not -- not dealt with.

If something is -- is relatively a straightforward issue, for example if you have an air level of something and you have a risk number, a concentration number and so forth, well then, I can come up with at least an upper bound risk estimate in those circumstances. And I've done that, for example,

with power plant emissions.

Q So are you saying that if it's the very simple equations associated with risk assessment, you're qualified to do that, but when it gets into the area of complex modelling, that gets beyond your area of expertise?

A In general I'd say that's -- that's correct. There's some parts of risk assessment that are relatively straightforward. There are other parts of it, like I say, where the -- where it's a whole specialty unto itself that I wouldn't be qualified to do.

MR. HANLEY: Off the record briefly.

(Discussion off the record.)

BY MR. HANLEY:

Q When I asked you to describe the opinions that you intend to offer in the case or what opinions you've formed, I think you hit on three areas: benzene and solvents not causing non-Hodgkin's lymphoma, Mr. Molina's exposure not contributing to his non-Hodgkin's lymphoma, and a discussion of the other possible considerations or associations with non-Hodgkin's lymphoma and their relationship to Mr. Molina.

First of all, did I get those three areas

correct?

A Well, the only thing that I would say is when we talk about solvents, I'm talking about the solvents that are specifically involved here. Solvents are a huge big category of different kinds of chemicals, and so we're talking about the types of petroleum-based solvents that are -- my understanding that are at issue in this case.

Q All right. And are there other areas or general topics that you have formed opinions on or understand that you'll be offering in this case?

A Not that I'm aware of. Those are -- I mean, the issues -- I mean, in terms of general -- the general approach of a toxicologist, what is toxicology, what is dose response, how one evaluates studies as a toxicologist to come up with the kinds of opinions that I have come up with, I think that I would also offer opinions regarding those sort of general areas of -- of toxicology.

MS. KAHN: I do have a copy of the expert witness designation here in case you want to refer to that.

MR. HANLEY: And are there any other areas that you want to chime in on, state of the art is another one that occurs to me might be an issue? And

I'm directing that question to counsel, essentially.

THE WITNESS: Well, I can answer. I do have some opinions about state of knowledge regarding -- especially the issue of when it was known that -- or when it was generally accepted that benzene can cause acute myelogenous leukemia.

MS. KAHN: To the extent Dr. Whysner's opinions are different from Dr. Infante's opinions, there may be some criticism, if you want to characterize it like that of Dr. Infante's opinions or how he goes about arriving at his opinions, his methodology. I think that's probably implicit in the ultimate opinions that Dr. Whysner expressed at the onset today, but I'll state it in case it's not apparent.

MR. HANLEY: Okay. I would have taken it that way.

THE WITNESS: That would include Dr. Weisenburger as well.

BY MR. HANLEY:

Q Yeah, I think that all goes into the causation issues that you described, and I'll ask you about those.

Was there some point during your time reviewing materials in this case that you had enough

information to conclude that the benzene or other petrochemical hydrocarbon solvents involved in this case did not cause or are not capable of causing non-Hodgkin's lymphoma in your opinion?

A Well, I can say that this isn't the first case I've been involved in regarding benzene or petroleum-derived solvents and non-Hodgkin's lymphoma, and so I would say that -- I mean, I re-reviewed the literature to see if there was anything that changed in that literature, but that was an opinion I had throughout my review of the material, and I was reviewing the material to confirm that opinion.

Q When you say you're reviewing the material to confirm that opinion, what do you mean exactly?

A Well, there are always new studies coming out, and, as I said before, I have been involved in a few other cases of NHL and materials like these solvents. In fact, one case that actually dealt with the same solvents, and that was the Hooper case, and so in that case I had the opinion that Mr. Hooper's NHL was not caused by these solvents.

And so when I reviewed the material for this case, I began -- I mean, that was where I started from, and I reviewed all the literature and read Dr. Infante's and Dr. Weisenburger's opinion to

confirm the fact that I still held that same general causation opinion, which would -- which was that benzene and these solvents have not been causally related to the development of NHL.

Q When you started your review in this case, when you had zero hours invested into it, you already had the opinion that benzene and other hydrochem- -- excuse me -- petrochemical hydrocarbon solvents did not cause non-Hodgkin's lymphoma; is that right?

A As I said, based upon the work that I had done in other cases leading up to that case, that had been my opinion.

Q Did you review any new literature that you had not seen before in preparation for this case?

A I think there were a couple of studies that -- that had come out and a few studies that -- that the other experts mentioned that I didn't have in my file. So I took a look at those. I can't recall specifically which ones those are right now, but I think there were a few additional studies.

Q What are the couple of new studies that had come out?

A I'd have to look at my files to see if -- to identify those.

Q Would you, please? What do we have here by

the way, these stacks of documents?

A We have lots of both literature and reviews by IARC. These are studies of either benzene or solvents and NHL, scientific studies.

Q Are these -- is this your complete library of scientific studies relating to these -- the areas that -- at issue in this case?

A Yes.

Q All of this is (indicating)?

A Well, with the exception -- this is -- this is -- now, here's an example of a new document that had been in draft before. This is the Toxicological Profile for Benzene from the Agency for Toxic Substances and Disease Registry and this has just and out in August of 2007.

Q Okay. So that's one of the things -- that's one of the new studies that has come out?

A Right. And as I -- I try to be aware of things that government organizations have summarized the data or IARC, and in general I don't rely upon review articles except where they are done by an organization like ATSDR or IARC or EPA, and so I try to keep up with -- with those.

Q And -- and why -- explain your reasoning behind your not relying on review articles generally,

but trying to keep informed of what agencies such as IARC and the EPA and the ATD --

A ATSDR.

Q -- the ATSDR conclude?

A Well, because in terms of -- I want to form my own opinion about the scientific literature rather than relying upon what somebody else has concluded about the scientific literature, in terms of, for example, an individual when they review the literature or a group of individuals. But when it is a -- a sort of what I would call an authoritative group like the IARC or the ATSDR, I try to take that into account because -- because they have made an effort in almost all cases to come up with sort of an objective and a rather complete review of all of the scientific literature in the area.

Q So is it your belief that governmental organizations like the EPA and world health organizations, IARC, when they speak as to a particular issue on agents that cause cancer, for example, that what they have to say is typically authoritative and more objective and more complete than other reviews in the peer reviewed scientific literature?

A I'd say -- I'd like to characterize it as

perhaps being more thorough, and, you know, I don't agree with every review that has been performed by these organizations, but, you know, for example, I've served on the IARC working groups that do the reviews and produce the monographs, and so I know sort of the level at which it is done, and -- and, you know, there's usually somewhere between 20 and 35 scientists from around the world involved in that process for any particular chemical, and I think it lends itself -- the process lends itself, especially IARC, to a more sort of objective and thorough review of the literature.

Q So we were talking about new studies that had come out since your previous review or that you've reviewed in the last year in relation to this case, and one was the Toxicological Profile For Benzene that you've pointed out to me. Are there any other such studies or articles?

A I'm sorry. I'm -- another one is -- came out in December of 2007, which is -- the IARC has recently reviewed -- re-reviewed painting, in other words, the occupation of being a painter. They had previously concluded that it was -- carcinogenicity was associated with that and focused on lung cancer and bladder cancer, and of course there are various

kinds of solvents that are used in painting. And so they recently reviewed that and there's a summary -- they haven't published the monograph, but there's a summary been published in -- I think this is called Lancet Oncology from December 2007, and apparently they've reconfirmed that -- that those are the two issues again that come up with being a painter. One is lung cancer. The working group concludes there is sufficient evidence in humans that occupational exposures of painter causes cancer of the lung and urinary bladder.

And they said additionally there was limited evidence in humans, namely on the basis of maternal exposure that painting is associated with childhood leukemia, and those are apparently the only positive findings.

And I know that they've looked at the NHL issue because I have the monograph from the previous time. So apparently they did not conclude that it was associated with non-Hodgkin's lymphoma. So that just came out recently.

Q That's what you take from that, is that they have concluded that it's not associated with non-Hodgkin's lymphoma?

A Well, yes, because they stated what they

found was sufficient and what was limited, and I know that the way that IARC works and so I know that they have reviewed -- I'm almost positive they reviewed those issues because they reviewed them previously, and did not conclude that it was -- that that information supported carcinogenicity in humans.

Q Is there anything else that you've reviewed that falls into the category of the new studies that are always coming out? Hold that one aside so we can mark it.

A Okay. Well, there are some other articles that had to do with the literature regarding other risk factors that I also found. There were some new studies.

There was actually a study that when I -- I looked at this issue, which was published actually a couple years ago in 2006, but it was -- I was looking up Dr. Weisenburger and this is an article that he did on agricultural pesticide use with the subgroup of non-Hodgkin's lymphoma, which would correspond to the follicular non-Hodgkin's lymphoma, this t(14,18). So I found that.

Q Okay. If that's one of the new ones, why don't you keep that out.

A There's an article on occupational risk

factors of non-Hodgkin's lymphoma by Richardson that looked primarily at occupations and said the risk was elevated among agricultural workers, blacksmiths, machine tool operators, and they found that -- and they went through some chemical exposures, but none that were benzene or any of these solvents.

There was a new article in 2007 about U.S. veterans with hepatitis C. I believe these were all since --

Q All since Hooper?

A Well, certainly since Hooper. There was also a fairly large meta-analysis about the issue of smoking and non-Hodgkin's lymphoma that came out which -- well, here's another one. Hepatitis C. I did find a review article by Alexander, which I -- I looked at primarily because of other -- all the other risk factors, not necessarily the -- these solvents or the benzene exposure, because they had -- some extraneous material attached to it, which was our seminar schedule at Columbia for some reason, but that's not part of that. So there was that review article. But I think I actually knew about this before the last case that I was involved in.

But there was -- oh, there's one about nutrients contributing to one carbon metabolism and

risk of non-Hodgkin's lymphoma subtext which had to basically do with folic acid.

Oh, here's the one I was thinking of. The non-Hodgkin's lymphoma, and I think this one was on obesity. It was a pooled meta-analysis and another article on hepatitis C.

Q If these fall into the new articles that you've reviewed, then go ahead and keep them in that same stack of those so that we keep all those together, if you don't mind.

A Okay. That's about it.

Q So would this then -- if we take this U.S. Department of Health and Human Services' document that you've given me in the large binder --

A By the way, I had seen the draft of that before, but I think the draft was a 2005 document.

Q And if we take this document, the -- and these articles that you've handed to me now, these are all the things that you've reviewed within the last year that would be new materials in terms of the articles?

A Right. Like I said, I reviewed everything -- oh, and there's also the 2007 -- this comes out every year. This is the TLV booklet. I just looked at this to see if there had been any

changes in any of these, and the Alexander review I think I actually was aware of this before the last time I reviewed the literature. So I don't know if I'd consider that absolutely new.

But, as I said, I re-reviewed everything again for this case and in relationship to Dr. Weisenburger's and Dr. Infante's deposition as well.

Q All right. I understand you read both of their depositions given in this case; is that right?

A Correct.

Q And with the caveat that you've just given me, then these materials are the -- the new materials that you've read and what you were referring to when you said there were new studies always coming out within the last year?

A Correct.

Q All right. So let's --

A At least to the best of my recollection, and I might have missed one here somewhere.

Q Okay, all right. So let me mark this (indicating) as Exhibit 3 and we'll mark then this collection of -- did I get the whole collection or do you still have some of them there?

A No.

Q Because you had reviewed them before?
A Yeah.
MR. HANLEY: We'll mark this collection as Exhibit 4 that will be -- so that we have Exhibit 3 and Exhibit 4 are the new studies and articles that you've read as you just described and were going through them, right?
THE WITNESS: I'd say in the last six months.
MS. KAHN: Plus the TLV booklet that he identified.
MR. HANLEY: Plus the TLV booklet that I'm not going to mark.
MS. KAHN: Is there, for clarification, a difference, Dr. Whysner, between things you reviewed in relation to the issues in this case and things that you rely on?
THE WITNESS: Well, I'm always reading things and if they're not really relevant to his case, then they wouldn't be in these files. I mean, that's the only distinction that I can think of. But in terms of everything that I could find related to studies of NHL and either benzene or the types of solvents that we're dealing with here or even solvents in general and including petroleum worker studies,

this is, as far as I can remember, the new materials. Like I said, there may be one or two other papers mixed in here that I haven't found.

MS. KAHN: Great.

(Whysner Deposition Exhibit 3 & Exhibit 4 were marked for identification and were attached to the deposition transcript.)

THE WITNESS: Can I take a break?

MR. HANLEY: Of course.

(Recessed at 10:20 a.m.)

(Reconvened at 10:25 a.m.)

BY MR. HANLEY:

Q This document that we marked as Exhibit 3, did it -- how has it informed your opinions? Does it support them or contrast them or --

A I have a -- the one page here that --

Q Okay.

A So I can find it. They reviewed the literature and basically they said that -- that the possible association -- they talked about the Hayes study -- has been suggested by the Hayes study, but then they say, however, other studies didn't support that. The association found in the Chinese study, the Hayes study -- they're talking about benzene versus -- the profile on benzene. So in general what they say

is that their overall conclusion -- my read is that it supports my conclusion regarding the NHL and the literature.

Q What page are you looking at exactly?

A 101.

Q And what exactly do they say on that page that leads you to the conclusion that it is consistent with your opinion? And just so we're clear, you're talking about the opinion that benzene is demonstrated to cause AML but no other forms of the blood cancers or lymphatic cancers?

A Well, specifically they're talking about benzene and NHL in this case is what we're -- what we're talking about. I didn't really focus on actually what they had said about some of the other blood cancers.

Q So what is it exactly on page 101 that leads you to say that you believe that their position is consistent with your position that benzene is not demonstrated to cause NHL or non-Hodgkin's lymphoma?

A Well, this paragraph says, although the results of the NCI/CAPM Chinese study, Hayes, et al., indicated a possible association between exposure to benzene and NHL, other cohort mortality studies found no significant increases in NHL mortality, and they go

on to quote the Rinsky study, the Collins study, the Bloemen, B-l-o-e-m-e-n, et al., study, and the meta-analysis of the 26 cohorts of petroleum workers by Wong and Raabe, and then they go on to say, "Furthermore, case controlled studies provides no indication of association between benzene exposure and risk of NHL," and then they quote a number of studies.

Q And do you take that as a conclusive statement that benzene has not been demonstrated to cause non-Hodgkin's lymphoma?

A Well, I mean their analysis of those studies is consistent with my analysis of those studies and my conclusion.

Q That's not exactly the question I asked you. My question is, do you take this paragraph to say that their conclusion is that benzene is not demonstrated to cause non-Hodgkin's lymphoma?

A As I said, I'm -- I wasn't looking at it for a conclusion. I was looking at it for a -- their analysis, and what they have said is there is one study that suggests an association, but then all of the other studies did not find those associations in their analysis, and I'm not going to try to put words in their mouth in terms of any of their conclusions. But what I'm saying is this is consistent with my

conclusion.

Q But are they making the conclusion, are they stating that benzene is not demonstrated to cause non-Hodgkin's lymphoma?

A I'd have to go through the rest of the document to see if they say that anywhere else. I don't really know if they say that anywhere else in the document.

Q Do you consider this to be an authoritative and reliable text as to the issues it addresses?

A Well --

MS. KAHN: Overbroad.

THE WITNESS: Yeah, I don't know that I would consider it to be -- I mean, it's addressing a lot of benzene -- different things on benzene, different aspects of it, but I would say it is -- it is one that I would consider along with EPA and IARC in terms of groups that have reviewed this literature, and I would consider it to be certainly something that I would take into account in terms of how they review the literature and what they said about these individual studies.

BY MR. HANLEY:

Q In terms of its place in the medical and scientific literature that exists on the issues

relevant to this case, as to the issues that are addressed in this document, do you consider it to be a reliable source of information and authoritative as to those issues it addresses?

A Well, for example, you asked about the issue of conclusion. ATSDR's role is not to classify carcinogens like EPA does, like IARC does, like the NTP does. So, you know, they -- they don't -- as far as I know at least, they don't really, you know, make that kind of overall determination in terms of a particular issue.

But what I -- what I can say is that as far as it goes, I think that they have reviewed those studies accurately, with the possible exception of the fact that I'm aware that the -- that the Rinsky -- I mean, the people who did the Chinese study actually published a subsequent article after that Hayes study in 2000, where they said that their study did not necessarily indicate an association between NHL and benzene, that it might have been due to other chemical exposures, and they said that it was because of the wide differences in -- in the findings between different industries in their study, and the chemical industry was the highest.

And so this was a Hayes publication from

2000, and so they -- they sort of came to a little different conclusion than the -- the ATSDR just says that there's a suggested association, but the actual authors of the study said that that association might have been confounded by exposures to other chemicals. So I would have a little difference of -- you know, in terms of the way that they even reviewed the -- the Chinese study.

Q Okay. So you've just described some ways in which you disagree with the analysis by the authors of Exhibit 3, correct?

A Well, only in the sense that they didn't mention that as a confounding -- I mean, they correctly said that there was an association described in that study, but there weren't associations found in these other studies, but I just would point out that they -- at least in that part of the document, they didn't consider the -- the qualifications that the authors of the Chinese study put on those findings.

Q When you refer to the ATSDR, you're referring to Exhibit 3, correct?

A Correct.

Q Is -- and I guess I'm just going to press for a yes or no, if it's possible, because I really want to ask this question about all of the literature

that you have here today. So let me ask it with respect to all of this literature, what we've marked as Exhibit 3, what we've marked as Exhibit 4, and what we will mark -- because I want to mark your library of these materials -- are all of these authoritative and reliable texts as to the issues that they address? Whether or not you agree with everything that's in them, in terms of what they are, are they authoritative? Are they reliable sources of information to draw upon in forming conclusions relating to the issues in this case?

MS. KAHN: The question as framed is overbroad, compound, lacking in founding, vague, and ambiguous.

THE WITNESS: I don't think I can answer that question for everything. I think if we go through the studies, I can -- I can point to things that -- you know, I mean that are relevant and are not relevant to this case, for example, and to some issues regarding whether or not they really show an association or don't show an association. Because, for example, the solvent literature, when we talk about, quote, the solvent literature, I think it becomes very complicated because there are two things here.

First of all, this is just a chapter heading from Cassarett, C-a-s-s-a-r-e-t-t, and Doull, D-o-u-l-l, and so these are various solvents, and so in some cases these studies didn't identify what solvents they were talking about, which would have included chemicals that aren't in this case. So I can't really rely upon them to tell us anything about these particular solvents because it includes things like chlorinated solvents, which, my understanding, are not in this case. Or it may include things like glycol ethers or alcohols, okay. So --

BY MR. HANLEY:

Q Well, let me ask you this. Is that next book a reliable and authoritative text for the issues that are addressed in that textbook?

A Well, I think it's -- I mean, that's kind of a hard question to answer because I don't -- I rely upon my own review of the scientific literature, okay. I go through study by study. So I can't embrace and say, gee, this book got everything right or the ATSDR got everything right from my standpoint. Because I reviewed all of this literature, and, you know, it has to be reviewed. It has to be reviewed according to the -- you know, the Bradford Hill type of analysis to see whether or not things are consistent, to see

whether or not the associations are strong, to see whether or not they've taken into account what are certain obvious confounding variables.

So, like I say, I think that it's a -- it's an enterprise that requires a lot of detail, and so I can't make overbroad generalizations, even about a book like Cassarett and Doull. My purpose for showing you this list was just to illustrate the fact that people throw around the word "solvents" and all that means is -- all a solvent is, is something that can dissolve something. It doesn't really have any toxicological meaning because they're all different chemicals.

Q Now, on -- you referred me to page 101 of Exhibit 3, which is part of chapter 3 of that document entitled "Health Effects," correct?

A Yes.

Q And this document is about benzene, correct?

A Yes.

Q And the section that is included in page 101 actually begins on page 97 under the subtitle of Cancer, correct?

A I'd have to see the document.

Yes.

Q And the way this document is organized,

because these governmental agencies tend to write these documents in a certain format, first of all, correct?

A Yes.

Q They kind of follow a certain formula for the way that they present their information?

A Yes.

Q And the way this section is organized is similar to the way that many of these such governmental documents are organized in that under the section in the Cancer they first state some general principles that are not really subject to much dispute and state their conclusions, and then they go through and summarize what is out in the literature and tend to summarize the different positions of the different people who have published that scientific literature on that subject; is that correct?

A Well, they do it different ways. They don't -- I mean, for example, IARC doesn't provide any summary at the beginning. Their whole summary is at the very end of the document, where they have section -- I think it's usually Section 5 called the Overall Evaluation. So there -- there -- they tend to be different.

Q But in this particular document, the way

they've done it is on page 97, they start their discussion of benzene and cancer and in the first paragraph is where they set forth their -- the conclusions of these authors; is that right?

A I'm not sure that that's the case here. I'd have to go through the document and --

Q Why don't you read the first paragraph and see if you wouldn't agree with that? That would be the bottom of page 97 and the top of 98, just that first paragraph. Is that not a conclusion, the conclusion of the authors based on their review of the literature that they're just about to then go through and itemize in some detail?

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

THE WITNESS: I think it pretty much says what you were reading beforehand, except it -- except this statement sort of lumps together NHL and multiple myeloma, and it's talking about the one -- one suggested evidence, which is the Hayes study, for NHL, and then I believe when they talk about multiple myeloma, they're referring to the Rinsky 1987 study. So they've made a statement here that -- but all they have talked about is the one study which we mentioned previously, but they didn't talk about all the ones

that didn't show associations with -- with NHL.
BY MR. HANLEY:
Q My question is, do they not summarize in the first paragraph under Cancer their general conclusions relating to benzene and cancer and then from there go on over the next several pages, including page 101 that you quoted from and then beyond, describe the literature in terms of what the authors in that literature have concluded in their various studies?
A No. I think I would characterize it more as an introduction rather than a conclusion.
Q And they introduced the subject of cancer saying that benzene clearly causes AML and ANLL and -- as -- and the studies provide suggested evidence of it causing non-Hodgkin's lymphoma and multiple myeloma?
MS. KAHN: Objection. The document speaks for itself.
THE WITNESS: Well, I think what they have said is -- is -- I'll put it this way in terms of introduction, which I would interpret to say, okay, there are several studies that show that at least at a sufficient dose benzene is capable of -- is associated with AML, and then they say, and there are some suggestions in the literature of multiple myeloma and NHL and they quote the two studies that provide those

suggestions and then they go on later to discuss them in detail and say there is this one study that provides some suggested association, but then there are all these other studies that do not.

So, again, I would see this as kind of a -- as not a conclusory statement, but a statement that's indicating why they're -- why they're reviewing this.

BY MR. HANLEY:

Q Do you agree with everything in that first paragraph under Cancer?

A With -- with the caveats that I just mentioned, which says that -- that this is not really their analysis. This is an introductory paragraph.

Q Do you agree with each statement made in that paragraph?

A I -- I don't know. I would have to go back and take a look at -- well, no. Because, as I mentioned before, I don't believe that the authors then when they subsequently published in 2000, they also said that the evidence that they had found linking -- associating benzene with NHL needed to be -- I can -- I can read it to you what they said. They said that it might have been confounded by exposures to other chemicals. And so I don't even believe that these Hayes studies, according to what

the authors concluded later, provide suggestive evidence of an association.

Q Which statements in that first paragraph do you not agree with?

A What I just said.

Q Well, why don't you go through sentence by sentence, since it's only one paragraph, read the sentence and tell me whether or not you agree with that sentence? If you could start with the first sentence and then stop after that sentence and tell me whether or not you agree or disagree with that sentence and then go through each sentence by sentence of that first paragraph.

A Okay. Well, I -- I can tell you --

Q You know, I really want to try to get this deposition done today, if it's going to be possible. I'm not sure at this point. But so that we keep it clear and so that we go by question and answer and hopefully only answer my question, not that I'll ever cut you off, but I just want to make it clear and go through this clearly.

So the first sentence says epidemiological studies and case reports provide clear evidence of a causal relationship between occupational exposure to benzene and benzene-containing solvents and the

occurrence of acute nonlymphocytic leukemia, ANLL, particularly the myeloid cell type acute myelogenous leukemia, AML.

Do you agree with that statement?

A I don't know what case reports they're talking about. I agree with the rest of the statement, but I'm not sure that -- case reports I don't think can ever provide clear evidence because they're just case reports. I believe that the epidemiological studies and the ones that they've mentioned, in particular the Rinsky study, provides clear evidence of a causal relationship.

Q Okay. So you disagree with the part about case reports, but otherwise agree with it?

A I don't know what they're talking about. I mean, I -- I would -- you know, in general I would say case reports can't provide clear evidence, but I would have to say that I would have to see what case reports -- I would have to know what case reports they're referring to that are included in this string of references, and I just don't know what they are.

Q It next says some studies also provide suggestive evidence of association -- excuse me -- associations between benzene exposure and non-Hodgkin's lymphoma, NHL, and multiple myeloma.

Do you agree or disagree with that sentence?

A First of all, I don't agree that the Hayes study even provides suggestive evidence because of the chemical confounders that they later identified could have been responsible for those results. The issue of multiple myeloma, I've not reviewed and so I don't have an opinion on that.

Q So you don't know whether or not benzene causes multiple myeloma?

MS. KAHN: Objection, misstates the witness's testimony. Lack of foundation.

THE WITNESS: What I said is I have not reviewed it recently for this purpose, and so I don't know if I agree with that study regarding the Rinsky study.

BY MR. HANLEY:

Q Okay. But let me ask you another question. Do you have an opinion as to whether or not benzene can cause multiple myeloma in humans?

MS. KAHN: Objection. Beyond the scope of what the witness was asked to look into for this case. Beyond the scope of the expert witness designation.

THE WITNESS: I would have to review the literature first in order to define my opinion on that.

BY MR. HANLEY:

Q Okay. So as you sit here today, you don't know whether or not benzene causes multiple myeloma?

A I think I've just answered that question.

Q I just want to be clear on it. Are you saying as you sit here today you do not know whether or not benzene causes multiple myeloma?

MS. KAHN: Objection, asked and answered.

THE WITNESS: Well, what I can tell you is it's my impression that the literature does not reach a -- is not strong enough to provide a causal association. That's what I remember from reviewing the literature in the past, but as I said right now sitting here, I haven't really reviewed the literature recently regarding that issue.

BY MR. HANLEY:

Q Okay. And is that to say then that as you sit here today you don't know whether or not it does because you haven't done a recent -- done recent research on that subject?

MS. KAHN: Objection, misstates the witness's testimony. Argumentative. Asked and answered.

THE WITNESS: Could you repeat the previous question and answer?

BY MR. HANLEY:

Q No, she will not repeat the previous question and answer. I am entitled to ask you whether that means, as you sit here today, you do not know whether or not benzene causes multiple myeloma.

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

BY MR. HANLEY:

Q That's a yes-or-no question. You can answer it yes or no and explain it however you like.

MS. KAHN: It's been done.

BY MR. HANLEY:

Q You know, either as you sit here today you know or as you sit here today you don't know?

MS. KAHN: Do you have anything to add over what you already said, Dr. Whysner?

MR. HANLEY: I ask the questions today, not Ms. Kahn.

THE WITNESS: Fine. I don't have anything to add. What I can tell you is that the last time I reviewed that literature -- as I mentioned, with each case, I re-review the literature, okay, to see if my opinion has changed because of anything that has come out in the literature. My recollection is the last time I reviewed this literature there was not a causal

link between multiple myeloma and benzene exposure.
BY MR. HANLEY:
Q You'll agree that biological plausibility is an important consideration in determining whether or not an agent can cause a particular cancer. Would you agree with that?
MS. KAHN: Objection, overbroad. Vague and ambiguous.
THE WITNESS: Depends on what you mean by biological plausibility.
BY MR. HANLEY:
Q Well, what -- well, explain that.
A No. I'm asking you what you mean by biological plausibility.
Q All right. You don't understand the question?
A I don't know what you mean by biological plausibility.
Q All right. Biological plausibility is a Bradford Hill criteria for attribution, correct?
A I've got the paper right here. I can read it.
Q Is that correct? Do they not refer to biological plausibility as being a criteria for causal inference?

A No. Bradford Hill specifically says that what he is laying out here are not criteria.

Q Biological plausibility is one of the considerations one needs to consider when determining whether or not a toxin is capable of causing a particular cancer, correct?

A Well, it's one of the things that one -- one needs to look at. It says -- I can read you what it says. It says, "It would be helpful if the causation we suspect is biologically plausible, but this is a feature I am convinced we cannot demand. What is biologically plausible depends upon the biological knowledge of the day." And it also depends on what people mean by biological plausibility.

Q Would you not agree that whether or not benzene is capable of causing NHL is informed in part by our understanding of whether or not benzene is capable of causing AML, ANLL, or multiple myeloma?

MS. KAHN: Objection. Overbroad. Vague. Ambiguous. Causing it in humans? Causing it in animals? It's overbroad.

THE WITNESS: Could you reread the question.

(The reporter read the record as requested.)

THE WITNESS: I don't agree with -- with

that because these are all specific individual diseases.

BY MR. HANLEY:

Q Isn't the biological plausibility of benzene being able to cause one informed by our understanding of the mechanism, for example, of the toxin reaching the target organ?

MS. KAHN: Objection, overbroad. Compound. Vague and ambiguous and lack of foundation.

THE WITNESS: Not necessarily.

BY MR. HANLEY:

Q Explain that.

A Well, because in general chemicals reach a lot of organs, and these organs don't necessarily respond. For example, it used to be thought that you could take a look at a chemical and say, okay, what -- this is going back decades now. People used to think that you could look at the bodily distribution of a chemical and get some idea from the bodily distribution where the chemical would have its effect, but it turns out that it's a poor predictor. Now with one exception and that is -- well, a couple of exceptions. There's the blood brain barrier that prevents some chemicals from reaching the brain, but in general it's not a good predictor.

Q I didn't ask about it being a predictor. You don't even get to the point where you consider whether or not a toxin can cause a particular cancer unless the toxin actually is demonstrated to reach the target organ, correct?

A Well, I could -- in general you might be correct, but, you know, our analytical capabilities are so good these days that it's -- you know, it's hard not to measure things in almost all organs. So I could say if you had a good analytical technique and you didn't find the chemical in a particular organ system of the body, it would make a difference in terms of your thinking of whether or not it was biologically plausible that it would have a toxic effect on that particular organ.

Q And isn't whether or not benzene can cause AML or ANLL important to the consideration of the biological plausibility of whether or not it can cause NHL?

MS. KAHN: Objection, overbroad. Compound. Vague and ambiguous.

THE WITNESS: Well, I -- I -- you know, it's hard for me to imagine that. For example, the diseases that you mentioned, AML, ANLL arise in the bone marrow. The issue -- what we're dealing with

here today, follicular NHL, by definition is a B-cell lymphoma that is thought to arise from the follicle in the lymph nodes, which are not part of the bone marrow. So I don't -- I mean even in terms of tissue distribution, which is what we were talking about before, the fact that something arises in the bone marrow versus something arising in the lymph nodes, I don't see where the -- where you draw the connection.

BY MR. HANLEY:

Q All right. The next sentence states, "The epidemiological studies are generally limited by confounding chemical exposures and methodological problems, including inadequate or lack of exposure monitoring and low statistical power due to small numbers of cases, but a consistent excess risk of leukemia across studies indicates that benzene is the causal factor."

Do you agree or disagree with that sentence?

A I think I mostly agree with it. I think there has been some exposure monitoring that certainly went into the dose reconstruction of the Rinsky study, but, you know, you always want to have more if you can, and -- but I think -- I think that in terms of the Rinsky '87 study, I think that for leukemia or AML, they have reasonably good dose response

information, and so in general I would agree with that statement, but I would qualify it to say we're talking about acute myelogenous leukemia in terms of benzene being a causal factor.

Q The authors don't so limit it, do they?

A Well, I think that people tend to sometimes talk about leukemia, but when they actually mean a particular kind of leukemia, it says -- next sentence says many of the earlier studies are additionally limited by a lack of information on leukemia types other than AML, and so -- some of the studies lump all leukemias together. Some of the studies have distinguished AML from other leukemias and some of the associations were found with leukemia, but these seem to have been due to acute myelogenous leukemia.

Q You're not suggesting that these authors in this document have not been careful about their choice of words and their specific use of leukemia in a general sense, are you?

A Well, I think in the Rinsky study, in the 1987 study there were --

Q Sir, I asked you about these authors of this document in Exhibit 3, in their use of leukemia. I didn't ask you about the authors in the Rinsky study.

A Well, no, I agree with what they said.

There's a consistent excess risk of leukemia across studies, but as I said, I think that the underlying -- which they discuss in some sense in the next sentence, but I think the underlying cause for that excess risk is an increase in acute myelogenous leukemia.

Q The next sentence reads, "Many of the earlier studies are additionally limited by a lack of information on leukemia cell types other than AML because leukemia used to be considered a single diagnostic category for epidemiological purposes due in part to historical nomenclature, small numbers of death by cell type, and unavailability of cell type specific rates for comparison."

Do you agree with that statement?

A I agree with the statement. I -- I'm -- you know, I'm not as familiar maybe with some of the people who wrote this document about the unavailability of cell type rates for comparison, but I don't see anything wrong with that statement.

Q Now, going back to the issue of biological plausibility, it is true that although leukemias develop in the bone marrow and lymphomas develop in the lymphatics, that in a number of these cancers between leukemia and lymphoma, we're talking about the same cells developing the cancer or becoming

cancerous. It's just a matter of whether it's diagnosed in the bone marrow or in the lymphatics, correct?

MS. KAHN: Objection.

THE WITNESS: No.

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

THE WITNESS: No.

BY MR. HANLEY:

Q How is that not correct?

A Well, you said diagnosed and it depends where they're diagnosed, but in actuality we're talking about where they develop, not -- the issue is you made it sound as though they just happened to be diagnosed in the lymphatics, but we're talking about a different biological process.

Q How is it a different biological process?

A Well, because the cancer has -- has arisen in a different organ through different mechanisms that are specific to it arising in that cell type once it has -- it in the environment of the -- of the lymph node.

Q When we're talking about the various lymphomas and in comparison to leukemias, we're often talking about the same types of cells getting cancer,

and it's a matter of whether that cancer arises in the bone marrow or in the lymphatics, which determines whether it's a leukemia or lymphoma, even though it may be exactly the same cells, correct, or the same cell types?

MS. KAHN: Objection, compound. Overbroad.

THE WITNESS: Well, you said two things. You said -- I don't know what you mean by cell types and exactly the same cells.

BY MR. HANLEY:

Q Not the same cells, the same cell types.

MS. KAHN: Same objections.

THE WITNESS: Well, I mean, they're obviously not the same cell types or you would have developed a leukemia rather than a lymphoma. I mean, the -- there is something -- these cells are different when they are in the lymph node. They have already changed into lymphatic cells rather than being bone marrow cells. I mean, it's -- it's -- I don't know quite how to explain it beyond that. You know, they just -- they're arising in different places.

Now, there is one type of NHL, which is not what we're talking about in this case, where there is some similarity between the cells in the bone marrow and the -- and the type of lymphoma, and that's CLL

and small-cell lymphoma, chronic lymphocytic leukemia and small-cell lymphoma, but as far as I know that's the only type of lymphoma that is related to a B-cell leukemia.

BY MR. HANLEY:

Q And other than CLL -- well, let me ask you this first. CLL and small-cell lymphoma involve a cancer of the same cell type, a B-cell, one arising in the bone marrow, the other arising in the lymphatics; is that correct?

A Well, I don't know that it's entirely clear where the -- whether or not the cells arise in the bone marrow and then are in the lymph system, found in the lymph system or whether -- I mean, I think you're -- in terms -- in terms of that, I'm not sure that that's really known.

Q So --

A I know from a classification system, the World Health Organization is -- considers the CLL and the small-cell non-Hodgkin's lymphoma to be on -- what they call on a continuum. In other words, it's maybe different stages of the same disease, but in terms of the kind of lymphoma that we're talking about, it is a distinct different type of lymphoma that has not been related to a leukemia.

Q So CLL and small-cell lymphoma are essentially the same disease, correct?

A Well, I mean, they have different clinical manifestations. The -- and this is not my area of expertise. The treatment may be somewhat different as well. I'm not -- I'm not really sure. I don't -- that's something I don't give an opinion on. That's something I'm not that knowledgeable about.

But all I know is that there are enough similarities that it is considered to be, if not the same disease, but have enough similarities that they believe -- that there is a belief that they are related to each other.

Q Are there any other leukemias and lymphomas that have any such sort of the same kind of relationship?

A Not that I'm aware of.

Q And can you go through and give me a list of the different kinds of leukemia and which cell type is involved in getting cancerous and do the same thing for lymphomas?

MS. KAHN: I'm going to object. That's beyond the scope of this witness's expert witness designation and beyond the scope of what his assignment was in this case. I think another witness,

John Bennett, who's going to be deposed later this week, will address all that.

BY MR. HANLEY:

Q Go ahead.

A Well, there are myeloid cells, okay, and these are the -- both acute myelogenous leukemia and CML arise from the myeloid cell line. There's a difference though between these two kinds of leukemia in that -- in that CML is almost always related to a very specific translocation defect in the genetic material that was discovered 30 -- at least 30 years ago called the Philadelphia chromosome, and this is a translocation between chromosomes 9 and 22, and so it's a very distinct type of leukemia in terms of that and also in terms of its clinical diagnosis and course. It can have an acute phase, but it's still a different disease from acute myelogenous leukemia.

Acute myelogenous leukemia has been found to have a wide variety of different kinds of chromosomal defects. There isn't any one type of chromosomal defect that is associated with that. The -- and then there are lymphocytic leukemias and they can either be CLL or acute lymphocytic leukemia. Those both come from the lymph cell line and there in general can be -- well, in general you can say that the ALL is

almost always a childhood leukemia. That's the common form of leukemia that one sees in children is acute lymphocytic leukemia.

I think you asked about the -- did you ask me about lymphomas also?

Q Yes. Yes. Thank you.

A So lymphomas, the major forms of lymphomas are diffuse and follicular, and then there are, you know, the -- I think the type that we talked about before that's related to CLL forms a much smaller percentage, less than 10 percent of NHLs, and then there are --

Q I'm sorry. That last statement again was the CLLs?

A The type of NHL that we talked about, the small-cell CLL, represents less than 10 percent of NHLs, okay. But more common ones are the diffuse and the follicular. And we're talking about B-cell lymphomas. Now, there can also be T-cell lymphomas. They're less common. And those -- there are a number of different types of those T-cell lymphomas.

Now getting back to the B-cell lymphomas, you also have lymphomas that arise in parts of the gastrointestinal tract. You can have ones that arise in the skin and -- you know, for example, the one that

arises in the gastrointestinal tract has been associated with the bacterial Helicobacter infection. There's a Burkitt's lymphoma which is found predominantly in Africa and that's been associated with Epstein-Barr virus infection. So I think those are the major -- major general types of lymphomas.

Q Okay. So that -- so the NHLs include the CLLs and either a diffuse B-cell lymphoma or a follicular B-cell lymphoma or diffuse T-cell lymphoma or a follicular T-cell lymphoma?

A Well, the T-cell -- now you're getting a little beyond what I've reviewed for this case and I don't really -- I don't remember what all the T-cell lymphomas are, whether you would characterize them as diffuse or follicular.

Q Do any of the leukemias involve B-cells?

A The CLL is a B-cell leukemia and, like I said, it's related to this one subtype of NHL, the small-cell -- small B-cell, you know, lymphoma.

Q Would you agree that if benzene or other petrochemical hydrocarbon solvents are demonstrated to cause any lymphomas, that it informs the biological plausibility of whether or not it can cause other types of lymphomas?

MS. KAHN: Overbroad. Objection, compound,

undefined, vague, and ambiguous.

THE WITNESS: With all of these kinds of biological plausibilities, the devil is all in the detail, and so I could answer your question if I knew that benzene caused NHL and how it causes it, okay, but without knowing that and without -- my opinion is that it doesn't cause NHL, I can't really answer that question.

BY MR. HANLEY:

Q Would you agree that benzene and other petrochemical hydrocarbon solvents reach the B-cells in the human lymphatic system?

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

THE WITNESS: I don't know specifically. I've never -- I've never seen any measurements of that.

BY MR. HANLEY:

Q Well, regardless of seeing measurements of it, do you know one way or the other whether benzene and other petrochemical hydrocarbon solvents ever come into contact with lymphatic B-cells?

MS. KAHN: Objection. The question is overbroad, compound, vague, and ambiguous.

THE WITNESS: As I said, I have never -- I'd

have to speculate on this in order to answer the question because I've never seen any actual measurements.

BY MR. HANLEY:

Q Well, are you saying you've never seen any evidence in the scientific literature one way or the other as to whether or not benzene or other petrochemical hydrocarbon solvents come into contact with B-cells in the human lymphatic systems?

MS. KAHN: Same objections.

THE WITNESS: I actually have never looked at that literature. I don't even know if people have actually analyzed lymph node cells -- lymph nodes to see if it contains those constituents.

BY MR. HANLEY:

Q Do you know whether such literature exists?

MS. KAHN: Objection, lack of foundation.

THE WITNESS: You know, I -- you know, people do do distribution studies of -- of chemicals. They look at that, and, you know, like I said for this matter, I've focused on the epidemiology studies to see whether or not it causes -- there has been a causal relationship with non-Hodgkin's lymphoma, but not looked at the chemical -- what you're asking for is the chemical analysis of benzene or these chemicals

in lymph nodes, and I have not looked at that.
BY MR. HANLEY:
Q Actually what I asked you was, do you know whether or not such literature exists?
MS. KAHN: If he hasn't looked into it, I don't know if he could know it exists. It lacks foundation.
BY MR. HANLEY:
Q Then the answer may be I don't know whether or not such literature exists, but that is -- whatever the answer is, that's the question I'm asking. Do you know whether or not such literature exists?
A I believe it might.
Q Thank you. Do you know of any reason why or why not benzene or other hydrocarbon petrochemical solvents would reach the B-cells in the human lymphatic system?
MS. KAHN: I object to the question that it's overbroad. It's vague and ambiguous. If you ask as to benzene, I don't have an objection, but when you ask benzene or petrochemicals and you don't identify the petrochemicals and the witness has referred you to a document that identifies a number of categories of items that could be considered petrochemicals, the question is overbroad, and frankly I don't think it

can be answered.

MR. HANLEY: Counsel, I'm not deposing you, if that's okay.

THE WITNESS: Could you repeat the question.

(The reporter read the record as requested.)

MS. KAHN: The question is also beyond the scope of this witness's designation, beyond the scope of his assignment, and beyond the scope of his retention.

THE WITNESS: I don't know any reason why they would or would not.

BY MR. HANLEY:

Q I'm not sure I understand that answer. Are you saying you don't -- well, what is your understanding of where these chemicals go in the human body when they're breathed in or dermatologically absorbed?

A Well, I think I mentioned to you before, I mean in general I think the statement could be made that most chemicals are distributed widely throughout the body.

Q Okay. So is that to say that --

MS. KAHN: Let him finish, please.

BY MR. HANLEY:

Q I don't mean to cut you off. I'm sorry. I thought you were finished.

A And so I would -- as I said, it's -- it's the measurement techniques have gotten so good that we can demonstrate most things in most places, and so it would surprise me if they didn't.

Q In other words, you presume they do. You haven't reviewed the literature on it, but presumably they do; is that what you're saying?

MS. KAHN: Objection. That's vague and ambiguous. Assume they do what?

MR. HANLEY: I didn't say assume. I said presume.

BY MR. HANLEY:

Q Go ahead.

A Well, I think based on my general knowledge of chemicals and distribution patterns of chemicals, I would think that these would fall into the category where we would expect to find them distributed throughout the body.

Q Just based on our understanding of what our body does with toxins, wouldn't it be fair to presume that if a molecule of benzene gets into the lungs, that that gets taken and -- and presumably filtered through the lymphatics, attacked by macrophages, and

removed including through the lymphatics?

A Well, I think it's low levels of most chemicals are metabolized by the body so that they are excreted, and so what you're asking me about is -- there's sort of a race to a finish line, if you will. For example, the body is trying to take these chemicals and make them into something else so that they can be excreted, and there are a number of enzyme systems to do that.

So I would think that in general, at least going up to a certain level of a chemical, that the body is pretty efficient at trying to eliminate it so that it doesn't reach all parts of the body. Again, I guess the answer is it depends on a lot of things. It depends on dose. It depends on route of exposure for a particular chemical, and I haven't really looked into that specific question for -- in this -- in this matter.

Q Do you know whether or not the lymphatics is part of the human body's defense mechanisms against benzene or other petrochemical hydrocarbon solvents?

MS. KAHN: Objection. The question as framed is overbroad, compound, vague, and ambiguous.

THE WITNESS: By defense you mean in terms of metabolizing them and getting rid of them?

BY MR. HANLEY:

Q Yes, part of our body's defense mechanism.

A The lymphatic system -- I would say in general no. The lymphatic system is primarily aimed at dealing with infectious agents.

MR. HANLEY: All right. If we might, I'd like to take a quick break.

(Recessed at 11:28 a.m.)

(Reconvened at 11:36 a.m.)

BY MR. HANLEY:

Q Do you have an opinion on whether or not the benzene, toluene, and other petrochemical hydrocarbon solvents that Mr. Molina was exposed to at his work at Firestone reached the target organ for his non-Hodgkin's lymphoma cancer?

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

THE WITNESS: Well, I mean, I really didn't look -- I mean, I looked into the solvents that he was using or presumably using, at least that were furnished, and I really don't know whether or not the levels of those chemicals would have been sufficient that they would have reached -- you're talking about the lymphatic system -- to any -- you know, I mean I

don't know how to say like significant degree or any measurable amount. I wouldn't have any way of knowing that.

BY MR. HANLEY:

Q So you don't have an opinion one way or the other whether or not the benzene, toluene, and other petrochemical hydrocarbon solvents that he was exposed to at the Salinas Firestone facility reached the target organ for his non-Hodgkin's lymphoma?

MS. KAHN: Objection, asked and answered. Same objections that I just stated.

THE WITNESS: Like I said, there's too much missing information here for me to have an opinion on that.

BY MR. HANLEY:

Q What information is missing in order to inform an opinion on such an issue?

A Well, first of all, I would need to know the -- I mean, I would have to come up with a number in terms of the amount that he was exposed to, to know whether or not that actually he received a dose and then I would have to know whether or not that dose was sufficient to overcome the metabolic capacity of his particular system that would allow the chemicals to be distributed to the lymphatic system.

Q Can you point me to some particular information that would allow you have to have an opinion on that one way or the other?

A I haven't been asked to look into that in this case and so I couldn't. I didn't -- I didn't focus on that particular issue.

Q Which particular issue? The levels at which he was exposed to these petrochemical hydrocarbon solvents?

A Well, that, but also the issue of the metabolic capability and how much it takes in order to overcome an individual's metabolic capacity to -- to excrete the chemical.

Q If someone's exposed to 7 ppm years of benzene over a working career, is that enough to overcome someone's metabolic capacity to handle those toxins without reaching the lymphatics?

MS. KAHN: Objection. The question presents an incomplete hypothetical, misstates the evidence, lacks foundation, it's overbroad, compound, vague, and undefined.

THE WITNESS: Could you repeat the question.

(The reporter read the record as requested.)

THE WITNESS: That's not something that I've

looked at.
BY MR. HANLEY:
Q So the answer is you don't know?
A For that -- no. For that particular number, I mean, there are too many -- too many unknowns so that I wouldn't -- I wouldn't have an opinion on that.
Q What more would you need to know in order to have an opinion?
A Well, I'd have to know Mr. Molina's particular metabolic capacity for metabolizing benzene.
Q Anything else?
A And the -- well, the -- I mean, the -- certainly the amount of benzene that was in the atmosphere at various points in time rather than a -- maybe a general ppm year number.
Q Explain that.
A Well, I'd want to know whether or not you're assuming that there is a -- if you take -- you said a 7 ppm year, and if you're dividing that by 17 years and then dividing that by 8 hours per day five days a week, that number actually is -- so we're talking about a number that would be below 1 ppm in the atmosphere. Depends whether or not there's a -- depends what the levels are in the atmosphere rather

than the number of ppm years is what I'm trying to say.

Q Would situations of higher ppm peak exposures make someone more inclined to have their metabolic capacity for dealing with that toxin overcome?

MS. KAHN: Objection. Overbroad, compound --

MR. HANLEY: Do you want to just reserve all of your objections?

MS. KAHN: -- incomplete hypothetical.

MR. HANLEY: Do you want to just have a continuing every objection in the world is reserved and you can raise it at any time so that I don't have to be interrupted today?

MS. KAHN: I'll think about that.

MR. HANLEY: Okay, please do.

MS. KAHN: I'll think about that.

BY MR. HANLEY:

Q Go ahead.

A Okay. Well, as I said, it depends upon how you divide it. If you say that there's a very low level of exposure that's occurring all the time, you would use one -- I mean, I can't answer the question either way because I'd have to -- I'd have to have the

inputs in the model in order to come up with the kind of answer you're talking about. This is an area called pharmacokinetics, and I don't really consider myself to be an expert in pharmacokinetics. It's a -- as a matter of fact, I don't teach that class at Columbia.

Q What's pharmacokinetics?

A Pharmacokinetics is understanding the exact quantitative distribution of a chemical through the body, you know, its absorption, metabolism, distribution, blood levels at various points in time, and I don't really consider myself to be, in general, an expert in that area.

Q Is one of the things you're saying, that the higher the ppm concentration in the air that someone is breathing, a higher concentration of ppm concentration for benzene, toluene, or other petrochemical hydrocarbon solvents, the higher that ppm intensity, the more likely that person's metabolic capacity is to be overcome and, therefore, allow the toxin to reach the target organ?

MS. KAHN: Objection. It's overbroad, compound, vague, and ambiguous, and I don't want to have a standing objection. I'd rather object to specific questions as they come up with the hope that

maybe you'll reframe the question if you know what my objection is to the form.

THE WITNESS: Well, I mean in general higher exposure levels result in -- in greater dosages of any chemical. Now, when we get to benzene and other chemicals, it depends, because sometimes even high -- short-term high exposure levels, there's a certain capacity in the system that they don't -- that certain systems are not saturated, and so they have not -- there is the ability of the body to absorb a certain amount of material and retain it over a short period of time. It's a -- it's something that I really haven't had a chance to look into for this -- that particular question.

BY MR. HANLEY:

Q What are the mechanisms by which someone has any metabolic capacity for exposure to benzene, toluene, or other petrochemical hydrocarbon solvents without any adverse health effects?

MS. KAHN: Objection. The question is overbroad, compound, vague, and ambiguous.

THE WITNESS: In general we call it metabolism or detoxification, and benzene is metabolized through the addition of alcohol groups to the -- to the benzene molecule and then those alcohol

groups can get conjugated to water-soluble things like glucuronidation, which then leads to urinary excretion.

BY MR. HANLEY:

Q So how exactly is benzene or other petrochemical hydrocarbon solvents metabolized by the human body?

A Well, each one is done differently. What I've talked about is related primarily to benzene, and to be quite honest sitting here, if you're talking about toluene or xylene, these other ones, I could refer to the IARC monograph to see if they say, but sitting here I can't remember the exact metabolic pathway for those chemicals.

Q Okay. What's the exact metabolic pathway for benzene?

A I just said what it was.

Q The liver?

A The liver, adding of hydroxyl groups to it, creating things like phenol, hydroquinone, benzene trial, and then those get conjugated to -- there are a number of different water-soluble groups that then -- then facilitate their excretion in the -- in the urine.

Q Is everything you just described things that

occur while the benzene is in the liver?

MS. KAHN: Objection, vague and ambiguous.

THE WITNESS: Primarily in the liver, but these metabolic systems are also present in other parts of the body.

BY MR. HANLEY:

Q How does the benzene that is encountered through the air by breathing into someone's lungs get from those capillaries in the lung to the liver to be excreted out through urine?

A Well, primarily it would be through -- there are two ways. One would be hepatic artery and the other way would be the circulation that goes through the gastrointestinal tract that winds up in the portal system of the liver.

Q What's the hepatic artery?

A Hepatic artery is a branch of the aorta that specifically distributes blood to the liver.

Q So take me from the capillaries in the lung through the -- to the toilet, if you will, through these two different pathways. Describe these two different pathways where the benzene goes from the time it's inhaled to the time it's excreted.

You talked about the hepatic artery and the GI tract, two different pathways. So I'm asking you

to take me from the lungs and through however it gets to the hepatic artery to the liver and then excreted and then take me from the lungs to however it gets to the GI tract and to wherever it gets to from there.

MS. KAHN: Starting with the lungs, not the nasal passages -- you're assuming it's in the body to start?

BY MR. HANLEY:

Q Well let me ask you. Benzene's inhaled, right? I mean, it's one of the ways in which --

A Right.

Q And the other way in which a person suffers exposure to benzene is through dermal absorption, right?

A It's possible, mm-hmm.

Q All right. So starting with benzene that's inhaled, could you take me from that inhalation through the human body through those two pathways and how it gets from those two pathways into the urine to be excreted, and if those are the only two pathways, and if there are others, I'll ask you to describe those as well, and we'll talk about dermal absorption later and how it gets to where it goes.

A Well, the pulmonary vein -- first of all, it goes through the alveolus, and then in the alveolus

there are capillaries, and then from the capillaries in the alveolus, capillaries get into different parts of the pulmonary venous drainage.

Q So it goes from the alveoli into the oxygen and carbon dioxide diffusion mechanism into the blood; is that right?

A In essence, it's following the same pathway as oxygen is.

Q So it's getting into the blood and then from the blood in the lungs going where?

A Pulmonary vein. Pulmonary vein goes into the heart. The heart pumps the blood to the aorta, and then the hepatic artery is branched off of the -- I can't remember if it's directly off the aorta but it comes eventually from the aorta into the pulmonary artery. Then there are other arteries that distribute the blood to the gastrointestinal tract and --

Q Okay. So just to stay in our -- we're in the blood and the blood is coursing through the body and one of the areas blood is going is to the -- through the hepatic artery and that leads to the deliver?

A Yes.

Q And is that oxygenated blood that's going to the liver?

A Yes.
Q And does all of our blood in our body -- I guess it goes to different places and some of the blood goes to the liver and some of the blood goes to the brain and everywhere else, correct?
A Correct.
Q And does the benzene then make its way to the liver to get metabolized there just in the -- I mean, there's no way in which the benzene somehow gets channeled to that hepatic artery. That hepatic artery just carries whatever benzene happens to be in the body's blood at any given point, correct?
A In general, yeah.
Q And that same blood that has presumably the same amount of benzene coursing throughout is also going to all of the other parts of the body that need oxygenated blood; is that right?
A That's correct.
Q So the benzene can go to the brain and your fingers and back to your lungs and everywhere where oxygenated blood is being used by the body, correct?
A Yes.
Q Then the benzene when it gets to the liver, the body has a way of dealing with such toxins in the liver, and you described that briefly before; is that

right?

A Well, yeah. I mean, when it gets to the -- as I mentioned, there is -- also the benzene in the blood, because benzene would be relatively fat soluble -- you know, the reason that it's -- one of the reasons these solvents are used is because they dissolve things that are fatty, in essence, grease or whatever. It will be sequestered somewhat in the blood into certain areas like -- that are usually transporting fats.

So then when it gets to the liver, the liver will have a mechanism of extracting the lipids, because the liver is also responsible for metabolizing lipids that are absorbed in the gastrointestinal tract. Because, remember, the other way that the blood gets to the liver is all of the blood that's distributed to the gastrointestinal tract is then drained through the portal vein system directly to the liver. So the liver actually receives most of its blood supply from the drainage of the gastrointestinal tract rather than from the hepatic artery.

In the liver then, the liver is responsible for extracting the lipid fraction also from the blood, which in this case would contain the benzene because it's lipid soluble, and so it would do that and at the

same time it's metabolizing the fats that are in that system, and then there's an enzyme called cytochrome P450 and it's a heme protein and that's the enzyme that's responsible for activating oxygen that then reacts with benzene and forms these hydroxyl groups, which are -- contain oxygen, OH groups.

And then as I said, those will be conjugated with things like glucuronidation or might get conjugated with other things that are then responsible for taking -- making this very water soluble so that it can be excreted in the kidney.

Q You may have said it, but I didn't quite follow, but tell me how the benzene gets from the lungs to the GI tract?

A Same way. Pulmonary vein.

Q Through the blood?

A Through the blood, and then it gets distributed to the intestines, stomach through arteries and then the veins that drain those same organs, the stomach and the intestines, there's a very specific drainage system called the portal system, and instead of then that blood going back to the heart, it goes directly to the liver.

Q Would it be fair to say, given this understood pathway of benzene in the human body, that

there's no question it gets to the target organs for leukemia by virtue of it gets into the blood and, therefore, necessarily going through bone marrow?

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

BY MR. HANLEY:

Q Or is that a misunderstanding of the anatomy?

A Could you repeat the question.

(The reporter read the record as requested.)

THE WITNESS: I don't understand the question.

BY MR. HANLEY:

Q Does that blood that we talked about that would have benzene in it also in the normal course go to the target organ of leukemia -- the target organs for leukemia such as bone marrow?

A Well, don't forget it's not the benzene. It's the benzene metabolites that are thought to cause leukemia: hydroquinone, phenol. It's not really well understood, but it's not the benzene itself that is believed to be involved in leukemia formation. So that's not really answering your question. I'm sorry. But I think you're heading in that direction.

Q Okay, I appreciate the clarification. Is the process which turns the benzene into the agent understood to cause these benzene-related cancers something that happens in the liver?

MS. KAHN: Objection. Overbroad, compound, vague, and ambiguous, benzene-related cancers.

THE WITNESS: If we're talking about acute myelogenous leukemia, which is the only thing that I know that's been -- been -- that is -- like I say, the -- the metabolism in the liver leading to the formation of these hydroxylated metabolites and those hydroxylating metabolites being distributed to the bone marrow is the -- is -- it's not really understood how that all occurs, but the metabolites are the ones that are thought to be responsible for the development of leukemia or acute myelogenous leukemia.

BY MR. HANLEY:

Q The AML?

A Right.

Q And are those metabolites created in the liver as part of its metabolizing benzene or is that --

A Yes.

Q It is?

A It is.

Q And somehow they get from the liver back up to the bone marrow and that mechanism is not well understood?

A Well, the mechanism that's not well understood is -- here's the problem. I mean, hydroquinone itself is not know -- or phenol, known to be leukemogens. There's scientific information that leads one to believe that you have to have this metabolism in order to -- in order to cause leukemia, but there -- there are amazingly a lot of unknowns about the fundamental biology of how AML is produced by the benzene metabolites.

Q When you talk about a person's metabolic capacity for -- for handling benzene, you're talking about a person having a capacity to be exposed to a certain level or a certain amount of benzene without any appreciable risk for developing a cancer or any other -- like an AML or any other benzene-related disease; is that right?

A That's correct.

Q And in the description that you've given me of where benzene goes in the body and what happens, it sounds like a lot goes on in the liver and are you saying that then some of that is excreted and the benzene or the metabolites that it creates are

excreted and then some of that is not excreted and goes to other parts of the body that can cause DNA damage in certain cells?

MS. KAHN: Objection, overbroad. Compound. Vague.

THE WITNESS: Well, as I mentioned, the only one that we know about is the metabolites going to the bone marrow causing DNA damage in the bone marrow and producing acute myelogenous leukemia, and there have been various hypotheses about how that happens, and I have written a review article on the -- on the genotoxicity of benzene and, you know, I mean my working hypothesis is that it inhibits an enzyme called Topoisomerase 2, T-o-p-o-i-s-o-m-e-r-a-s-e 2 -- we can just call it Topo 2 -- and that seems to be one of the -- I think the best explanation for the kind of genotoxicity that we see with benzene.

BY MR. HANLEY:

Q And that's a paper you've written that was funded by the API?

A Yes.

Q There was a -- oh, so before I move on from this area, describe for me your understanding of the mechanism by which benzene gets into the human body through dermal absorption and how the body expresses

any metabolic capacity it may have for exposure by that mechanism?

A Well, I mean, benzene I think has some limited ability to get through the stratum corneum which is the barrier -- the barrier part of the skin, you know, the cornified epithelium, and from there it would go to the venous system and be distributed just as we mentioned from the -- from the lungs.

Q Okay. And so by the venous system, you mean the veins taking blood back to the lungs?

A Yes.

Q All right. Another statement in Exhibit 3 that I want to ask you if you agree or disagree with is the next sentence, where we left off before. The first sentence of the next paragraph, where they say, "Two series of studies on workers exposed to benzene in Ohio, the Pliofilm study, and China, the NCI/CAPM study, have yielded particularly strong data on the leukemogenic potential of benzene and are summarized below."

Do you agree with that statement that those studies have yielded particularly strong data on the leukemogenic potential of benzene?

A Well, I certainly would say with the Rinsky study and I think also the Chinese study, but there

are -- I believe there are -- well, I think that's a fair statement, mm-hmm.

Q When the scientific literature talks about the leukemogenic propensity of benzene, do you understand that to be referring specifically only to its ability to cause leukemias and to the exclusion of non-Hodgkin's lymphoma, or is that phrase leukemogenic propensities sometimes used to describe the class of cancers that would include leukemias and lymphomas?

A Well, I know in the case of the Rinsky study they didn't find any increase in non-Hodgkin's lymphoma in the study. So I would interpret this statement to be only talking about leukemia and, from my perspective, specifically about acute myelogenous leukemia.

Q Okay. That's a slightly different question. My question was about the use of that phrase in scientific literature generally, including the context of the sentence we're reading again now, but let me ask both questions. In the sense that this is used -- in the context that this is used here, do you believe that it's referring specifically to just leukemias or specifically only to AML leukemia, or to a broader class of cancers and lymphatics and leukemias?

A Well, it certainly wouldn't mean the broader

category. It would not -- because they would -- if -- if -- they would not have said leukemogenic because that -- if you were talking about lymphomas, you would use a different terminology.

Q Okay. They are talking about leukemias as a general class including not only AML but other leukemias as well in the context of that sentence, correct?

MS. KAHN: Objection. It calls for speculation as to how the authors are using the term and what the authors meant.

THE WITNESS: I don't know how they're using the term, to be quite honest.

BY MR. HANLEY:

Q Well, doesn't the Pliofilm study, at least according to those authors, demonstrate benzene as a cause of different kinds of leukemias?

A No. I mean what they did in the Pliofilm study, they lumped all of them together, but it really doesn't tell you which of the leukemias was responsible for the increase. I think other people have reanalyzed that data later and found that the increase was due to an increase in acute myelogenous leukemia, but they just didn't -- they just didn't differentiate.

Q All right.

MS. KAHN: Let me know when you're ready to switch topics because that might be a good time for a lunch break.

MR. HANLEY: Yeah, that would be now.

(Whereupon, at 12:14 p.m., the above-entitled matter was recessed until 1:05 p.m.)

CONTINUED EXAMINATION BY COUNSEL FOR PLAINTIFFS BY MR. HANLEY:

Q Sir, did you have a chance to talk to your office about the number of hours that you billed in this case so far?

A Well, John Jackson was out to lunch, but I left him a message, and I told him to give me a callback on my phone.

Q If you get that call, will you tell me so we can interrupt our proceedings and take it?

A When we interrupt, I'll check my voice mail and I'll get back to him.

Q All right. Now, how was the time broken down, would you say, in terms of the different materials you reviewed? Was essentially all of that 50 to 70 hours reviewing either depositions or medical articles or medical records?

A Well, those plus the -- I did review the

MSDSs and some of the other records. There were depositions that had been taken of toxicologists and employees of the various defendants also, and I reviewed those to a certain extent, although I wasn't really asked to give an opinion on the -- on the issue of exposure in this case.

Q Okay. So how was your time broken out in terms of by percentage or approximately how did it break down among the different documents?

A Well, let me just modify one thing. When I say exposure, I did look to see what kinds of chemicals were involved, but not in terms of any kind of a quantitative thing of exposure. Boy, that would be really hard for me to estimate.

I would say at least half of it would be re-going through my literature and making sure I had everything, and I'd say the other half would probably be spent reading other people's depositions and reviewing the medical records, but I don't know that I could get more specific than that.

Q All right. What was it that you were reviewing any of these materials for in order to draw any information that might help you form or change an opinion?

And the reason I'm asking it, because I'm

getting a little bit of a blank look back, is since you already knew -- I assume you knew from the very beginning when you were retained in the case that this was a case of non-Hodgkin's lymphoma. You already had the opinion that benzene and other petrochemical hydrocarbon solvents do not cause non-Hodgkin's lymphoma. So my question is, what was the point of those next 50 to 70 hours of review of these documents for?

A I don't know it was just 50 to 70 hours for these documents, but I sort of expected that you would be wanting to go through these studies with me, and so I tried to make it efficient in terms of -- you see I have different colored tabs on them so that -- you know, I use these studies, you know, in cases that aren't non-Hodgkin's lymphoma. They might be AML cases. They might be CML cases.

I've been involved in CML cases, and so the last time I used this -- and this literature is not all just dedicated to non-Hodgkin's lymphoma. So I've got to go through all the literature that I have to reorient it to an NHL case, and then I went through, and in the last NHL case that I was involved in, only benzene was at issue. So now we have other of these solvents, petroleum-based solvents and including

toluene I understood was at issue particularly. So I had to go back through the studies, and, as I said, I put little tabs on the tables that support my opinion so that we could go through them efficiently, and that takes a lot of time.

Q So is that fair to say then, that the 50 to 70 hours that you put in this case were not so much for you to be able to determine what your opinion was, but more for preparation for testifying in the case, whether it be at deposition now or at trial in the case later?

A I'd say that's true, yeah, that I have to -- I have to prepare and I wanted to bring all the documents that were relevant in this case, and so I had to go through everything.

Q Because when you started your review, one of the first things that you learned about the case, I take it, from the attorneys was that this was a non-Hodgkin's lymphoma case?

A Yes, I believe that they told me that at the time, yes.

Q And with just that much information, you knew that your opinion would be no amount of benzene or other petrochemical hydrocarbon solvents was going to increase his risk for contracting that disease,

regardless of how much he was exposed to, for example?

MS. KAHN: Objection, overbroad and compound.

THE WITNESS: Well, I mean, as I said, at the beginning of my case, I had reviewed the literature previously, and, you know, my opinion, of course, is based upon what's available in the scientific literature. For example, there have been certain exposures that have been described in the scientific literature, and at least his exposure didn't exceed any of those that were in the scientific literature. So to the extent that the -- that his exposures fall within the -- the extent that have been studied in the scientific literature, yes, that's true.

BY MR. HANLEY:

Q Did you review the exposure assessment reports of Dr. Nicas and Dr. Reiss in this case?

A Yes. I had more time to do Nicas. I just saw Reiss's earlier today.

Q Would you agree that both of those gentlemen document significant exposures to benzene, toluene, and other petrochemical hydrocarbon solvents?

MS. KAHN: Objection. The question is overbroad and it's vague and it's ambiguous. It's not

clear what the word "significant" means.

THE WITNESS: Well, that would be my -- that was what I was going to say. From a -- from a medical standpoint, I don't -- I wouldn't characterize the exposures as medically significant. That would be in relationship to the known threshold limit values and -- and also in terms of the other things that have been reported in the scientific literature.

But I know -- I recognize that both of them have come up with part per million year estimates of benzene exposure -- no, I'm sorry. Reiss came up with part per million year estimates of benzene and toluene, and I think total hydrocarbon exposures, to the extent that he could, based on both the numbers that Dr. Nicas had generated and upon his understanding of Mr. Molina's testimony.

BY MR. HANLEY:

Q What in your mind is a medically significant exposure to benzene or other petrochemical hydrocarbon solvents?

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

THE WITNESS: Can you just tell me which one -- I'll give them to you individually. Okay. Well, okay. I'm basing this, for example, on the ACGH

TLVs. So -- so, for example, for rubber solvent, they have a time weighted average threshold limit value of 400 parts per million, and that's based upon eye and ear irritation --

BY MR. HANLEY:

Q Let me interrupt you. Is what you're saying or about to say in a much longer way that in order for an exposure to be medically significant in your opinion, it should exceed the ACGIH TLVs currently in place?

A In general that's true because ACGIH base their TLVs upon the medical literature and occupational medical literature in particular, where they documented that at certain levels, certain effects begin to be seen, and they try to set those numbers below those levels.

Q So your bottom line for what is medically significant regarding solvent exposures is whatever the ACGIH TLVs are published?

A Well, that would be a starting point, but, you know, I wasn't asked to in this case so I didn't really review the literature. I wasn't asked to comment on anything other than the question of non-Hodgkin's lymphoma and these various solvents. So I mean I brought the ACGH TLV as a starting point. If

you were going to ask me for certain effects that don't have to do with non-Hodgkin's lymphoma for benzene or for these other petrochemical solvents, as you characterized them, I would have to do a literature review and verify for my own mind that the ACGH TLV numbers would be correct.

Q To what extent exactly do you rely upon the ACGIH TLVs as being an indicator of whether a significant -- whether an exposure to a solvent would be medically significant or not? To what extent do you rely on the ACGIH TLVs for that kind of analysis?

A Well, I just said I used them as a starting point. I mean, if it's something that is of particular concern either in a case or regarding an individual who's in one of our medical surveillance programs or something like that, I go -- I look at the TLVs, but then I usually go beyond that and look at the documentation for the TLV and also do my own independent literature survey. So I don't -- it's just part of the materials that I rely upon.

Q Was Mr. Molina exposed in a medically significant way to benzene in his work at the Salinas Firestone facility?

A No.

Q Why not?

A Well, because -- well, again, let me say that I'm primarily focusing on the issue of -- of any of the scientific literature that we have available regarding non-Hodgkin's lymphoma and any of these chemicals, and my view of the literature is that they are not causally related to NHL, and there are certain exposure levels that have been studied in -- in that literature, and so certainly Mr. Molina did not exceed those exposure levels that have been studied.

Q What does that mean?

A Okay. Well, let me give you an example.

Q No, I don't want an example. I want to know what that means.

MS. KAHN: I think he's got to give you an example to explain what that means.

MR. HANLEY: I don't know that --

THE WITNESS: Certain studies -- okay. Certain studies have studied, for example, benzene exposure up to including, I believe, hundreds of parts per million of exposure, hundreds of parts per million years of exposure, okay. And my conclusion is that that hasn't been related to non-Hodgkin's lymphoma.

Now, if indeed it were the fact that Mr. Molina was exposed to way more than that, then I wouldn't have any scientific literature to base an

opinion upon.

Does that make sense?

BY MR. HANLEY:

Q Okay, I understand. Thank you.

A Okay.

Q If Mr. Molina was exposed to ten times the amount of benzene that is described by Dr. Nicas, would that be a medically significant exposure to benzene as relates to non-Hodgkin's lymphoma, in your opinion?

A No.

Q If Mr. Molina was exposed to benzene to the extent described by Dr. Nicas, would that be a medically significant exposure to benzene as relates to his risk for developing AML?

A No.

Q Why not?

A Well, because my -- my review of the literature says that you have to -- you have to get somewhere into the range of 40 to 200 part per million years before we see an increased risk for acute myelogenous leukemia.

Q What scientific literature do you rely upon for that proposition of a 40 to 200 ppm years threshold for benzene causing AML?

MS. KAHN: I'm going to object that that's beyond the scope of the witness's retention in this case and beyond the scope of the expert witness disclosure.

BY MR. HANLEY:

Q Go ahead.

A The primary one is Rinsky 1987.

Q Anything else?

A Well, the other literature I think is -- there's another article by Constantini that finds a similar -- and the Chinese study doesn't get -- I'm -- let me just take a quick look at the literature here. Constantini is -- et al. was 2003. And the Chinese study I'm pretty sure doesn't go into statistically significant increase until above 40, but to be quite honest, I really -- I didn't bring all of the necessary AML dose response literature with me because, well, this is a case of non-Hodgkin's lymphoma.

Q Is it fair to say that there was nothing that you were going to review in this case when you started it that was going to convince you that Mr. Molina's non-Hodgkin's lymphoma was caused by his exposure to benzene, toluene, or other petrochemical hydrocarbon solvents at the Salinas Firestone plant,

including the medical articles that you'd seen before and the medical records that you reviewed, and the deposition testimony that you read?

MS. KAHN: Objection to the form of the question.

THE WITNESS: Well, I think I mentioned the fact that I did re-look for any additional information to see whether or not it would affect my opinion, but my opinion was not changed by that review of both the existing literature, the new literature, or the testimony of any of the plaintiffs' experts.

BY MR. HANLEY:

Q I mean, you knew what you were going to see in the medical literature because you had reviewed it before, correct?

A Well, except for the ones that I looked -- I looked for anything new that was available, and I -- I -- as I -- as I mentioned before, I have been -- I have been involved in other cases that included similar exposures and non-Hodgkin's lymphoma, and so I had a general idea of what my opinion was, but I went through -- I went through the literature review to reconfirm that.

Q Going through the -- the only information that would possibly change the opinion you had would

have to be the new literature that had been published within the last year from now, correct?

MS. KAHN: Objection. Lacks foundation and calls for speculation.

THE WITNESS: Well, as I -- you know, I also mentioned that there were -- there were one or two articles that were mentioned by Dr. Infante and Weisenburger, and I can't remember which ones they are now offhand, that I re-looked at because I didn't remember having reviewed those before, that they had mentioned. So I really couldn't characterize them as all having been published in the last year.

BY MR. HANLEY:

Q You knew when you went through the medical records that there was nothing you were going to see in there that was going to impact your opinion regarding whether or not his non-Hodgkin's lymphoma was caused by benzene or other petrochemical hydrocarbon solvents, correct?

MS. KAHN: Objection. Lacks foundation and calls for speculation. It assumes that the non-Hodgkin's lymphoma was the correct diagnosis and no other interpretation could be derived from the medical records.

THE WITNESS: Well, I have to confirm for

myself that the medical record indicates non-Hodgkin's lymphoma being diagnosed, and -- and I also, as I mentioned, looked for other things because the literature on other risk factors is really, I think, changing a lot more than the benzene and petroleum solvent literature in the sense that there are a lot more new studies going out. So I was also reviewing his medical records to see about any other possible risk factors.

BY MR. HANLEY:

Q Do you have an opinion that Mr. Molina has any risk factor for non-Hodgkin's lymphoma to a reasonable degree of medical certainty?

A I think the reason for his non-Hodgkin's lymphoma is not known.

Q So it's an idiopathic cancer?

A Correct. There are -- there are some things that -- that -- other risk factors that have been studied that I would put in the -- more in the possible category that he has, but none that have been -- you know, my opinion none that have been clearly causally related to non-Hodgkin's lymphoma.

Q Okay. So you believe that his cancer is idiopathic, correct?

A The cause is not -- yeah, idiopathic,

meaning the cause is not -- we don't know the cause.

Q And you do not believe that Mr. Molina's weight or any obesity that he may have was a substantial factor in contributing to his risk for developing non-Hodgkin's lymphoma; is that correct?

A All I can say is this. It may be, but I can't say that for certain.

Q You cannot say to a reasonable degree of medical certainty that this weight was a substantial factor contributing to his risks for developing non-Hodgkin's lymphoma; is that correct?

A That's correct.

Q And the same would be true for any exposure to pesticides that he may have had in his life?

A That's correct.

Q Would the same be true for any noninflammatory steroid drugs that he may have ever taken in his life?

A Yes.

Q Would the same be true for any HIV infection that he may or may not have ever had in his life?

A Well, if he had had HIV, I would say it was involved, but I didn't see anything in the medical record that indicated to me that he had HIV infection.

Q Did you see anything in the medical records

to lead you to believe that he had been taking any drugs which caused immunodeficiency in him?

A No.

Q And so you don't believe that drugs which cause an immunodeficiency are a substantial factor in contributing to his risks for developing non-Hodgkin's lymphoma to a reasonable degree of medical certainty, correct?

A That's correct.

Q And would the same be true for any other potential or possible causes of non-Hodgkin's lymphoma currently being investigated as causes of non-Hodgkin's lymphoma?

A Yes.

Q You said that the medical articles that you brought here have tabs on them, and did I understand you to say that the color of the tabs have some particular significance?

A For the most part, especially this -- there's one file here that includes -- let me see if this is the one. Yeah, especially this file where there are lots of studies that deal with certain solvents as well as benzene. The benzene is in green and the solvents are in yellow, and the red markers might be something different, like occupation or -- or

something like that.

Q And that collection of articles that you're holding in your hand, what is the -- what is that collection? Why is it separated from the other collections of articles?

A As I said, these are case-control studies, almost all of them, and they're ones in which -- in which because they're case-control studies, meaning they're studies of non-Hodgkin's lymphoma and risk factors for non-Hodgkin's, examination of various either exposures or occupations or -- or whatever. Often these studies will deal with both benzene and other solvents, including chlorinated solvents sometimes. So that's why they're in this pile.

I have a -- I have a separate collection of cohort studies that have to do with benzene exposures, and cohorts means you have a group of workers who were exposed to benzene and you're comparing them to either people who are not exposed or to the general population in terms of cancer rates.

And then I have another collection that deals specifically -- well, those are the two main collection of articles, although I have another collection of people who were involved in -- in -- mostly cohort studies that had to with exposures to

toluene or to -- or these were people who were, like, painters, like some underlying studies that looked at painters who were exposed to toluene and other kinds of solvents that might be at issue in this case.

And then I have another folder that has to do with -- with the question of chemical interactions, which has to do with benzene threshold effects and how certain chemicals like toluene can actually inhibit the metabolism of benzene to the -- I mentioned that benzene has to be metabolized in order to produce the metabolites that we think are associated with producing AML, and concurrent benzene -- toluene exposure inhibits that kind of metabolism. So it's been found to reduce the, for example, chromosomal aberrations that are produced by benzene. So I have a file on that.

Q Does that mean having exposure to benzene and toluene at the same time increases the adverse impact of the benzene, or are you saying it's the other way around?

A It's the other way around. It appears to decrease it.

Q Did I interrupt you? I'm sorry.

A Well, I don't know if you wanted me to describe all of these files.

Q I do. Let me ask you this about them generally. Have you attempted to include a comprehensive collection of articles, whether they support your opinions or are contrary to your opinions?

A Yes, I have. I have.

Q And is that included here, what we have here today?

A Yes. And I can't guarantee I have every single article, but I have all the articles I could find.

Q All right. And it's your intention to include whatever helps you or doesn't help you; is that right?

A Yes.

Q All right. Let's go through each collection and have you briefly describe what that particular collection is and why it's, you know, in the same bucket, for example, same folder, and then -- and have you describe the different ones and we'll mark them as we go just so we can keep track.

A Okay.

Q So you've started to do that already with what we'll mark as next in order which will be 5, but why don't you just briefly again describe what the

collection is of Exhibit 5.

A Okay. Exhibit 5 includes mostly primarily case control studies on non-Hodgkin's lymphoma that also includes exposures to benzene and/or some of the solvents.

Q Okay.

A And I've also included two other articles. One is in this because I think it's important to evaluate these kinds of studies, and that's the Bradford Hill article and also this little list from Cassarett and Doull about the fact that solvents -- there are lots of different kinds of solvents. So these studies have to be looked at with that in mind.

Q Okay. We'll mark that as five.

A And this is the file in which -- unless I made some mistakes, I have benzene marked with the green and the other solvents marked with yellow.

Q Great, thank you.

(Whysner Deposition Exhibit 5 was marked for identification and was attached to the deposition transcript.)

BY MR. HANLEY:

Q The next folder we'll mark as Exhibit 6 and briefly describe what Exhibit 6 is.

A These are various cohort studies. For

example, the rubber workers, studies of rubber workers. Oh, I just realized that there was one in here that was a recent one that just came out, but I have it in this folder. It came out in 2007, a more recent rubber worker study by Beall, B-e-a-l-l.

It also has people who were exposed to toluene in various industries, and in here I also have the IARC monograph on paint -- painters, because -- because toluene is often described in that.

MR. HANLEY: That's Exhibit 6.

(Whysner Deposition Exhibit 6 was marked for identification and was attached to the deposition transcript.)

THE WITNESS: And this file has some -- a few articles on dose response issues for benzene, benzene threshold, the chemical interaction one we just talked about with toluene and benzene, and also the -- my basis for believing that inhibition of Topoisomerase 2, Topo 2 is the -- is a -- not only a mechanism that I believe is most likely for the effects of benzene, but also that that mechanism would have a threshold, and there's some studies that have looked at that particular issue for Topoisomerase 2.

MR. HANLEY: We'll mark that as Exhibit 7.

(Whysner Deposition Exhibit 7 was

marked for identification and was attached to the deposition transcript.)

THE WITNESS: This is a file that contains studies of petroleum workers that include information about NHL rates.

MR. HANLEY: We'll mark that as Exhibit 8.

(Whysner Deposition Exhibit 8 was marked for identification and was attached to the deposition transcript.)

THE WITNESS: Except for the Health Watch studies from Australia, which there are various iterations of here, it's one of the groups, but they published a lot of documents.

BY MR. HANLEY:

Q So that's what's in Exhibit 9 then?

A Yes.

(Whysner Deposition Exhibit 9 was marked for identification and was attached to the deposition transcript.)

THE WITNESS: This file contains all of the benzene cohort studies that have information about NHL.

MR. HANLEY: Okay. We'll mark that as Exhibit 10.

(Whysner Deposition Exhibit 10 was

marked for identification and was attached to the deposition transcript.)

THE WITNESS: This is the literature about other risk factors that have been studied, like smoking and obesity, so on and so forth.

BY MR. HANLEY:

Q None of which particularly apply to Mr. Molina?

A Well, they apply to Mr. Molina, but as we said, I can't say that these have been clearly causally related to non-Hodgkin's lymphoma, but I mean they are -- they describe some of the things -- characteristics of Mr. Molina, obesity, smoking history, working as an agricultural worker and so forth. So I've included them here.

Q Is there any author who suggests that their studies have demonstrated that there is a causal association between non-Hodgkin's lymphoma and any of these other risk factors?

A I couldn't tell you without reading the papers if they've actually said that.

Q Is there any study that you're aware of that says that smoking is related to non-Hodgkin's lymphoma in a causal way?

A I don't know.

Q Same question for obesity.
A I mean, people -- I mean, what I'm having trouble with is if people have a finding, you know, and some of them do have these findings, these associations, I can't remember if anybody actually used that terminology.
Q What? That it's a cause?
A Correct.
Q And cause being the sense of a causal association. So the association it has a strong enough statistical power to be considered not random and not by chance, but causally associated, there's -- you don't know of any studies that say that for non-Hodgkin's lymphoma and smoking or obesity?
A No, I don't. I don't know offhand that any studies would say that. As I said before, like with the studies on benzene or solvents, you have to go through and look for consistency. My take is you have to go through and look for consistency among the studies in order to come up with a causal determination, and I've said that I don't think any of these are causally related or have been demonstrated to be causally related, but I can't vouch for what one of the authors might or might not have said.
MR. HANLEY: All right. So that is being

marked as Exhibit 11, I believe.

(Whysner Deposition Exhibit 11 was marked for identification and was attached to the deposition transcript.)

THE WITNESS: I'll start a new pile. This is the gasoline toxicological profile from ATSDR, and I've included this because they -- at least when that was written, they had reviewed the studies on gasoline, and gasoline is a petrochemical that has, you know, a percentage of benzene in it.

MR. HANLEY: Okay. We'll mark that as 12.

(Whysner Deposition Exhibit 12 was marked for identification and was attached to the deposition transcript.)

THE WITNESS: And then this is what I call benzene state-of-knowledge file. It has to do with -- there are a few documents in here that have actually, I think, been presented by plaintiffs' experts. I think there's the -- there's some old articles from the Journal of Industrial Toxicology -- Journal of Industrial Hygiene & Toxicology. There's an API toxicological review. There's a NIOSH document from 1974.

MR. HANLEY: Okay. Let's mark that as next in order, Exhibit 13.

(Whysner Deposition Exhibit 13 was marked for identification and was attached to the deposition transcript.)

BY MR. HANLEY:

Q Is that collection of articles in Exhibit 13 comprehensive on the issue of what was historically known about benzene and its hazards?

A Well, I certainly didn't include every article that has studied benzene or talked about benzene in terms of that. I think -- the reason that a few of these articles are here is that I think plaintiffs' experts had brought up the API review, and so I included that, and also the underlying articles that the API was quoting when they made their statements. And the 1974 NIOSH document speaks for itself. It talked about what was known about benzene at that time.

Q All right. What's the last folder you have there?

A These are various monographs or -- well, I have -- you probably don't need this since we have the whole thing, but that's from the -- this is from that ATSDR.

I have the monograph on rubber industry. Actually, this doesn't need to be in here. This is

just a list of all the monographs. The benzene one from 1982, some petroleum solvents, the -- the toluene IARC monograph, toluene and -- no, xylenes is in here as well.

Q Okay. Did you have any objection to leaving this list of the IARC monographs in that folder?

A No, I don't think so. It's out of date, but -- it stopped with '77, but I can leave it in. That's fine.

MR. HANLEY: Okay. Then we'll mark that folder as Exhibit 14.

(Whysner Deposition Exhibit 14 was marked for identification and was attached to the deposition transcript.)

THE WITNESS: Now, the rest of my materials -- oh.

BY MR. HANLEY:

Q Go ahead.

A Yeah, the rest of my materials are depositions or materials specific in this case. You know, Weisenburger's, Nicas's report, one of Weisenburger's review article, Nicas's deposition testimony. This was the document that I got this morning, Reiss's report, some old summaries of Wilson's deposition, Jones', co-workers, Molina's

deposition, some selected medical records, and depositions of Dr. Phan, P-h-a-n, Dr. Meng, M-e-n-g.

I excerpted some things from all of these other files that you have a list of. University of North Carolina reports, stuff like that that were -- you know, the IH reports that were done at Salinas. Deposition of Dr. Infante. This is the -- another North Carolina IH report. Deposition of Robert Helm. Here are a couple of things that were from Nicas's deposition, things like that. These are all --

Q Okay. Does that conclude itemizing everything that you brought with you today?

A Everything except the volumes of medical records that I have. They're all in this other box right here (indicating).

Q And that's about a banker's box full of medical records?

A Yeah.

MS. KAHN: For the record, it looks like there are Post-It notes on various pages of the materials that Dr. Whysner has identified.

BY MR. HANLEY:

Q All right. Can you tell me what the import was of your review of the medical records?

A Well, again, the -- as I said, the medical

literature has been changing a lot about how they evaluate these other risk factors.

First of all, I wanted to make sure that Mr. Molina didn't have what I would consider to be a causally related risk factor: HIV infection, immunosuppressive drugs, family history that would indicate that he might have had some kind of immunodeficiency syndrome.

And then there were all of the -- there was other factors, and since I knew that the literature on all of these other things like smoking and obesity and somewhat -- were somewhat of a moving target, I wanted to document whether or not he had any of these other conditions and, you know, just to understand his -- his medical history so that I could describe it if asked to do so and the confirmation of his NHL and the type of NHL that he had. Those are the reasons that I reviewed the medical records.

Q Would you agree that cancers are usually a result of some environmental exposure?

MS. KAHN: Objection. Vague, ambiguous, and overbroad.

THE WITNESS: Well, I think causes of most cancers are not known. That's my -- that's what I would -- certainly in terms of an individual. Now, if

you include environment, if you include things like smoking and diet and all of that, I would say that cancers can be the -- many cancers can be the interaction of those types of environmental factors with the genetic backdrop of that particular individual or individuals. But environment, when we talk about environment, we're talking about all those things, you know, alcohol, smoking, and so forth.

BY MR. HANLEY:

Q Causes external to the body is what I'm getting at when I talk about environmental exposures. So whether it's smoking or asbestos or benzene or nickel or whatever it is, most cancers are caused by some sort of toxic exposure to the target cell; is that correct?

MS. KAHN: Objection, overbroad. Vague. Ambiguous.

THE WITNESS: I don't know if I could characterize it as most cancers. All I can say is that we know that there are certain what we call potentially preventable cancers which have been studied and -- and many of those can be related to environmental factors like the ones you described.

BY MR. HANLEY:

Q Do you have an opinion as to what percentage

of cancers that occur are potentially preventable cancers as you've described them versus spontaneous cancers that arise for literally just no reason?

A Well, that'd be very difficult to do. I mean, you know, the largest cause of cancer in women is breast cancer and the major risk factor for breast cancer is estrogen and most of the estrogen is internally generated and so things like that make it difficult to make those kind of determinations. Is it preventable if the woman had an oophorectomy when she was young. Could you prevent her cancer? Would you really call that a preventable cancer? I think that's a different thing to try to quantify.

Q Are you aware of any cancers that have been established to be spontaneous as in not like the example you gave where there's estrogen is considered a causative factor, but where there's not only no known cause, but it's determined that that cancer is somehow just spontaneous?

MS. KAHN: Objection, vague. Ambiguous. Overbroad.

THE WITNESS: I'm having a little trouble with the word "spontaneous." We know that certain cancers are very genetically determined. The example I gave about -- I mean, breast cancer with estrogen,

since, you know, a woman's estrogens are generated by their own body, then, in essence, even if the estrogen were involved, it would still be sort of spontaneous, I guess.

I mean, there are obviously some cancers like lung cancer that we know are strongly related to, you know, both smoking and from an occupational standpoint asbestos exposures, but there are other cancers where most of them we don't seem to see any cause for that kind of cancer, like non-Hodgkin's lymphoma, and so I would -- I would say that most non-Hodgkin's lymphoma is idiopathic and I guess one could characterize that as spontaneous, although I don't -- that's not really -- I don't know that's really a good medical term to use.

BY MR. HANLEY:

Q Do you agree that for most people, even if they have a genetic predisposition, they won't get a given cancer without exposure to some environmental toxin such as cigarette smoke; is that right? I mean, someone can be genetically predisposed to getting lung cancer but that genetic predisposition won't end up resulting in cancer unless there's exposure to a carcinogen like cigarette smoke or asbestos or both or something like that; is that true?

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

THE WITNESS: I don't think so. For example, let's take the cancers that are related to something called Li-Fraumeni syndrome. These are people who have a mutated P53 gene, okay, and we know that P53 genes are very important in cancer causation. These people have a very high incidence of breast cancer, brain cancer, and also I think cancer of the colon.

In that case -- just take the brain cancer, for example. We don't know the causes of any brain cancers. There's never, I mean, been convincingly described except for ionizing radiation a chemical cause, but we do know that people who get brain cancer, and it's mostly in men in their 30s and 40s get a type of brain cancer that starts out with P53 mutation, just like the people who have this Li-Fraumeni syndrome.

I think we can be pretty certain that what's called loss of heterozygosity takes place in people who have Li-Fraumeni syndrome without any kind of chemical interaction, because these kind of events can occur just through cell replication. There are errors that occur, genetic errors that occur when cells are

replicating their DNA, and through that mechanism we know we can get these kinds of -- instead of just having one P53 gene mutated, we can get the other P53 gene mutated as well spontaneously.

And so I think there's good evidence that many -- that's an example, but there's evidence that other people have these kind of genetic defects and they can be -- they can -- they can -- they're very prone to getting cancer without any kind of environmental interaction.

BY MR. HANLEY:

Q Is the P53 gene a tumor suppressor gene or an onco gene?

A Tumor suppressor gene.

Q And the P53 tumor suppressor gene can suffer genetic mutations from, for example, exposure to asbestos; isn't that right?

A Well, I think it can -- from a number of agents, yes, it can.

Q And that includes benzene, doesn't it?

A I don't know that P53 mutation has ever been particularly associated with benzene exposure. A lot of genetic mutations and translocations -- not genetic mutations, but deletions, translocations, and trisomies that have been associated with benzene are

found to occur, but I don't think that's one of them, you know, but I'm -- I'd have to look that up.

Q You suggested, and I think it was in Exhibit 11 or 12, but you can tell me, that there were certain articles that supported your idea or your opinion that there is a threshold below which benzene does not cause AML. Can you direct me to those studies which you believe support that opinion?

A Yes. Well, one of them is the Rinsky study, where -- where there -- from exposures of zero to 40 ppm years, and that's laid out in their '87 paper, where they didn't find any increase, and they didn't find any statistically significant increase until you get over 200 parts per million years.

Q So that's one of the articles contained in Exhibit 10?

A Right.

Q And do they say that they're -- that they find that there's a threshold below which there's not a risk for developing cancer?

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

THE WITNESS: I don't know that they have used the word "threshold" in their article. I mean, I'd have to read it through again to see that they did

but --
BY MR. HANLEY:
Q Is it fair to say then you don't know whether they do or not, but that's your interpretation of their data?
A Well, that's my interpretation of their data. I don't see them saying that here, but, like I said, in the zero to 40 ppm years, the standardized mortality ratio was basically 1. It was 1.09. So that's -- but this is just part of my -- I mean, there's the epidemiological basis and then there is also the -- the mechanistic basis, which is my analysis of the genotoxicity literature, my papers included in that file, leads me to believe that the best explanation for the way in which benzene causes acute myelogenous leukemia is through inhibition of this enzyme called Topoisomerase 2, and that type of toxicity would be expected to have a threshold because you'd have to -- you'd have to inhibit a certain amount of that enzyme before you would begin to see chromosomal breaks and translocations, and there is -- there's some substantiation for that in the scientific literature as well.
Q I'd asked you to direct me to any articles that support your opinion that there is a threshold

below which benzene does not confer a risk for developing cancer at all, including AML. You've pointed me to the Rinsky article.

Is there any other scientific or medical publication that you can point me to that supports in your opinion your belief that there is a threshold below which benzene is not a cancer risk?

A Well, so when I was talking about what we call biological plausibility, meaning the mechanism and what we believe is true about the mechanism and how that mechanism could have a threshold, I thought I was being responsive to your question.

Q Okay. Which article in particular are you referring me to?

A Oh, okay. Okay. Well, these would be in part of Exhibit 7. There's an article by Lynch called Investigations Into the Concept of a Threshold For Topoisomerase Inhibitor Induced Clastogenicity. Benzene is considered to be a clastogen.

There is an article -- I got the web site from FDA because a lot of drugs are Topoisomerase inhibitors, and so FDA's interested in this issue about whether or not there is a threshold.

And I reviewed -- as I mentioned, I reviewed the studies on genotoxicity that lead me to that

belief of the mechanism.

Q Identify that article, please.

A It's an article that I wrote in mutation research and was published in 2004.

An article by David Eastman about Topoisomerase 2 inhibition. And there are several other articles in here that are -- whoops. I got a second copy of the -- of the Lynch article in here.

This is an article about a drug called bimolane, which I think is about the closest to benzene -- you know, the Topoisomerase inhibiters are used as anticancer drugs, and in their anticancer drug treatment, they cause a certain number of secondary leukemias, acute myelogenous leukemias as well, which is another reason to believe that Topoisomerase inhibition, which has been shown to occur with benzene metabolites, is a likely or plausible mechanism for the development of acute myelogenous leukemia because we know it has in people with certain Topoisomerase inhibitors.

Q Okay. So are there any other articles then that support your opinion that there's a threshold below which benzene does not pose a risk for cancer?

A All I can say is there are a few other articles in this file that also deal with this

Topoisomerase thing.

Q Which file is that?

A This is that -- the file in Exhibit 7 called Benzene Threshold Topo 2.

Q Okay.

A And then there's a file in there also that has to do with the chemical interaction, saying that if benzene -- if there's an exposure to benzene and toluene, it inhibits the benzene effects, which I consider that to be also part of the threshold issue when we're talking about AML and solvents that contain benzene, that the other constituents of those solvents inhibit the ability of benzene to produce leukemia as well, which would be part of the threshold argument.

Q How do you know that they do?

A Well, like I say, there are some studies that say that they inhibit the metabolism of benzene to its metabolites, and those are the metabolites that I think are pretty universally agreed upon are involved in the formation of acute myelogenous leukemia, that benzene has to be metabolized, and if toluene inhibits the benzene -- the metabolism of benzene to those metabolites, then it should inhibit its effects.

But getting back to the other, there's

another study by Constantini. Where is it? That might be in the -- well, there's an article by Constantini in which he finds a similar dose response relationship with no increase documented until above 40 parts per million.

Q All right. And is that everything then that supports that opinion?

MS. KAHN: I'm just going to object to the extent it calls for speculation. The witness said he didn't review all the leukemia literature because this isn't a leukemia case. So a question asking if that's all the literature on the subject I think calls for speculation.

MR. HANLEY: Okay, thanks, Coach Ruth.

BY MR. HANLEY:

Q Doctor, have you described everything that you believe supports your opinion that there's a threshold below which benzene does not cause cancer?

A Well, the other studies -- well -- well, there are some studies. Like there's a study Bloemen, B-l-o-e-m-e-n, 2005, and they have cumulative exposure benzene with -- we're talking about AML essentially. They call it acute nonlymphocytic leukemia. Less than 28.3 parts per million and the SMR is .87, but this is actually a smaller study than the Rinsky study. So

that would support it as well, and the -- I don't know that I have the right papers from the Chinese study with me for that. It might be in one of the other folders.

Q So are you saying you would add the Chinese study?

A I can't remember right now how. I know they didn't -- I'm pretty sure they didn't find any statistically significant unless you get above 40 ppm years, but I don't remember what the SMR was below 40, whether it was so close to one or not or whether there were other doses involved. I can't remember right now.

Q All right. Setting aside that point then, can you point me to any scientific literature or governmental regulatory agency or any statements in any of these documents or any that you're aware of in the published literature where the author concludes that -- and states explicitly that there is a threshold below which a person is not at risk for cancer from benzene?

A You mean -- well, first of all most -- in most of these cases, for example for the government agencies, the way in which they proceed with doing a -- most of the conclusory statements are made by

these regulatory agencies, and regulatory agencies in some cases have come to the conclusion that some chemicals would not cause an increase in cancer before you reach a certain dose. But I don't believe they have come to that conclusion regarding benzene at this time.

Q Are you able to direct me to anywhere in the published literature anyone who says that there is, in fact, a threshold below which benzene does not confer a risk for developing cancer?

MS. KAHN: Objection, asked and answered.

THE WITNESS: Well, all I can say -- all I can do is again talk about the -- the -- what is available in the literature, and that is that it hasn't been demonstrated to occur above a certain dose, but I don't know that I could -- I could answer that question for you in terms of other -- what other people have said.

BY MR. HANLEY:

Q Well, if you don't know of anywhere in the literature where someone has said there is a threshold below which benzene does not confer a risk for developing cancer, then the answer is no, you don't know of that. Whether it exists out there somewhere and you can't remember it or it exists out there and

you've never seen it or you've never seen it because it doesn't exist out there, I understand it could be any one of those three possibilities.

But as you sit here today, are you able to point me to any literature where the author states that there is a threshold below which benzene does not confer a risk for development of cancer?

MS. KAHN: Objection, the literature speaks for itself.

THE WITNESS: I believe there is, but I couldn't point you to which specific article might have said that. I mean, I believe I have read that. I remember that, but I can't really point you to the particular article right now.

BY MR. HANLEY:

Q All right. Would you agree that a cohort study that shows both a causal connection between an agent and a given cancer and also a dose response relationship is especially persuasive on the issue of causation?

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

THE WITNESS: I don't believe a single study can establish a causal relationship, so I don't agree with your premise, or if that was a premise. But I

just don't agree with the statement you just made.
BY MR. HANLEY:
Q Would you agree that it is of significance when a study shows or a cohort epidemiological study shows not only an association statistically significant with sufficient power but also a dose response relationship, that those two elements being contained in a single cohort is of significance in evaluating that piece of literature?
MS. KAHN: Same objections.
THE WITNESS: Not necessarily, and the Chinese study is a good example, and I mentioned that because they do have -- if you look at it sort of on face value, they had an increase in relative risk which was at least of borderline statistical significance or it depends on how they analyze the data. But on the other hand when the authors later looked at the differences among different industry groups -- this is from Rinsky 2000 -- they said, "We noted, however, that an excess of NHL was found in our study," but they also said, "which could potentially be attributed to other exposures as the excesses were significant only in the subset of benzene-exposed chemical workers."
So you can have a study that shows an

increased cancer rate and you can have a study, that same study could show a dose response relationship, but you can't rule out the fact that that dose response relationship and the other is due to the fact that other chemicals might be involved and that when people get a greater dose of benzene, they're also getting a greater dose of these other chemicals because of concurrent exposures. And this is, I think, what the -- what Hayes, et al., in 2000 were referring to.

So, again, a single study, even if it has an elevated rate of cancer and a dose response relationship, does not necessarily mean that it shows a causal relationship. You have to look at other studies like the Rinsky study or the Bloemen study or the Collins study and see whether or not there is a consistency among those studies that would -- that would lead you to believe that there is a causal relationship.

BY MR. HANLEY:

Q Are you suggesting that somehow because a cohort demonstrates a causal association from an increased relative risk and a dose response relationship that somehow that suggests that confounding factors might be involved?

A I don't think that's what I said. I said -- I know of -- the example, and since we're talking about benzene and NHL, I brought up this example, and I'm just saying that -- I believe your previous question was how much weight do you give to a study because it has both of those elements, and what I was saying, if we're talking about what we're talking about here, which is benzene and NHL, then there is a prime example of something where you did have to take those confounding chemicals into account.

Can we take a short break? Are we moving into something else?

Q Sure, that's fine.

(Recessed at 2:28 p.m.)

(Reconvened at 2:33 p.m.)

BY MR. HANLEY:

Q Is it your opinion, sir, that exposures to benzene up to 40 ppm years of total cumulative exposures are safe and do not confer a risk of cancer?

MS. KAHN: Can I have the question read back, please.

(The reporter read the record as requested.)

THE WITNESS: Well, based on the fact that AML is the only cancer that has been causally related

to benzene and there is no increased risk below 40, I would say yes.

BY MR. HANLEY:

Q What is your confidence in the dose reconstructions used to report those numbers of 40 ppm years in the literature for reasonable and scientific accuracy? How do you know that the cohorts are, in fact, exposed to that high of levels?

A Well, the reconstruction is documented in a paper by Rinsky, et al. You know, the Rinsky study was done by NIOSH, as I'm sure you're aware. It also involved Dr. Infante, and so these dosage reconstructions are documented in American Journal of Industrial Medicine, 1981, and these have been examined by others as well, including the U.S. EPA, and I know that Dr. Crump -- I have in here the Iris overview, and they talk about the -- using -- they used the Rinsky study for their -- their risk assessment that they do for benzene. And so it's been looked at by EPA, and I think Crump was a contractor for EPA who looked at that dose reconstruction. So I have a reasonably high confidence in it.

Q Is there any level of toluene below which you believe it does not confer a risk of cancer?

A Well, toluene has never been, as far as I

know, found to cause any form of cancer, and one of the -- and I believe that includes in animals, experimental animals. So it's not classified as either a human carcinogen or an animal carcinogen. The last time that IARC looked at it, they -- they put it in group 3, which means that it's -- it's not either a human or an animal carcinogen.

Q So you don't believe there's any level of toluene that confers any risk of cancer?

A That's correct.

Q What level of ppm years confers any risk of cancer from total petrochemical hydrocarbon solvent exposure?

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

THE WITNESS: Well, again, the -- the -- I don't believe that there has ever been a type of cancer that has been causally related to total hydrocarbon or at least petrochemical hydrocarbon exposure. The IARC reviewed solvents, and they were talking about petrochemical solvents, and the last time they reviewed it, I believe they also put it in group 3, non -- not classifiable, which means that they didn't find them to be either animal carcinogens or human carcinogens.

BY MR. HANLEY:

Q So is that to say it's your opinion that there is no level of ppm years of petrochemical hydrocarbon solvent exposures that you believe confers any risk of cancer?

MS. KAHN: Objection. Overbroad, misstates the witness's testimony, compound, vague, and ambiguous.

THE WITNESS: All I can say is not that has ever been found in studies.

BY MR. HANLEY:

Q So it's your opinion that no matter how many total petrochemical hydrocarbon solvent ppm years someone may have suffered exposure to, they will not be at risk for any form of cancer?

MS. KAHN: Same objections.

THE WITNESS: As far as we know, no. As far as we can base it on the scientific -- medical literature, that's true.

BY MR. HANLEY:

Q What -- what hydrocarbon petrochemical solvents do you believe are capable of conferring a risk of cancer, other than benzene?

A I can't think of any. I don't believe there are that have been demonstrated.

Q How do you define petrochemical hydrocarbon solvents?

A Hydrocarbon solvents that have been derived from petroleum.

Q And what are hydrocarbon solvents?

A They contain hydrogen and carbon.

Q And is both benzene and toluene a petrochemical hydrocarbon solvent?

A Yes.

Q What are all of the petrochemical hydrocarbon solvents that Mr. Molina was exposed to in his work at the Salinas Firestone facility?

MS. KAHN: Objection, overbroad. Lacks foundation.

THE WITNESS: Well, I can -- I mean, what was the list of constituents? I would have to base it upon looking at the -- the -- what I remember from the MSDSs, and mostly hydrocarbon solvents are involved with -- or have paraffinic alkanes. They're various lengths. Then there are aromatic chemicals such as benzene, toluene, xylenes, and -- and then there would be other smaller components such as like ethyl benzene, and I believe I remember seeing those in the list of components of -- of -- well, at least they would be -- let's put it this way. They would be kind

of typical of hydrocarbon petrochemical solvents having those -- those kinds of constituents.

BY MR. HANLEY:

Q How many different petrochemical hydrocarbon solvents do you believe Mr. Molina was exposed to at the Firestone facility?

MS. KAHN: Objection, calls for speculation.

THE WITNESS: I -- I was not asked to really look at that. I mean, I saw mention of a couple of Firestone numbers, and I looked at some of those. I think there was 356 and 374. Those are the two that I remember. But, I mean, I don't really have an opinion as to -- as to whether or how much of those chemicals he was exposed to because that was not something I was asked to look at.

BY MR. HANLEY:

Q Is there not a body of literature relating to the association between exposure to petrochemical hydrocarbon solvents as a class and the development of cancer?

THE WITNESS: Can you repeat that question.

(The reporter read the record as requested.)

THE WITNESS: Well, there's certainly the petroleum worker studies who at least have the

opportunity for exposure to all kinds of petroleum products, some of which would be included in the solvents and including gasoline. There are studies of -- and I've included those in my files. There are studies of -- of people who work in the -- who are painters who were involved with petrochemical solvents, but they'd also be exposed to a lot of other things as well.

There are people who were exposed to gasoline that have been studied, and then I have a whole file of, as I mentioned, case-control studies of non-Hodgkin's lymphoma when they've asked questions about exposures to certain, you know, solvents. Sometimes they've specified specifically that they were petroleum-derived hydrocarbon solvents. Most of the times they don't.

BY MR. HANLEY:

Q Has not both IARC and the EPA stated explicitly that there is no known threshold below which benzene does not confer a risk of cancer?

A Well, I'm pretty sure that IARC has never said that, and I -- I don't know about the EPA.

Q Has not IARC reported that with respect to benzene there is no known threshold?

A No, I don't think they have.

Q Have you had a chance to get from your office the number of hours that you've billed in this case so far?

A Oh, when I took that break, I forgot to call them, but I could do that.

Q At Washington Occupational, part of what you do is -- for that company is consult as a -- consult in litigation; is that correct?

A Yes.

Q And part of what you do is -- for that company is litigation support for clients who have hired you in litigation?

MS. KAHN: Objection, vague and ambiguous.

THE WITNESS: Well, let me see if I understand what you mean. Litigation support meaning that I'm consulting with them before they have designated me as an expert witness. Is that --

BY MR. HANLEY:

Q Sure.

A Okay, yes.

Q Would you describe how Washington Occupational Health lends support to companies in litigation?

MS. KAHN: Objection to the extent the question calls for speculation.

THE WITNESS: Well, I can tell you what I do. I can't really speak for everybody at Washington Occupational Health, but they lend support to me sometimes by helping me with literature reviews.

BY MR. HANLEY:

Q Who's they in that sentence?

A They would be research associates or assistants who --

Q For Washington Occupational?

A Yes.

Q Okay.

A So they would help me with that. Sometimes I've asked them to review medical records for me. I haven't done that in this case, but in some other cases, another physician has assisted me in reviewing medical records. We -- of course, you know, in terms of litigation consulting, I think a large part of what we do is to help the attorney understand the case better, understand the literature, provide feedback regarding the medical records and what the literature shows that is relevant to the case and, you know, of course sometimes I'm asked to write a report. I wasn't asked to do that in this case.

Q Do you know why you weren't asked to do that in this case?

A My understanding -- I mean, I'm not a lawyer. So my understanding is certain jurisdictions require reports and certain jurisdictions don't require reports, but that's as far as I can sort of speculate on that answer.

Q The medical surveillance work that Washington Occupational Health engages in, is that largely on behalf of -- or paid for by insurance companies?

A Most of the work we do, as I said before, is work directly for the federal government.

Q Is it fair to say in the last several years about 50 percent of your work at Washington Occupational is litigation-related work?

MS. KAHN: Objection, asked and answered.

THE WITNESS: Yeah, I think I indicated it varies somewhat, but I would say recently it's been more. It's been somewhere between 50 and 70 percent, something like that.

BY MR. HANLEY:

Q Actually I think this morning you said 50 to 75 percent. Does that sound right?

A Oh, okay. 50 to 75.

Q And is that true for the last several years?

MS. KAHN: Objection, asked and answered.

THE WITNESS: No. It's varied. I think it's -- as I said, it depends upon the year, and in the past I've done more work with Superfund sites and administrative hearings related to things like permitting power plants, things like that, and as I mentioned before, until 2002 my primary employer was not Washington Occupational Health. I was at a research institute, where I was doing research.

BY MR. HANLEY:

Q In the Topo 2 work that you talked about earlier that was sponsored by the API, was that sponsorship disclosed on the face of the publication?

A Yes, I believe so.

Q And would you agree that it's important to disclose the funding for research on the face of the publication of it?

MS. KAHN: Objection. That's vague and ambiguous. Important for what purpose? Important to whom?

THE WITNESS: Well, I think it's -- it's the convention to do it now. I mean, that has changed over the years by the way. You know, people didn't used to do that, and I would say over the past ten years there's been more and more of a tendency that people are -- either do it. They're often required by

the journal that they're publishing in to do it. I mean, I have to go back and check, but let me just look and make sure I didn't give you the wrong answer on that, but I'm pretty sure that we disclosed that in our publication.

Yes, we did.

BY MR. HANLEY:

Q And my last question was, do you believe it's important to disclose such information generally for authors publishing their findings and conclusions in peer reviewed scientific literature, do you believe that it's important that, as a general proposition, those authors disclose their funding sources?

MS. KAHN: Objection, vague and ambiguous. Asked and answered.

THE WITNESS: I think that it's a good idea.

BY MR. HANLEY:

Q And why is that?

A Because I think that some -- it's important to know if somebody is -- you know, for example, this last go-around about the study that was funded by the tobacco companies that had to do with the idea that you could discover lung cancer 80 percent through CT scans, I think it's important for people to know that that information was funded by tobacco companies

because you would look at that -- I think as people described it, people would look at that with some skepticism, more skepticism, and scientists tend to be skeptical about things, and I think you'd look at it with more skepticism if you knew it was funded by a tobacco company.

Q Now, is it correct that you worked on the Topo 2 research for about 40 years consuming about 20 to 30 percent of your time during those years?

A No.

Q How would you accurately state that?

A Topoisomerase, it certainly wasn't 40 years. It might have been four years.

Q I thought that's what I said.

A Oh, I'm sorry. I thought you said 40 years.

Q No, I'm sorry. I meant -- if I did, I misspoke. I meant to say 4 years.

A Four years and what was the percentage?

Q About 20 to 30 percent of your time.

A I think that's probably right, somewhere in that ballpark.

Q And the funding provided for that was on the order of about a quarter million dollars?

A I don't specifically recall, but I wouldn't argue with that. I mean, is that an answer I gave in

another case or something?

Q I'm just asking.

A Well, I can't specifically recall right now, but I wouldn't -- I mean, I didn't receive the funding. The American Health Foundation received the funding to do that work, and it was not specifically to look at Topoisomerase 2. The project was to analyze all the genotoxicity information and to come up with what we thought added to the understanding of the development of AML, and that was something that we came up with on our own. I mean, it had been suggested in the literature by David Eastman, but it sort of actually surprised us that all the genotoxicity literature seemed to fit that particular model.

Q And how were you compensated for that -- for the work on that study? On an hourly basis or as getting a portion of the grant and then having certain things that had to be accomplished for that? How did that work?

A No. I was just a salaried employee of American Health Foundation. Didn't change my compensation at all.

Q Now, it's a presumption maintained, including maintained through today, by the EPA and the

World Health Organization and IARC that there is a no threshold model for carcinogens, correct?

A Well, there's -- there's been a lot of discussion about this. First of all, let me just clear one thing up. IARC in general does not get into dose response issues. They get into what we call hazard identification. They're strictly involved with classifying carcinogens based upon the data. So it would surprise me if IARC would have made a stand -- or a stance on this because IARC doesn't do quantitative risk assessment work.

Q Well, you have seen where the IARC has published, for example relating to asbestos, that not only is there no known threshold below which it does not confer a cancer risk, but also that that is true for all types of asbestos, including chrysotile.

Have you seen their monograph on that, which I dare say states just exactly what I said?

MS. KAHN: Objection, beyond the scope of the witness's assignment. Beyond the scope of our expert witness designation in this case.

THE WITNESS: I haven't seen that.

BY MR. HANLEY:

Q If that does exist, that would be inconsistent with what you're describing being the

normal practice at IARC as you were just describing in your answer; is that right?

MS. KAHN: Objection, lack of foundation. Beyond the scope of this witness's designation.

THE WITNESS: My -- my -- what I know about IARC, and like I said, I've worked on the working groups, we don't get into what happens at different doses. I'm not disputing the fact that IARC might have done a special publication on something like asbestos, but I certainly don't -- it wouldn't fit with what I know that IARC's mission is.

BY MR. HANLEY:

Q Is there a presumption maintained by the EPA and IARC and the World Health Organization of a known threshold model for carcinogens?

A For risk assessment purposes and regulation like setting drinking water standards and so forth, EPA has traditionally taken a default position, which means that that's the position they take unless you can prove differently. That carcinogens in general do not have thresholds, and that was based upon a particular model that was accepted 30 years ago, 40 years ago by which we believe that all chemicals cause cancer. Now we know today that that's not true anymore. So EPA has accepted the concept that if the

underlying mechanism by which the chemical produces cancer has a threshold, then cancer itself would have a threshold dose.

In other words, if you can show that a particular toxic event has to occur, and that that toxicity has a threshold, then the production of cancer would have a threshold dose. That's the kind of thing I'm talking about with the Topoisomerase thing. That's my best analysis of the way in which benzene produces AML, that there is this underlying toxicity mechanism, and if that toxicity mechanism indeed has a threshold, then it would make biological plausibility -- make sense that -- that the cancer produced would have a threshold.

But, as I said, this is -- the EPA has taken a regulatory stance, health protective, you know, bend over backwards to protect the public, but it doesn't really -- it's very different from doing a causal determination like we're doing here.

Q They have taken a different position as to some toxins, for example, asbestos and benzene, where they have affirmatively described there being no threshold for those toxins; whereas, they have not made the same affirmative statements about other toxins; is that correct?

A I have -- you know, as I said, for risk assessment purposes in general, they take the position that there are not thresholds and they do these risk calculations at these extremely low levels, and I think that -- I think the EPA in its original risk assessment guidelines as a matter of fact said that the true risk is anywhere from the number that is calculated from the risk assessment to zero, and you don't know where the risk is because it's totally dependent upon the model that's used for the low-dose extrapolation.

So EPA has, I think, pretty consistently acknowledged the possibility of there being a threshold, but for their regulatory purposes, which is again not what we're dealing with here because we're trying to determine whether or not something actually did cause the disease, but from a regulatory standpoint, they take that position to do things in the most health protective way that they can.

Q Is that a long way of answering my question yes with that explanation?

A Well, I don't know -- if you had a document you wanted to show to me about what EPA said affirmatively about benzene, I'd be glad to comment on it, but if you're asking me to, in essence, sort of

second-guess that and sort of -- try to figure out where such a statement might have come from, well then that's the best answer I can give you.

Q You were discussing a moment ago about a benzene threshold being indicated by your Topo research; is that right?

A Well, at least it's consistent with that research.

Q Have you or anyone else published anywhere in the open peer review scientific literature how those findings are consistent with or support the notion of a threshold for benzene causing cancer?

A Let me just check and see if we --

MR. HANLEY: I'm going to have to go.

(Discussion off the record.)

THE WITNESS: It may be in my paper. I can't locate it right now. I can't tell you sitting here.

BY MR. HANLEY:

Q And you can't tell me if anyone else has ever published that notion; is that right?

A That particular idea, I couldn't tell you sitting here right now if they have or not.

Q Has anything changed about our understanding of the mechanisms of carcinogenesis that would or has

changed or influenced our understanding about threshold for carcinogens?

MS. KAHN: Objection, speculative. Vague and ambiguous.

THE WITNESS: Well, there's been a lot of information that has come out. I mean, two of the working groups that I was involved in for IARC, we were actually dealing with some of these toxicity mechanisms, and in some of those cases not only did we say that -- that at low levels the toxic effect wouldn't occur and would not be expected to produce cancer, but in some cases we concluded that the toxic effect wouldn't even be expected to happen in people at all, and so, therefore, the -- the findings -- and we were talking about the extrapolation of animal studies to humans. The findings in animals wouldn't be at all relevant to human cancer.

BY MR. HANLEY:

Q And what toxic agent was that related to?

A Well, there are various ones. There's limonene, l-i-m-o-n-e-n-e, which is the principal ingredient in citrus oil which is used in air fresheners and all sorts of things. It's a naturally occurring compound. It also produces kidney tumors in rats, and it produces it by a particular mechanism

along with a whole range of other chemicals that wouldn't be expected to cause cancer in -- in humans. One of these being, by the way, unleaded gasoline.

There was a -- there were -- the whole issue of thyroid tumors in humans, we did a whole monograph on that saying that at low level exposures, one would not expect -- the exposures that one would see in humans, one would not expect to see cancer in humans because of differences in the mechanism by which the thyroid regulates its growth.

Q Let me ask you this. Describe anything that has occurred in our understanding of the mechanisms of carcinogenesis that would have anything to do with establishing or understanding a threshold for benzene below which a cancer risk is not conferred.

MS. KAHN: Objection, calls for speculation. I don't know what you mean by our understanding, and it's vague and ambiguous.

THE WITNESS: Could you repeat the question, please.

(The reporter read the record as requested.)

THE WITNESS: Well, I was describing the Topoisomerase research and the epidemiology studies that have been done for benzene. I think that those

would be the things that I've already mentioned that would indicate that.

BY MR. HANLEY:

Q Would you agree that for purposes of risk assessment, that the EPA has recently reconfirmed its acceptance of the no threshold model for benzene?

A If you're thinking of a particular document, I would be glad to comment on it.

Q Is your answer you don't know?

A Well, I don't know of any document that EPA has put out recently regarding benzene.

Q Now, sir, isn't it true that your position that the only cancer benzene is demonstrated to cause is AML is one that is outside the mainstream of current medical and scientific thinking?

A Could you repeat that question, please.

(The reporter read the record as requested.)

THE WITNESS: Well, that is what -- IARC concluded when it reviewed benzene, says, it is established that human exposure to commercial benzene or benzene-containing mixtures can cause damage to the hematopoietic system, including pancytopenia. The relationship between benzene exposure and the development of acute myelogenous leukemia has been

established in epidemiological studies. Reports linking exposure to benzene with other malignancy were considered inadequate for evaluation.

That's what IARC said when they evaluated benzene and that hasn't changed.

BY MR. HANLEY:

Q What are you reading from?

A The IARC monograph on benzene that was the original one that was published Volume 29, and so I don't think that has changed since then.

Q Okay. That's a part of which exhibit?

A Exhibit 1.

Q And that publication you just read from is dated from when?

A '82.

Q And you don't think that anything has changed on the thinking on that point in the medical and scientific community since '82?

A Well, I can certainly tell you that IARC hasn't published any update that would indicate that that has changed and that's my opinion, and I think it's -- I believe it's the mainstream scientific opinion.

Q Are you familiar with the phrase that epidemiology can only detect a catastrophe?

MS. KAHN: Objection, lack of foundation. Vague and ambiguous.

THE WITNESS: I hadn't heard that before.

BY MR. HANLEY:

Q Are you familiar with the concept? Does that make sense to you?

A No, I don't think it makes sense to me.

Q And why not?

A Because, I mean, a catastrophe to me means something that is obvious even without epidemiology.

Q Would you agree that if something confers a 1.5 relative risk, that of three cancers that one of those could have been avoided?

MS. KAHN: Objection, incomplete --

THE WITNESS: Doesn't make any sense.

MS. KAHN: -- hypothetical. Vague and ambiguous.

BY MR. HANLEY:

Q Let me try again. Would you agree that if a particular toxin causes an increased relative risk for cancer of 1.5, a 1.5 relative risk, that in a group of three people with that particular cancer based on that relative risk, one of those cancers could have been avoided?

MS. KAHN: Same objections.

THE WITNESS: I don't believe that with three cancers that would be a statistically significant finding, and so I think the uncertainty would be so great that one couldn't reach that conclusion.

BY MR. HANLEY:

Q That's not what I'm asking you, sir. This is not a hypothetical of a cohort. I'm saying that the result, the net result of an increased risk of cancer of just 1.5 relative risk means that of a group of people, one out of three of those cancers would be eliminated by taking away that particular toxin and that exposure?

MS. KAHN: Objection, overbroad. Lacking in foundation. Vague and ambiguous.

THE WITNESS: This would be based upon the assumption that you would truly know that that cancer, that additional cancer was caused by exposure to that chemical. So I think it speaks for itself.

BY MR. HANLEY:

Q But that does reflect the degree to which a relative risk as low as 1.5 can make a difference in an affected cohort; does it not?

A Now you're mixing --

MS. KAHN: Objection, lack of foundation.

Vague and ambiguous. Overbroad and compound.

THE WITNESS: Now you're mixing apples and oranges. You're doing exactly what you said you weren't doing. I started to talk about an epidemiology study you wouldn't be able to with three people make that determination. You're going back now and describing this as an epidemiology study and I disagree with that.

BY MR. HANLEY:

Q Do you believe that secondhand smoke can cause lung cancer?

A You know, I really -- I know that at one time that was controversial, and I've never -- let's put it this way. I've never really looked into the literature on secondhand smoke so I don't have an opinion on it. I believe -- and again -- I'm sorry. I'd have to be speculating to even know whether or not IARC has concluded that it causes cancer. I believe it has, but I can't be certain sitting right here.

MS. KAHN: Don't speculate.

THE WITNESS: Yeah, I'm not speculating. Sorry. It's not something I've looked into.

BY MR. HANLEY:

Q Are you familiar with the literature that reports the relative risk from secondhand smoke being

approximately 1.5?

MS. KAHN: Objection, beyond the scope of the witness's retention and our expert witness designation.

THE WITNESS: No, I'm not familiar with that.

MR. HANLEY: All right. I have to catch a plane. I have got -- I've got a fair bit more questions. I'm not going to be able to finish it today. I wouldn't finish today if we went all day.

MS. KAHN: I will point out the deposition was noticed to continue day to day until completed. It's now 3:15. We are prepared to put in a full day today. We are prepared to come back tomorrow if needed as called for by the deposition notice. We object to producing Dr. Whysner for a subsequent session.

There's no reason we can't complete the deposition today or this week. The witness has given reasons why it is difficult for him to conclude next week. We have three more depositions this week, three to five depositions next week. I don't know when we're going to find time to do it and you take a risk that you may not be able to complete the deposition if you leave at 3:15 today.

MR. HANLEY: Well, it's 3:25 now and I do need to leave.

MS. KAHN: Additionally, the court reporter's going to have the exhibits. So even if we can find a day next week to do the deposition and you're willing to do it by telephone, I don't know that the witness is going to have his originals back. So that's a real problem. We are not agreeing to produce him.

Now I will meet and confer with you about it. If we can find a way to get it reasonably done, we will try and accommodate you, but you are taking a risk by ending the deposition now and leaving to catch a plane.

THE WITNESS: Can I say something about my daughter' surgery coming up?

MS. KAHN: Sure.

THE WITNESS: My daughter's having surgery week after next and we have to prepare her next week for her upcoming surgery, which is fairly serious, and so I don't see how I can be available.

MR. HANLEY: Okay, I certainly appreciate that, Doctor, and I appreciate the offer to meet and confer. I would be glad to do it by phone. Obviously we need to work out the logistics with the documents

to the extent that I need to ask him about them, but I appreciate the --

MS. KAHN: It's up to you. You ordered the court reporter.

MR. HANLEY: I appreciate the effort to -- for everyone to do their best. Thank you.

Thank you, Doctor. You know, I owe you some money here. Let me see if we can get that.

MS. KAHN: It's going to be 5.5 hours' worth since we had about half an hour for lunch. Actually, we were here at 9 o'clock. It's a little more than 5.5 hours' worth.

MR. HANLEY: All right. I have checks for 450 unfortunately. I'm going to give you the four checks I have.

THE WITNESS: It's made out to Washington Occupational Health, and you have the correct tax ID.

MR. HANLEY: It is.

MS. KAHN: I wish you would reconsider and try and finish the deposition today or tomorrow since we're here.

MR. HANLEY: I simply can't do it. I'm absolutely unavailable. I've been out here for a week. It's been lovely, but I do need to get home.

I owe you for 5.5 hours at a rate of 550.

I've given you four checks for 450 each. We will send out tomorrow that balance.

MS. KAHN: I also live 3,000 miles away. I also have been out here the same length of time you are. I came to do a job. I would like to finish the job. I'm disappointed that we cannot do that.

(Signature having not been waived, the deposition of JOHN WHYSNER, M.D., Ph.D., D.A.B.T. was concluded at 3:25 p.m.)

I, JOHN WHYSNER, 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)

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JOHN WHYSNER, M.D., Ph.D., D.A.B.T.

CERTIFICATE OF SHORTHAND REPORTER-NOTARY PUBLIC

I, Joan V. Cain, Court Reporter, the officer before whom the foregoing deposition was taken, do hereby certify that the foregoing transcript is a true and correct record of the testimony given; that said testimony was taken by me stenographically and thereafter reduced to typewriting under my direction and that I am neither counsel for, related to, nor employed by any of the parties to this case and have no interest, financial or otherwise, in its outcome.

IN WITNESS WHEREOF, I have hereunto set my hand and affixed my notarial seal this 7th day of April, 2008.

My commission expires:
June 15, 2009

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NOTARY PUBLIC IN AND FOR THE
DISTRICT OF COLUMBIA