Exhibit 590

UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NORTH CAROLINA
SOUTHERN DISTRICT

IN RE:
CAMP LEJEUNE WATER LITIGATION No. 7:23-CV-00897
THIS DOCUMENT RELATES TO:
ALL CASES.

_____

VIDEOTAPED DEPOSITION OF
RICHARD HOPPE, M.D., FACR, FASTRO, FARS
Monday, June 9, 2025

Reported By:
KATHLEEN A. MALTBIE, STENOGRAPHIC REPORTER
California CSR 10068, Nevada CCR 995, Texas CSR 12212, RPR-RMR-CRR-CCRR-CLR-CRC-RDR

VIDEOTAPED DEPOSITION OF

RICHARD HOPPE, M.D., FACR, FASTRO, FARS

BE IT REMEMBERED that on Monday, June 9, 2025, commencing at the hour of 8:48 a.m. thereof, before me, Kathleen A. Maltbie, RPR-RMR-CRR-CCRR-CLR-CRC-RDR, a Certified Stenographic Shorthand Reporter, in and for the State of California, Nevada and Texas, personally appeared RICHARD HOPPE, M.D., FACR, FASTRO, FARS, a witness in the above-entitled court and cause, who, being by me first duly sworn, was thereupon examined as a witness in said action.

APPEARANCES OF COUNSEL

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ALSO PRESENT:

Alejandro Zamora-Ruiz, Videographer

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JUNE 9, 2025 8:48 A.M. PACIFIC TIME

P R O C E E D I N G S

MORNING SESSION

THE VIDEOGRAPHER: We are now on the record. My name is Alejandro Zamora-Ruiz. I am the videographer for Golkow. Today's date is June 9, 2025, and the time is 8:48 a.m. Pacific Time.

This video deposition is being held at the Hilton Garden Inn, Palo Alto, at 4216 El Camino Real, Palo Alto, California 94306, in the matter of In Re: Camp Lejeune Water Litigation, for the United States District Court, for the Eastern District of North Carolina.

The deponent is Dr. Richard T. Hoppe.

Counsel will be noted on the stenographic record, and the court reporter will now introduce herself and swear in the witness.

THE REPORTER: Good morning. My name is Kathleen Maltbie. I am a certified Stenographic Court Reporter, License No. 10068, also licensed in Texas and Nevada. Today's date is June 9, 2025. We are now on the record.

Would you raise your right hand, please?

MS. HORAN: Dr. Hoppe, you might need to put your microphone on.

RICHARD HOPPE, M.D., FACR, FASTRO, FARS,
having been duly sworn,
was examined and testified as follows:

MS. HORAN: I guess we should notice appearances. Alanna Horan on behalf of the United States, and I'm joined by my colleague, Matthew Elliott.

MR. MCGOWAN: Chad McGowan and Randy Lee for the plaintiff.

EXAMINATION BY MS. HORAN

BY MS. HORAN:

Q. Good morning, Dr. Hoppe.

A. Good morning.

Q. Could you please state your full name for the record?

A. Richard Thomas Hoppe.

Q. And what is your current address?

A. My current address is 340 East Edith Avenue, Los Altos, California, 94022.

Q. Is that a personal home address or a business address?

A. That's my home address.

Q. My name is Alanna Horan. I met you just a

moment, and I am an attorney with the Department of Justice representing the United States in this case.

Do you understand that?

A. Yes.

Q. I'm going to go through a couple of ground rules.

But you've been deposed before, correct?

A. Yes, I have.

Q. Roughly how many times?

A. About a dozen times.

Q. I'll go through some general rules of the road. You're probably familiar with them, but just to make sure we're on the same page.

During this deposition today, the court reporter will record and transcribe everything we say while we're on the record.

You understand that, right?

A. Yes.

Q. To make sure that everything gets transcribed properly, I'll ask that you answer your questions verbally and clearly. For example, if you say "yes" or "no," please respond verbally instead of shaking your head; is that fair?

A. Yes.

Q. We'll do our best to speak at a reasonable

pace for the court reporter, but if for whatever reason you can't understand me, will you let me know?

A. Sure.

Q. If you don't hear or understand my question, will you please ask me to clarify. And if you answer the question, I'm going to assume you understood it; is that fair?

A. Yes.

Q. I will do my best to always let you finish speaking before I start speaking, and I just ask that you do the same; is that fair?

A. Okay.

Q. If you wish to take a break at any point throughout today, that's entirely fine with me. Please just ask for a break. If a question is pending, meaning I've asked you and I'm waiting for you to respond, I just ask that you respond to the question before we take the break; fair?

A. Yes.

Q. Is there any reason why you would be unable to give truthful and accurate testimony today?

A. There's no reason.

Q. During the deposition today, you might

hear your attorney object to some of my questions.

Unless your attorney instructs you not to answer the question, I ask that you answer the question; fair?

A. Okay.

Q. What did you do to prepare for your deposition today?

A. I generally reviewed the materials that I submitted, the reports regarding the two plaintiffs in this action. I spoke briefly with the attorneys yesterday. And that really is the extent of my preparation.

Q. For how long did you speak with the attorneys yesterday in preparation of today?

A. An hour and a half.

Q. And was that over the phone, in person, Zoom?

A. It was in person.

Q. Other than reviewing your reports, did you review any other written materials, such as studies or medical records?

A. Not -- not immediately prior to the deposition, but I have over the course of the last several months --

Q. Sure.

A. -- reviewed those materials.
Q. But not in preparation for today?
A. No. But I had summarized the medical records and the specific causation reports that I produced, and I reviewed those.
Q. And the summaries of the medical records, is that the section in your report that goes through medical histories, or is the summaries a separate document?
A. No. No. It's what's in this -- in the specific causation report.
Q. Understood.
And are the attorneys that you met with yesterday the attorneys that are here today?
A. Yes.
Q. Have you communicated with anyone besides the attorneys to prepare for your deposition today?
A. No.
Q. How did you first become aware of the Camp Lejeune Water Litigation?
A. I guess I first became aware, there was some announcement on TV or some TV coverage regarding the existence of groundwater contamination at Lejeune, I think advertising for plaintiffs at the time.

Q. Do you recall if it was before or after you were retained in the case?

A. I really can't recall. It may have been around the same time, but I don't know if it was before or after.

Q. Do you recall who first contacted you about working on this matter?

A. It may have been Patrick Thelan, T-H-E-L-A-N.

Q. Prior to this matter, had you worked with Patrick Thelan before?

A. No, I did not.

Q. Did Patrick Thelan reach out to you or did you reach out to Patrick Thelan to work on that matter?

A. Oh, he reached out to me.

Q. And I had a chance to look at your retainer agreement, and I believe it says you were retained around June of 2023.

Is that accurate, to your memory?

A. Yes. But I'd have to look specifically at those documents to confirm.

Q. Sure. Sure.

And in -- it looked like you might have signed two retainer agreements.

Do you recall that?

Or been provided with two retainer agreements?

A. I saw in the records, yes, that I had two retainer agreements.

Q. One of those retainer agreements was signed with PLG.

Is that your memory as well?

A. Not sure who PLG is.

Q. Okay. Do you know why you have two retainer agreements?

A. Well, there were two plaintiffs. Perhaps that's the reason.

Q. Sure.

Do you recall when you received the second retainer agreement?

A. No, I don't.

Q. Okay. What has been your assignment in this case?

A. My assignment was to review the medical records of the two plaintiffs in this action and to give an opinion on whether I thought their diagnoses of non-Hodgkin lymphoma may have been related to their exposure to groundwater contamination at Camp Lejeune.

Q. Other than -- strike that.

The two plaintiffs' records that you looked at are Cometto Davis and Allan Howard, correct?

A. That's correct.

Q. Did you ever look through any other -- or offer -- strike that.

Did you ever look through any other plaintiffs' records or have you only ever worked on Mr. Davis' and Dr. Howard's?

A. I've only ever worked on those two.

Q. I'm not asking about time working with attorneys, but did anyone ever help you prepare your reports and opinions, outside of attorneys? Perhaps a student might have helped you --

A. No.

Q. -- or kind of an assistant.

A. They were generated totally by myself, and nobody reviewed them.

Q. Sure.

MS. HORAN: I'm marking as Exhibit 1, Hoppe Exhibit 1. This is the specific causation expert report of Richard Hoppe for the case Davis versus United States.

/ /

BY MS. HORAN:

Q. And Mr. -- or Dr. Hoppe, I'm sorry, I would just ask that you use this copy because it's marked.

(Whereupon, Deposition Exhibit 1 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, do you recognize this report?

A. Yes, I do.

Q. Is this the expert report you've submitted in this case as it relates to Mr. Davis?

A. Yes, it is.

Q. Does this report contain all the opinions you intend to offer in this case about Mr. Davis?

A. Well, I'm not sure I can say that. There may be some questions that you ask that I haven't addressed in this report, and I would feel obligated to respond to those questions.

Q. Sure.

So setting aside any questions I might ask you today, you're not here with new opinions that you intend to offer that are not contained in this expert report --

A. No.

Q. -- is that fair?

A. No. I'm not -- I'm not going to change any of my opinions.

Q. And you reviewed this report, which is marked as Exhibit 1, as -- in preparation for today, right?

A. I did.

Q. And in your preparation, is there anything that you wanted to edit or change while you were reviewing it?

A. Well, there were a few typos, but nothing of substance.

Q. And I just noticed, Dr. Hoppe, you have some documents in front of you.

What are those?

A. Those are the two reports that I prepared. The specific causation for Mr. Howard and Mr. Davis, and also a copy of the subpoena and a copy of requests for materials that I had reviewed.

Q. Okay. And your -- the copies that you brought with you of the reports, for Mr. Davis, is that the one that starts -- it starts on page 1 and ends on page 12, the copy you brought?

A. I'm sorry, but it's not paginated --

Q. Oh.

A. -- so -- but I only have --

Q. Let me reask this: The packet that I handed you includes references and your CV. I think it also has your prior testimony.

But the copy of the report you brought is just your opinions?

A. That's correct.

Q. Okay.

A. It does not include the references or my CV or other attachments.

MS. HORAN: I'm marking as Exhibit 2. This is a couple of different documents. These are the materials considered lists that we have received relating to your report for Mr. Davis.

(Whereupon, Deposition Exhibit 2 was marked for identification.)

BY MS. HORAN:

Q. Could you just take a look through those and let me know if they seem complete and accurate as to the materials you considered for Mr. Davis?

A. You know, I believe this looks correct, but short of going through --

Q. Sure.

A. -- and checking each individual item, I can't say with certainty.

Q. Sure.

But nothing jumps out at you as missing?
A. No. No.
MS. HORAN: I'm marking as Hoppe Exhibit 3. This is the report that Dr. Hoppe submitted as relates to Alan Howard.
(Whereupon, Deposition Exhibit 3 was marked for identification.)
BY MS. HORAN:
Q. Dr. Hoppe, do you recognize this report?
A. Yes, I do.
Q. And are these the complete opinions you intend to offer in this case about Alan Howard, except for answering any questions I might have -- ask you today?
A. Yes.
Q. And you reviewed this report in preparing for today?
A. I did.
Q. And is there anything in your review that you wanted to edit or change about the report?
A. Well, there was, actually, and it applies to -- to both of these plaintiffs, a paper that came to my attention by Yu and others, Y-U, that I don't believe I included in my opinions, but applies to both of them.

Q. Sure.

And we'll get to the Yu paper. I did see that you updated your materials considered list to include that report.

But other than the Yu report, are there any edits or changes you wanted to make to Mr. Howard's report in your review?

A. Again, only possible typographic problems that are not of substance.

MS. HORAN: I'm marking as Hoppe Exhibit 4. These are the materials considered lists that we received relating to your report for Mr. Howard.

(Whereupon, Deposition Exhibit 4 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, if you could just review those and let me know if they seem complete or if anything is -- jumps out at you as missing.

A. Nothing jumps out at me as being missing.

Q. If you could turn to page 2.

Do you see on your list Number 3 and 4 are two declarations by Mr. Howard, one is from November 26th and the other January 30th?

A. Mm-hmm.

Q. Did you work with Mr. Howard in drafting those declarations or have you ever worked with Mr. Howard directly?

A. No. I've never met Mr. Howard or spoken with him.

Q. So those declarations were provided to you?

A. Yes.

Q. And then if you look at number 9, it says (as read):

> Allan W. Howard exposure profile/chart produced contemporaneously herewith.

Do you see that one?

A. Yes, I do.

Q. Did you create that exposure and profile chart or was that provided to you?

A. That was provided to me.

Q. And you can flip back to Exhibit 2 if it would be helpful, but I didn't notice any declarations for Mr. Davis.

Did you review any declarations for Mr. Davis?

A. If it's not listed, then I likely did not.

Q. And sitting here today, you don't recall

reading a declaration --

A. No.

Q. -- about Mr. Davis?

A. No.

Q. Have you read Dr. Ambinder's reports?

A. Yes, I did.

Q. I didn't see those on either of the materials considered lists.

Are there any other materials that were government expert reports that you reviewed, other than Dr. Ambinder's reports?

A. There was -- there was another -- I forget who generated the report. I'm sorry, I can't remember offhand.

Q. Okay. If you recall at some point today, would you mind letting me know?

A. Sure.

Q. I'll represent, I think I believe I did see that you saw the transcript of Dr. Goodman.

Does it -- does Dr. Goodman's name ring a bell to you?

A. Dr. Goodman's name is familiar.

Q. Okay. Do you think it's possibly Dr. Goodman's report that you saw?

A. I -- the report that I saw was a response

to my report of specific causation. Similar -- you know, same line as Dr. Ambinder.

Q. Okay. Does Dr. Bailey ring a bell?

No? Okay, if you can recall.

A. Sure.

Q. If you remember at some point throughout the day, just if you could let me know.

A. Okay.

Q. When did you first read Dr. Ambinder's reports?

A. When it was provided to me, which I'd have to say was a few weeks after I submitted my specific causation report.

Q. Are you familiar with Dr. Ambinder professionally?

A. Yes.

Q. And Dr. Ambinder is a respected physician in your field, correct?

A. Yes.

Q. And you did not file a rebuttal report for either Mr. Howard or Mr. Davis in response to Dr. Ambinder's report --

A. No.

Q. -- correct?

You didn't?

A. No, I did not.
Q. We received a number of your billing records through February.
Have you worked on this matter since February?
A. Yes.
Q. Roughly how many hours do you think you've spent since February on this case?
A. Since February, I would say between eight and ten hours.
Q. Okay. And -- strike that.
And are you currently serving as a treating physician or are you retired from that role?
A. I'm working full time, including responsibilities for treating patients.
Q. And what percentage of your time is spent as an expert witness?
A. Single-digit percent. 5 percent or less.
Q. And you said you've been deposed about a dozen times?
A. Correct.
Q. And how many times have you testified in court before?
A. One, two, three -- I can remember three

off the top of my head. So maybe four or five. But at least three.

Q. Okay. And have you ever served as -- provided an expert report in a case that you were not deposed in?

A. No. I don't recall ever doing that.

Q. Have you ever offered an opinion as an expert in a case about the cause of someone's disease?

A. Yes, I have.

Q. And what was the disease?

A. Mycosis fungoides.

Q. Do you recall the name of that case?

A. I don't.

Q. Was it in the last ten years?

More than ten years ago?

A. It was probably close to ten years.

Q. And what was the opinion you offered as to the cause of their disease?

A. That I did not know the data to be able to provide evidence of causation.

Q. And were you working for the plaintiffs or the defendants in that matter?

A. I was working, I think, for neither. It was a patient whom I had seen and managed his care,

so I'd have to say I probably was working for the plaintiff.

Q. In that case, did you offer a written report or were you there as a treating provider?

A. I was there as a treating provider, so it was a -- just a deposition only.

Q. Throughout today, if you happen to remember the name of that case, would you mind letting me know?

A. Okay.

Q. Have you ever served as an expert witness for a defendant?

A. Yes.

Q. And when was that?

A. Most recently, about three years ago.

Q. And do you recall the name of that case?

A. I'm sorry, I don't.

Q. Fair enough.

Were your opinions in that case about what caused the individual's disease?

A. No.

Q. Prior to this case, had you ever worked as an expert witness for any of the law firms that you're now working with for Camp Lejeune?

A. No.

Q. You attached your resumé to your expert reports. I think it should be the same for both of them. So if you could turn to Exhibit 1. And your resumé is, I think, like, maybe 15 or so pages in.

A. Yes.

Q. And this resumé is dated February 6th, 2025.

Do you see that?

A. Yes.

Q. Is there any updates you would like to make today to bring this current?

A. Well, nothing of substance. I have some additional leadership responsibilities within one of the oncology organizations and a couple of additional publications.

Q. So other than a couple of new leadership positions and a couple of new publications, this resumé attached to your report as Exhibit 1 is a complete representation of your educational and employment background?

A. Yes.

Q. And other than any updated publications, this document is a complete list of your publications --

A. Yes.

Q. -- prior to February --
A. Yes, it is.
Q. Have you offered any publications in your resumé that investigated the cause of NHL?
A. I have some general publications and textbooks that I may have edited and chapters in textbooks that may have mentioned cautions of lymphoma.
Q. Do any of those -- and you're welcome to look through it if it would be helpful to you -- jump out at you as any of the ones you listed as publications that include those causation analyses that you just mentioned?
MR. LEE: Can we go off the record for a moment?
MS. HORAN: Sure.
THE VIDEOGRAPHER: The time is 9:16 a.m. Pacific Time. We're going off the record.
(Whereupon, a recess was taken from 9:16 a.m. to 9:16 a.m.)
THE VIDEOGRAPHER: The time is 9:16 a.m. Pacific Time. We're back on the record.
THE WITNESS: In response to your last question, for example, under publications, monographs, number 1, I'm -- you know, may have

addressed causes in the general context of the non-Hodgkin lymphomas. However, that -- that was something that I published 40 years ago.

BY MS. HORAN:

Q. You said monographs?

A. Publications, dash, monographs.

Q. Would you mind reading --

A. Yeah.

Q. Oh, I see it. I see it.

That's Hoppe R., the non-Hodgkin's lymphoma pathology staging treatment current problems in cancer 1987?

A. Correct.

Q. Okay. Sorry about that.

(Reporter clarification.)

THE WITNESS: Okay.

BY MS. HORAN:

Q. Anything else jumping out at you, or just that one?

A. No. That's -- that's the one that I would remember.

Q. Are there any publications in your resumé or articles that you've published involving the causation analysis between TCE, PCE, Benzene and NHL?

A. No.
Q. And you mentioned you're currently treating patients?
A. Yes, I am.
Q. And what percentage of your work is treating patients?
A. Well, it's basically 60 percent of my time.
Q. And what do you do with the other 40 percent of your time?
A. So it's a combination of teaching, research and administration.
Q. And you are a radiology oncologist; is that fair?
A. Radiation oncologist, correct.
Q. Do you have any other specialties?
A. No.
Q. Radiation is a type of treatment for cancer, right?
A. That's correct.
Q. So if someone has a specific type of cancer, do they come to you for that specific type of treatment, or would they come to you as a primary oncologist to figure out what to do kind of to create a whole care plan for their cancer?

A. They come both ways. So some patients are referred to me from other physicians because the other physicians know that they require radiation treatment, or sometimes I see patients who just have a recent diagnosis of cancer, lymphoma primarily, since my practice is restricted to lymphoma. And if radiation is not appropriate, then I would refer them to one of my other colleagues in a different specialty.

Q. You said your practice is limited to lymphoma?

A. Correct.

Q. What did you -- what did you mean by that?

A. What I meant by that is that all of the patients I see and manage have a diagnosis of lymphoma, which includes Hodgkin's disease or Hodgkin lymphoma, the non-Hodgkin lymphomas, including the cutaneous lymphomas or skin lymphomas, which is another component of my practice.

Q. What percentage of the patients that you treat have non-Hodgkin's lymphoma?

A. Probably 90 percent have non-Hodgkin lymphoma.

Let -- let me correct that. It's probably 80 percent.

Q. Sure.

How often in your practice do you inquire into what caused a particular individual's non-Hodgkin's lymphoma?

A. So my routine would be to take a good occupational and social history. And if there were items that I learned from their occupational or social history that I think might have contributed to lymphoma, or that diagnosis, then I would follow up on them.

Q. What are the types of questions you ask or would want to know about someone's occupational or social history in order to conduct that inquiry?

A. Well, for example, if someone is -- has had an occupation as an attorney, I wouldn't be so concerned about potential occupational exposures to agents that might be associated with lymphoma or other diseases. But if their occupation was, for example, in the dry cleaning business, I might ask about their exposures to different chemicals in that context.

With respect to social history, we're always interested in tobacco and alcohol use. Also, drug exposures and -- and their sexual histories.

Q. And why is it important for you to know

their occupational and social history -- I guess why -- why investigate the cause of their NHL as their treating physician?

A. Well, patients are often wondering, you know, why did I get this. And those, you know, question -- responses to those questions may be helpful in us answering those questions from the patients.

Q. And how often does a patient ask you what caused their NHL?

A. Well, they -- they phrase it in different ways. They might say, you know, am I -- what did I do wrong to get this lymphoma.

And I would say that at least half of the time, patients ask.

Q. And after you've collected their social and occupational history, what steps do you take in order to be able to provide them an answer?

A. Well, it's based on my general knowledge and general experience.

Q. How often do you tell a patient that the cause of their NHL is unknown?

A. Probably half the time. At least half the time.

Q. Do you tell your patients that it's

impossible to know the cause of their NHL with any certainty?

A. Yes.

Q. In your practice, to the best of your knowledge, have you treated patients with NHL that were at Camp Lejeune between 1953 and 1987?

A. Not to my knowledge.

Q. Have you discussed Camp Lejeune with any of your NHL patients?

A. No, I haven't.

Q. You've never examined Cometto Davis or Allan Howard, correct?

A. That's correct, I did not.

Q. And you've never spoken to them in, you know, Zoom, phone call, in person?

A. Never.

Q. Have you ever told a patient that the cause of their NHL was from an exposure to TCE?

A. No, I haven't.

Q. Have you ever told a patient that the cause of their NHL was an exposure to PCE?

A. No, I haven't.

Q. Have you ever told a patient that the cause of their NHL was an exposure to Benzene?

A. No, I haven't.

Q. Have you ever given a presentation or spoke publicly about Camp Lejeune?
A. No, I haven't.
Q. You are not an epidemiologist, correct?
A. That's correct.
Q. And you don't have any certifications in epidemiology, correct?
A. That's correct.
Q. And you've never been a principal investigator for an epidemiological study; is that right?
A. That's correct.
Q. You've never published peer-review literature on epidemiology; fair?
A. That's correct.
Q. Have you ever taught any courses on epidemiology?
A. No, I have not.
Q. Do you have any experience as an epidemiologist?
A. No, I don't.
Q. And you are not a toxicologist, correct?
A. That's correct.
Q. You don't have a certification in toxicology?

A. No, I don't.
Q. You've never been a principal investigator for a toxicological study; fair?
A. That's correct.
Q. You've never published peer-reviewed literature on toxicology, right?
A. Correct.
Q. Have you ever taught any courses on toxicology?
A. No, I haven't.
Q. And do you have any experience as a toxicologist?
A. No, I don't.
Q. And you don't have any degrees in environmental health, correct?
A. That's correct.
Q. And you don't have any degrees in occupational medicine; fair?
A. That's correct.
Q. And you're not offering an opinion in this case on whether vinyl chloride causes NHL, right?
A. That's correct.
Q. And you are not an expert in environmental risk assessments; fair?
A. That's fair.

Q. Have you ever conducted any human health environmental risk assessments?
A. No, I haven't.
Q. You did not conduct a Bradford Hill analysis in this case, right?
A. That's correct.
Q. Are you familiar with what Bradford Hill is?
A. Somewhat, from reading the -- the papers and documents that are cited here.
Q. Prior to this case, were you familiar with what a Bradford Hill analysis was?
A. No, I was not.
Q. Your general causation analysis, which if you turn to Exhibit 1 and Exhibit 3, which are your two reports, that's found in the sections "Chemicals at Camp Lejeune." It's on page 2 of Exhibit 1.
A. Okay.
Q. And general causation reports of Dr. Felsher, Hu, Gilbert and Bird, which is the section following it. Is that fair or --
A. I'm not sure I understand your question.
Q. Sure.
Well, let me step back. You offer the same general causation analysis in your reports for

Mr. Howard and Mr. Davis, correct?

A. That's correct, although I always refer to them as specific causation reports.

Q. Sure.

Do you understand the difference between a general causation analysis and a specific causation analysis?

A. Well, I'm not sure I understand completely.

Q. What is your understanding between the difference of a general causation and a specific causation analysis?

A. Well, the specific causation analysis relates to an individual plaintiff and evidence that may relate to their exposure to a noxious agent and the likelihood of developing a disease. And the general causation would be a more overriding review of all of the epidemiological data related to causation of diseases related to exposures.

Q. Sure.

So kind of using those two ideas of what specific causation is versus general causation, you offer the same analysis for both Mr. Howard and Mr. Davis on whether TCE, PCE and Benzene are capable of causing NHL --

A. Yes.
Q. -- is that fair?
A. Yes, that's correct.
Q. Okay. And so in your reports, the opinions are the same for both Mr. Howard and Mr. Davis, correct?
A. Yes.
Q. Okay. So looking at -- or thinking of the general causation opinions, which are both -- are the same for both, they are found in your report for Mr. Davis on -- in your sections that are titled "Chemicals at Camp Lejeune"?
A. Okay.
Q. TCE, Benzene, PCE, ATSDR.
A. Right.
Q. And then general causation reports of Dr. Felsher, Hu, Gilbert and Bird.
A. Right.
Q. Are there any other sections in your report that go to general causation?
A. Well, unless you, you know, refer to my conclusions and the differential diagnosis methodology, but for the most part, I would say that the general causation is in those sections that you cited.

Q. Sure.

And if you turn to Exhibit 3, which is Mr. Howard's report, the general causation opinions you offer are in the sections 4, 5 and 6.

A. Correct.

Q. Were you -- or are you aware that the expert reports in this case are phased such that general causation was considered phase 2 and specific causation is considered phase 3?

A. I'm not familiar with that terminology.

Q. And you reviewed the reports of Drs. Felsher, Hu, Gilbert and Bird; is that fair?

A. Yes. I -- I recall reviewing --

Q. Page 6 --

A. -- Dr. Felsher and Dr. Hu, and the third one, Bird, yes.

Q. I think I asked you this already, but you don't recall whether you've reviewed the report of Dr. Goodman?

A. No. I -- I don't recall reviewing the report of Dr. Goodman.

Q. Have you reviewed the report of Dr. Lipscomb?

A. That name is not familiar.

Q. Have you reviewed the report of

Dr. McCabe?

A. I don't believe so.

Q. Have you reviewed the report of Dr. Shields?

A. Not to my knowledge.

Q. If it would be helpful, you're welcome to look at your material considered lists that were marked as Exhibits 2 and 4.

A. Oh, okay.

Q. And I believe you looked at Dr. Goodman's -- or you've considered Dr. Goodman's transcript.

To the best of your memory, have you reviewed any other of the United States experts' deposition transcripts?

A. To the best of my knowledge, no.

Q. Turning to your report, do you do your own general causation analysis or did you reply -- rely on the reports of Dr. Felsher, Hu, Gilbert and Bird for their determinations?

A. Certainly a lot of it was my own. I may have reviewed Dr. Felsher and -- and Hu's general causation, but I think I put together most of this based on my own review of the materials.

Q. If you turn to page 6 of Exhibit 1.

The section general causation reports of Dr. Felsher, Hu, Gilbert and Bird.

Do you see that section?

A. Yes.

Q. You state (as read):

> I have reviewed and considered the general causation reports of Drs. Felsher, Hu, Gilbert and Bird. Based on my background, education, and experience, the reports of these experts are robust and reliable.

Did I read that correctly?

A. Yes, you did.

Q. What did you do to conclude the reports were robust and reliable?

A. Well, I -- I read their -- their scientific analysis of the data, which, in my mind, seemed rigorous. And their conclusions were reasonable.

Q. Did you review all of the studies that they reference in their reports?

A. No, I did not review all of the studies that were referenced.

Q. The studies that you did review for

general causation are -- those are the studies that you cite to in your report, correct?

A. Yes.

Q. When you were developing your general causation opinions, what steps did you take to do so?

A. Well, I read a number of references, highlighted certain points, took some notes and then I synthesized that together to make this report.

Q. And to gather your references, did you run searches, or what steps did you take to gather your references?

A. I did some searches. Also initially, there was some references, citations provided to me by the attorneys.

Q. Do you recall what searches you ran?

A. They were on PubMed. PubMed searches for the PCA -- PCE, TCE and Benzene related to non-Hodgkin lymphoma.

Q. Do you recall any of the specific search terms you used?

A. PCE, TCE, Benzene, non-Hodgkin lymphoma. Probably also searched spelling out those chemical names.

Q. If you turn to -- or maybe you're still at

page 6 of your report for Mr. Davis.

A. Yes.

Q. You state (as read):

Based on all available evidence, I also agree that TCE, PCE and Benzene all cause NHL at or exceeding the "at least as likely as not" standard.

Did I read that correctly?

A. I'm not sure.

Where on that page is it?

Q. Oh, sorry. So it's the middle of the page. Just above your general causation reports of the other experts.

A. Okay.

Q. I can read it again. It's the last sentence. It says (as read):

Based on available evidence, I also agree that the TCE, PCE and Benzene all cause NHL at or exceeding the "as likely as not" standard.

A. Right.

Q. Did I read that correctly?

A. Yes.

Q. How did you decide to use the at least as likely as not standard?

A. I believe that it was the attorneys that advised me of that standard.

Q. Do you know if that standard is found in the Camp Lejeune Justice Act or not?

A. I don't know.

Q. Have you ever read the Camp Lejeune Justice Act?

A. I don't believe I have.

Q. Other than in this case, have you ever professionally used the standard at least as likely as not when offering an opinion on causation?

A. No, I haven't used that standard.

Q. So if you could keep Davis, page 6, open, and then also open -- or get the Howard report and open that to page 6.

A. Okay.

Q. In the Davis report, there's a paragraph that begins (as read):

> The data from the ATSDR reports.

Do you see that paragraph?

A. That's on page 6 of the --

Q. So on the Davis report.

A. Oh.
Q. I don't see it in the Howard report. That's what I want to ask you.
A. Oh, okay. All right. So back to the Davis report.
Q. Yeah.
A. So what did you want to bring to my attention there?
Q. Sure.
So there's a paragraph. It's the third one from the top.
A. Mm-hmm.
Q. It says (as read):
> The data from the ATSDR reports combined with the meta-analysis related to TCE exposure.

A. Yes.
Q. There's a parentheses (as read):
> Provide compelling evidence that the TCE exposure increases the risk for developing NHL. The ATSDR reports combined with the cohort study of Linet, et al. (2015) provide a similar degree of

evidence for the relationship between Benzene exposure and NHL. Do you see that?

A. Yes, I do.

Q. So if you could -- that paragraph applies equally to Mr. Howard, correct?

A. Yes, it would.

Q. So I didn't see it in the Howard report. That was not intentional or did you intend to leave that paragraph out of the Howard report?

A. No, I didn't intend to leave that out. You know, I could, I suppose, amend the Howard report and include that.

Q. Oh, they're combined? Okay.

MR. MCGOWAN: It appears as though it's just not another paragraph.

MS. HORAN: Oh, thank you, Chad.

MR. MCGOWAN: But it doesn't include -- I'm not saying all the words are identical, but there's something in there that's similar.

BY MS. HORAN:

Q. Okay. So that analysis applies to both Howard and Davis?

A. Yes.

Q. Okay. And then the paragraph above it, in

the Davis report in 2024, "the ATSDR were published," do you see that?

A. Yes.

Q. That paragraph is not in the Howard report, but would apply similarly -- or it's not in the same place in the Howard report.

A. Well, it would not apply because I -- in this paragraph, I deal with the specific diagnosis of marginal zone B-cell lymphoma, which is the cancer that Mr. Davis had, not the cancer that Mr. Howard had.

Q. Do you know if the 2024 Bove study also studied the cancer that Mr. Howard has?

A. Yes, it did.

Q. Okay. Why didn't you include that study for Mr. Howard in your general causation analysis?

A. Well, I -- I think I may have. Let me -- let me go back and look.

Q. So for Howard, if you turn to page 11, you have it in your specific causation analysis --

A. Okay.

Q. -- at the bottom, I think --

A. Yes, that's correct.

Q. -- that's the same study.

A. Yes.

Q. Okay. So there was no intention to leave it out of the general causation part of Mr. Howard's report?

A. No, because I didn't look upon a separation of general causation and specific causation --

Q. Okay.

A. -- when I put together these reports.

Q. Got it. Okay.

So that study applies equally to Mr. Howard as it does to Mr. Davis?

A. Yes.

Q. Okay. So keeping whichever report open, if you could turn back to page 6.

In your opinion, is there sufficient evidence to conclude that TCE causes NHL?

A. In my opinion -- could you rephrase the question, please?

Q. Sure.

A. Or repeat it.

Q. Sure.

Is there sufficient evidence to conclude that TCE causes NHL?

A. Yes. I believe that there is sufficient evidence that TCE causes NHL.

Q. In your opinion, is there sufficient evidence to conclude that Benzene causes NHL?
A. Yes. In my opinion, there is sufficient evidence that Benzene may cause NHL.
Q. In your opinion, is there sufficient evidence to conclude that PCE causes NHL?
A. In my opinion, there's equipoise and above evidence that PCE may cause NHL.
Q. Is equipoise and above the same as sufficient?
A. No.
Q. So in your opinion, there is not sufficient evidence to conclude that PCE causes NHL?
A. There's equipoise and above evidence.
Q. And equipoise and above is below sufficiency -- sufficient evidence for causation, in your mind?
Or how do those terms, "equipoise" and "above" and "sufficient" --
A. Yes.
Q. -- relate?
A. Equipoise and above is a lower level of evidence than sufficient.
Q. So in your opinion -- excuse me.
In your opinion, the evidence of causation

between PCE and NHL does not reach the level of sufficient evidence?

A. That's correct.

Q. Are you offering opinions on each chemical individually being sufficient to cause NHL or collectively mixed, as they've alleged to be at Camp Lejeune, were capable of causing NHL?

A. Well, I believe there's evidence in some studies that individual agents are related to the potential development of NHL, and in other studies, combinations of those agents. There wouldn't be any reason for me to think that the -- there would be any protective effect of multiple agents, that they may be additive or potentially synergistic.

Q. What research did you do into the synergistic impact of the chemicals?

A. Well, only reading papers where people were exposed to more than one of those agents. I have to say I did not uncover anything that appealed to the -- appeared to be synergistic.

Q. What do you mean you didn't uncover anything that appeared to be synergistic?

A. Well, there wasn't any evidence in any studies that indicated risks that would be more than additive.

Q. And you're offering the opinion that, to the standard of at least as likely as not, each of PCE, TCE and Benzene are independently -- or independently could cause NHL; they're not just a contributing factor; is that fair?

A. I'm sorry, but I don't understand your statement.

Q. That's all right.

You're offering the opinion that independently, TCE, PCE and Benzene are, themselves, independently can cause NHL?

A. Each one of those agents may cause NHL, yes.

Q. You agree with the principal that the dose makes the poison, right?

A. I'm sorry?

Q. The dose makes the poison.

Have you heard that before?

A. No, I have not. I'm not familiar with that expression.

Q. Do you agree with it?

A. I'm not familiar with the expression so I can't say I agree with it.

Can you explain it to me?

Q. Sure.

So the dose makes the poison, water can become toxic to an individual if they drink too much water, right?

A. Yes.

Q. But a glass of water would not be toxic; fair?

A. I hope not.

Q. So the dose of how much water you would have would determine whether water is toxic or poisonous to an individual --

A. Right.

Q. -- fair?

With that understanding, would you agree that the dose makes the poison?

A. In general, yes.

Q. Would you agree that the dose of TCE, PCE and Benzene that someone was exposed to is important in determining whether those chemicals are toxic to an individual?

A. Yes.

Q. You would agree that, in general, the risk of developing a disease from a chemical exposure increases with the dose they've been exposed to; fair?

A. In general, yes.

Q. Do you offer the opinion or know what level of exposure to TCE, PCE or Benzene is necessary to cause non-Hodgkin lymphoma?

A. No, I don't offer that opinion.

Q. Do you know?

A. Well, I would go by the studies that I've cited related to levels of exposure and likelihood of developing non-Hodgkin lymphoma.

Q. And you say you don't offer that opinion.

Did you do any analysis to determine whether Mr. Davis or Mr. Howard were exposed at levels necessary to cause -- wherein PCE, TCE or Benzene could cause their NHL?

A. Yes, I was provided with those data.

Q. And how did you determine that there was sufficient exposure to cause PCE, TCE and Benzene -- that TCE, PCE and Benzene could cause their NHL?

A. Well, by the -- the levels of exposure that were provided to me were in excess of levels that were reported in various studies to be related to the development of non-Hodgkin lymphoma.

Q. So you compared the levels that were provided to you of their exposure with the studies that you had reviewed --

A. Yes.

Q. -- fair?
A. Yes.
Q. Is it your opinion that exposure to any amount of TCE, PCE or Benzene is sufficient to cause NHL?
A. There may be threshold effects. I think it's been well demonstrated, for example, in Benzene exposure that very low levels of exposure are not associated with development of non-Hodgkin lymphoma, but after a certain threshold is reached, then they -- the likelihood increases.
Q. So as to Benzene, it's your opinion that there's a threshold amount wherein your risk of developing non-Hodgkin lymphoma would increase --
A. Yes.
Q. -- if exposed to Benzene?
A. Yeah.
Q. Is that your opinion for TCE and PCE as well?
A. I'm not as familiar with data -- strong data that say one way or the other on those chemicals.
Q. Is that something you looked into for your report?
A. Not specifically.

MR. MCGOWAN: We've been going about an hour. Do you want to take five, ten minutes?

THE WITNESS: Sure.

MS. HORAN: Sure.

MR. MCGOWAN: Is now a good stopping point?

MS. HORAN: Yeah. That's fine.

THE VIDEOGRAPHER: The time is now 9:58 a.m. Pacific Time. We're going off the record.

(Whereupon, a recess was taken from 9:58 a.m. to 10:09 a.m.)

THE VIDEOGRAPHER: The time is 10:09 a.m. Pacific Time. We're back on the record.

BY MS. HORAN:

Q. Dr. Hoppe, in doing -- in looking at general causation, you've both done your own analysis and relied upon the work of other experts, Drs. Felsher, Bird, Hu and Gilbert and Milan; is that fair?

A. Yes.

Q. Looking at -- and you're welcome to use your reports, if it would be helpful.

But can you identify any studies where PCE, TCE or Benzene exposure was found to have caused NHL at a dose that is similar to what is

alleged here?

A. I would have to say that my recollection is that in the various reports that I cited, the exposure levels were less than those that the plaintiffs experienced at Camp Lejeune.

Q. So generally, the studies you cited, you tried to cite studies where the exposure was less to PCE, TCE and Benzene at Camp Lejeune.

A. I didn't -- sorry for interrupting.

I didn't try to, but I think that those studies just happen to -- you know, they, in general, were lower levels of exposure.

Q. You would agree that people with no exposure to TCE, PCE or Benzene can still get NHL, right?

A. Yes. I agree.

Q. The exact relationship between the interactions of TCE, PCE, Benzene and vinyl chloride is not known, correct?

A. I'm not aware of any studies showing any interaction.

Q. I believe you said that you -- it was your understanding that there was an additive effect between TCE, PCE and Benzene; is that fair?

A. I don't think I said that.

Q. Okay. What is your understanding of the relationship between TCE, PCE and Benzene exposure?

A. Well, that each of them independently may be associated with the development of non-Hodgkin lymphoma. I think it's difficult to define additivity or synergy when -- when -- when multiple exposure -- exposure to different chemicals is apparent.

Q. So are you offering an opinion on this case on this synergistic of additive effects of TCE, PCE, Benzene and vinyl chloride on developing NHL?

A. No. I'm not offering an opinion on the additivity.

Q. Association is not the same as causation, correct?

A. Can you be more specific?

Q. When you're looking at a study, it might tell you association or it might tell you causation; fair?

A. Yes.

Q. Is it your understanding that when looking at whether, for example, TCE causes NHL, an association is not the same as a causation; is that fair?

A. I think that's a semantic issue. I'm

not -- I'm not sure I can answer that.

Q. So if a study showed that two -- that TCE was associated with the development of NHL, you would view that study as showing that TCE causes NHL?

A. That it may cause.

Q. May cause?

A. Yes.

Q. Okay. And what is the distinction you're drawing in "may cause" as opposed to "causes" when an association is shown between a particular chemical and the development of a disease?

A. Well, cause means -- if it causes, that means you have come to the conclusion that the etiologic agent is responsible for the development of the disease. If you say may cause, then it's possible that the agent caused the disease.

Q. So generating an opinion on causation is not as simple as just having a study that shows a statistically significant association between a substance and a disease, correct?

A. Could you repeat that, please?

Q. Sure.

Generating an opinion on causation is not as simple as just having a study that shows a

statistically significant association between a substance and a disease; fair?

A. I'm sorry, but that's a lengthy statement. Can you --

Q. Sure. Sure.

A. Before I say I agree or not.

Q. That's fair.

So you said that association, in your view, is the same as "may cause"; fair?

A. Yes.

Q. And "may cause" can be used as evidence of causation --

A. Yes.

Q. -- correct?

So if you're creating an opinion on causation, having a study that shows association or may cause is not in and of itself sufficient to provide you with enough evidence to decide -- to provide an opinion that something would cause the disease?

A. Not -- not one study that simply shows that.

Q. So you would not draw a conclusion about causation from just a single study showing an association or a may cause?

A. That's correct.
Q. And it's possible to have a study that shows statistically significant association or may cause between a substance and a disease and still not be able to conclude that the substance causes the disease; fair?
A. In an individual case.
Q. If there's a study that shows -- just one study that shows a statistically significant association, you'd want to see that study replicated to some degree so you can rule out that its findings were a result of chance, right?
A. Yes.
Q. And you would want to assess all of the available epidemiological literature on evaluating causation before you drew your opinion on causation, right?
A. Within reason, yes.
Q. What do you mean, "within reason"?
A. Well, the entire -- to review the entire field and all of the papers that have been published would be a challenge.
Q. Why would that be a challenge?
A. Because there are many hundreds of articles related to exposures and development of

cancer, and, you know, this is not my primary responsibility in terms of my career to do these type of analyses.

Q. And when you say this is not your primary part of your career, you're referring to determining whether a particular substance can cause a disease?

A. No. I'm -- I'm referring to my role on this -- in this case versus the rest of my responsibilities in life.

Q. And when you say your role in this case, are you referring to your specific causation -- your differential diagnosis, or what are you referring to?

A. I'm referring to my respons- -- my overall responsibilities in this case.

Q. Okay. How, then -- or I understood you to say there could be hundreds of articles about a particular chemical causing a disease.

How do you determine you've reviewed enough of them in order to be able to offer your opinion that a particular substance causes a disease?

A. Well, I judge the quality of the journals and the frequency of citations of different articles in -- in my -- in reading the literature.

Q. And if you continue to see the same ones you've known, you've seen a mass of them, or how does that work?

A. Yeah. So, you know, if I read, for example, a multivariate analysis and it cites publications that have a large number of patients with long followup, then I might go to that specific paper more selectively than a report that has a small number of patients and very limited followup.

Q. The authors of an epidemiological study provides statistical results that indicate the risk -- the levels of risk observed; fair?

A. Can you repeat that?

Q. The authors of an epidemiologic- -- epidemiology study provide statistical results that indicate the levels of risk observed; is that fair?

A. Yes.

Q. And it's important to analyze those risks odd ratios in a study, in the study results; fair?

A. Yes.

Q. And the risk ratio indicates the level of association observed?

A. Yes.

Q. And 1.0 or 1 indicates no association, correct?

A. Well, that indicates no significant increase.

Q. Right.

So it indicates that there's -- strike that.

What do you mean, "no significant increase"?

A. Well, that -- that would be, you know, conventionally, 95 percent likelihood that there's no increase in incidence.

Q. In your opinion, what level of increased risk reflects a moderate association?

A. I would say anything above 1.

Q. And what level of increased risk reflects a sizeable association?

A. Well, a hazard ratio in excess of, I'd say, 1.3.

Q. So anything over 1 reflects a modest association and anything over 1.3 reflects a sizeable association, in your opinion?

A. Yeah, I've never thought of it in that way, but I think that would be a reasonable way to define it.

Q. How do you typically think about it, if not in the increased risk as the risk ratio

increases?

A. Well, usually it's not in such quantitative terms. It's sort of more you know it when you see it.

MS. HORAN: I'm marking as Hoppe Exhibit 5. This is the United States Environmental Protection Agency, dated April 30th, 2019. The subject is Pyrethroids: Tier II Epidemiological -- Epidemiology Report.

(Whereupon, Deposition Exhibit 5 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, have you seen this before?

A. It's not familiar to me, so I'd have to say no.

Q. Okay. Could you turn to page 10? Or I guess turn to page 8.

Do you see Section 5 says, "Data evaluation"?

A. Yes.

Q. And then Section 5.2 says, "Study review and quality assessment"?

A. Yes.

Q. If you could turn to page 10. I guess about the third paragraph down, do you see it says

(as read):

Risk assessments -- strike that.

(As read):

Risk estimates, estimates of effect reported in epidemiological studies were generally characterized as follows.

And then there's five bullets that follow.

Do you see that section?

A. Yes.

Q. The first bullet reads (as read):

No evidence of a positive association between exposure and outcome, which is, e.g., OR equals 1, OR is less than 1.

Do you agree with that?

A. Yes.

Q. The second bullet reads (as read):

No evidence of a significant positive association, EGOR is greater than 1, but not significant.

Do you agree with that?

A. Yes.

Q. The third bullet reads (as read):

Evidence of a slight positive association, e.g., OR is greater than 1, but less than 1.3 and significant.

Do you agree with that?

A. Well, not -- it's not consistent with what I previous- -- previously said to you, would be my take on a modest increase and a substantial increase.

Q. Where did your belief that a modest association is between 1 and 1.3 come from?

A. Well, that's -- my feeling that if you have a risk of developing a disease of 1.3, that that's substantial.

Q. And how would you -- or strike that.

In a risk of developing a disease at 1.01, how would you characterize that?

A. That was -- that would be a minimal increase in risk.

Q. Turning back to Exhibit 5, the fourth bullet reads (as read):

Evidence of a positive association, e.g., OR is greater than equal to 1.3 or less than 2

and significant.

Do you agree with that statement?

A. I agree that that would be a positive association, yes.

Q. And then the last bullet (as read):

Evidence of a moderately strong, e.g., OR is greater than equal to 2 or less than 3 and significant or strong, e.g., OR is greater than or equal to 3 and significant positive association.

Do you agree with that bullet?

A. I agree with that bullet.

Q. It's -- you can set that exhibit aside.

It's important to analyze confidence intervals in a studies results, correct?

A. Yes.

Q. And a confidence interval evaluates how precise the risk assessment is; fair?

A. Yes.

Q. The wider the confidence interval, the less confidence in the point estimate; fair?

A. Fair.

Q. A confidence interval that includes 1 or the null suggests no association; is that fair?

A. Well, it suggests that a -- an association could not be demonstrated.

Q. And a confidence interval that includes less than 1 suggests that the association could not be found, correct?

A. Well, it suggests that it could not be found at the statistical level of significance, as it's commonly defined.

Q. Turning to dose, when the odds ratio increases as exposure increases, you can be more confident that an association is present, correct?

A. Yes.

Q. And considering the amount of exposure is -- or the dose is a more precise way to classify an exposed population than just exposed versus unexposed, correct?

A. Could you repeat that?

Q. Sure.

Assessing the dose that someone was exposed to is a more precise way to classify an exposed population than just as exposed versus not exposed?

A. Yes.

Q. An opinion on causation in the absence of a dose response should explain why a dose response

was not seen; fair?
A. I'm sorry.
Q. Sure.
A. Could you repeat that?
Q. An opinion on causation in the absence of a dose response should explain why a dose response wasn't seen; fair.
A. Reasonable.
Q. And that's because you would expect to see a dose response if there was causation, right?
A. It may be difficult at times, I think, to demonstrate that a dose response relationship exists.
Q. Why would that be difficult to demonstrate?
A. Well, if -- if a disease occurs with high frequency at even low levels of exposure, it may be hard to demonstrate an even higher risk with higher exposures.
Q. So if someone drew a conclusion about that chemical causing a particular disease and there wasn't a dose response for the reason you just stated, you would expect the paper to say as much, correct?
A. Yes. Yes.

Q. Would you agree that the number of participants in a study is relevant to the power of that study?

A. Generally, yes.

Q. In general, a study with a larger number of participants in the investigative population carries more weight than a study with a fewer number of participants; fair?

A. Yes.

Q. In assessing epidemiological evidence, how do you compare the value of a cohort study with an ecological study?

A. I'm not so familiar with ecological studies.

Q. What do you -- if you -- what do you mean, you're not so familiar with an ecological study?

A. I'm not -- I'm not sure about that term.

Q. Fair enough.

What is a cohort study, to the best of your understanding?

A. So a cohort study is where two different populations are compared and -- to look at incidence of events in one population versus another.

Q. And then I believe you said you're not familiar with the term "ecological studies"?

A. No.
Q. So you don't have an opinion on whether cohort studies or ecological studies are more valuable in an epidemiological evidence review?
A. Correct.
Q. I want to turn to your opinions on Mr. Davis.
I believe you testified that you have not communicated with Mr. Davis himself. But have you spoken to any of his family members, physicians or friends?
A. No, I've not spoken with any of them. I believe I had access to deposition by treating physicians.
Q. Sure.
But you didn't pick up the phone to call anyone --
A. No, I did not.
Q. -- about Mr. Davis?
So you've written two reports. Obviously, one on Mr. Davis and one on Mr. Howard. Some of your studies you reference in both reports, some studies are only in one. I -- to the extent you have any question about which plaintiff or if my question is not clear as to which plaintiff, or your

answer only applies to one plaintiff, please just ask for clarification throughout this day. But I will do my best to --

A. Okay.

Q. -- to be clear about who I'm talking about.

Deal?

A. Yes.

Q. Okay. Could you turn to Mr. Davis' report, which is marked as Exhibit 1, and turn to page 6?

A. Okay.

Q. The section "Concentrations of Contaminants at Camp Lejeune."

Do you see that?

A. Yes.

Q. And in determining the concentration of contaminants for which individuals were exposed, in that first paragraph, you reference Appendix H1 and state (as read):

It appears that the concentrations of PCE -- I believe you meant were --

A. That's correct.

Q. (As read):

-- in excess of 100 micrograms per liter of water peaking at 182 micrograms per liter in June 1984. Do you see that?

A. Yes.

Q. Are you offering any opinion on the reliability of the mean monthly concentrations of the various contaminants as determined by Mr. Maslia in his report?

A. No. I relied on those reports.

Q. Sure.

And you -- strike that.

And you took them at face value as correct?

A. Correct.

Q. How did you -- or why did you include or compare the June 1984 micrograms with 100 micrograms?

A. Are you asking why I used 100 micrograms.

Q. That's right.

Why 100 micrograms as a benchmark?

A. Well, I -- I don't think that was so much a benchmark as that was kind of the -- the approximate level of the minimum concentration reported during that period of time.

Q. And why report the minimum concentration?
A. Well, because, you know, they -- there are, you know, established levels of concentrations, that if they're exceeded, are associated with events.
MS. HORAN: I'm marking as Exhibit 6, Hoppe Exhibit 6. This is the expert report of Morris Maslia.
(Whereupon, Deposition Exhibit 6 was marked for identification.)
BY MS. HORAN:
Q. Dr. Hoppe, have you seen this report before?
A. No, I haven't.
Q. You see this is the expert report dated October 24th, 2024?
A. So I think I've seen portions of this report. But not -- not the entire report.
Q. Okay. Exhibit 6, though, you do see that that's the Morris Maslia report from -- dated October 24th, 2024?
A. Yes.
Q. If it's helpful to you, please reference Davis' report as needed.
But could you turn to page 185?

Do you see this is Appendix H1?
A. Yes.
Q. Is this the appendix you were referencing in your report for Mr. Davis?
A. All right.
Q. And if you could turn to page 195 of Exhibit 6.
A. Okay.
Q. Actually, if you could turn to page 196.
A. Okay.
Q. You see stress period 402, it's the 7th from the bottom, it says June 1984?
A. Yes.
Q. And then for single species using MT3DMS, it shows 182.13 PCE --
A. Right.
Q. -- micrograms per liter?
A. Right.
Q. Is that the number you were referencing in your report?
A. Yes. Although, I rounded it off to 182.
Q. Sure.
But this is where you got that number from?
A. Yes.

Q. And then if you keep looking on that row, it goes to -- there's four more columns, and across the top it says (as read):

Multispecies, multiphase using TechFlowMP model.

Do you see that?

A. Right.

Q. And that reports 158.14 micrograms per liter PCE.

Do you see that?

A. Right.

Q. So why did you decide to rely on the MT3DMS model instead of the TechFlowMP model in your report?

A. I don't recall.

Q. Do you know the difference between the MT3DMS and the TechFlowMP models?

A. I believe I read at the time, but I don't recall.

Q. You would agree that Tarawa Terrace, where Mr. Davis lived for a portion of his time on base -- and you have the chart in your report if you would like to reference it.

But you would agree that this H1 does not reflect that there was Benzene at Tarawa Terrace,

correct?

A. I would have to go back and look at this.

Q. Okay.

A. I don't recall.

Q. Turning back to Exhibit 1, which is your report for Mr. Davis, do you see the section that says (as read):

Camp Lejeune connection to NHL and Mr. Davis?

A. I'm sorry, do you have a -- oh, yeah. Okay.

Q. So middle of the page says (as read):

Camp Lejeune connection to NHL and Mr. Davis.

A. Right.

Q. The second paragraph, third sentence, says (as read):

According to the ATSDR 2017 report, Hadnot Point Chapter A fact sheet.

A. Yes.

Q. Do you recall which ATSDR report you were referencing there?

A. Well, I'd have to say it was the ATSDR 2017 report, Chapter A.

Q. Would that be -- if you could turn to your materials considered list, which for Mr. Davis is Exhibit 2.

A. Okay.

Q. Do you see number 2 is ATSDR public health assessment for Camp Lejeune --

A. Right.

Q. -- drinking water?

A. Right.

Q. Do you recall reviewing that study?

A. So I recall that I reviewed it. I can't recall specifics of what it says.

Q. Okay. Turning back to Exhibit 1, which is your report for Mr. Davis, if you could turn to page 10.

A. Okay.

Q. Do you see at the top of the page it says (as read):

> Similarly based on the Maslia report, Appendix H2, the water at Tarawa Terrace exceeded the MCL for those -- these contaminants for the entire time Mr. Davis was on base?

A. Yes, I see that.

Q. And could you turn back to Exhibit 6,

which is the Maslia report?

A. Okay.

Q. If you could turn to page 198.

Do you see Appendix 198 says -- strike that.

Do you see page 198 says (as read):

Appendix H2 Tarawa Terrace Water Treatment Plant reconstructed (simulated) mean monthly finished water concentration of single-specie tetrachloroethylene, PCE and a range of concentrations derived from the Monte Carlo simulation?

A. Yes.

Q. This is the Appendix H2 that you relied on or reference in your report of Mr. Davis on page 10, correct?

A. Right. Right.

Q. You would agree that Appendix H2 talks only about PCE?

A. Yes.

Q. Why reference the Monte Carlo simulation on page 10 of your report and then the MT3DMS numbers on page 6 of your report?

A. Can you tell me again where Appendix H1 was?

Q. It started on page 185.

A. I can't say with certainty, other than the fact that both of them -- both of those appendices indicated that there was an excess amount of PCE in the groundwater at those times.

Q. And excess in terms of what?

What are you referencing as the benchmark for excess?

A. Well, the absolute levels --

Q. Okay.

A. -- exceeded the -- the MCL.

Q. Are you offering an opinion on whether the MT3DMS, TechFlow or Monte Carlo simulation numbers should be used to determine exposure?

A. No. Well, I'm not -- I'm not saying that one or the other is preferable.

Q. Do you know if Dr. Reynolds relied on MT3DMS, TechFlow or Monte Carlo simulations in determining Mr. Davis' exposure?

A. I'm not aware.

Q. Okay. You can set aside Exhibit 6.

You rely on Dr. Reynold's exposure assessment for determining how much PCE, TCE and

Benzene Mr. Davis was exposed to; fair?

A. Yes.

Q. Did you do your own exposure analysis in addition to Dr. Reynolds or are you relying on Dr. Reynolds?

A. I'm relying on Dr. Reynolds.

Q. So you did not independently calculate the amount of TCE, PCE or Benzene to which Mr. Davis was exposed to during his time at Camp Lejeune?

A. I did not.

Q. If you turn to page 6 of your report, the last sentence says (as read):

> Considering his days at these locations and cumulative contaminant exposure concentrations, and based upon his deposition-based informed activities, his cumulative consumption total micrograms equals days multiplied by concentration per deposition exposure assumptions for TCE was 2,036,600 micrograms, for PCE was 503,816 micrograms, and for Benzene was 33,244 micrograms.

Did I read that correctly?

A. Yes, you did.
Q. Part of that sentence says (as read):
Based upon his deposition-based informed activities.
What did you mean by that?
A. Well, I believe that Dr. Reynolds took into account what Mr. Davis reported in his deposition regarding his exposure, which I detail at some point.
Q. Perhaps page 10.
A. Yeah. At the -- towards the bottom of page 10, according to testimony, Mr. Davis, and then I list four different responses that he gave in his deposition regarding his exposure.
Q. So if you turn to page 11, the first sentence, those are the same numbers as you provide on page 6; fair?
A. Yes.
Q. And these are the numbers you relied on in forming your opinion as to whether Mr. Davis' NHL was caused by his exposure to TCE, PCE and Benzene?
A. Right.
Q. Did you do any investigation into the reliability of Dr. Reynolds' exposure assessment?

A. No.
Q. Have you ever personally calculated the cumulative consumption of an individual of a particular chemical?
A. No, I haven't.
MS. HORAN: I'm marking as Exhibit Hoppe Exhibit 7. This is the cumulative exposure expert report of Kelly A. Reynolds. It's dated February 7th, 2025.
(Whereupon, Deposition Exhibit 7 was marked for identification.)
BY MS. HORAN:
Q. Dr. Hoppe, have you seen this report before?
A. I've seen portions of it.
Q. Could you turn to Appendix 17, which is about two-thirds of the way through or so?
A. Okay.
Q. And I'm just going to give you a sticky note if you want to just mark that page in case you ever want to reference it again.
Okay. Do you see Appendix 17 is for Cometto J. Davis?
A. Yes.
Q. Okay. And if you turn to page 1 of

Appendix 17, it's the Davis exposure data summary chart.

A. Okay.

Q. The numbers you attribute to Dr. Reynolds in your report are not in Dr. Reynolds' report for Mr. Davis, correct?

A. Can you repeat that?

Q. Sure.

So if we look back at your report, on page 6 or page 11, you would agree that Dr. Reynolds' does not opine that Mr. Davis was exposed to 2,036,600 micrograms per liter of TCE?

A. Well, I would have to review more of this to say that these numbers are not what he said.

Are you indicating to me that --

Q. We can step back.

Where did you get these numbers from Dr. Reynolds that are in your report?

A. I believe that tables provided to me by the attorneys included these numbers, tables from Dr. Reynolds.

Q. Okay. Okay. Well, do you have any reason to -- if you turn to the first page of Exhibit 7, which is Dr. Reynolds' report.

A. I'm sorry, which page?

Q. Just the cover.

Do you see that it's signed by Dr. Reynolds on the bottom?

A. Yes.

Q. Do you have any reason to doubt that this is a complete and accurate copy of Dr. Reynolds' expert report as provided in the case?

A. I have no reason to doubt.

Q. Okay. And you would agree that Appendix 17 states Cometto J. Davis' name, which is the name of the individual that you -- that you're offering specific causation analysis on?

A. Yes.

Q. And you would agree at page 1 of Appendix 17 at the bottom says, "Davis exposure data summary"?

A. Yes.

Q. And you would agree that 2,036,600 micrograms of TCE exposure is not found in this chart as attributed to Mr. Davis?

A. That -- that exact number is not listed in this table.

Q. And you would agree that 503,816 micrograms per liter of PCE is not listed in Davis' exposure data summary table?

A. Correct.
Q. And you would agree --
A. It is not listed in the table that I'm looking at.
Q. And you would agree that 33,244 micrograms per liter of Benzene is not listed in the Davis exposure data summary?
A. That's correct.
Q. Kind of looking through the rest of Exhibit 17 [sic], if you flip through it, it's about eight or so more pages.
To the best of your memory, have you seen these charts before?
A. I don't recall seeing these exact charts.
Q. Turning back to page 1 of Appendix 17, you see the first column says (as read):
Cumulative micrograms per liter-capital M.
I think that's for month.
A. Right.
Q. Do you understand what micrograms per liter-M means?
A. Yes.
Q. What does it mean?
A. Well, it means micrograms per liter per

month of exposure.

Q. So is it the addition of all of the months that Mr. Davis was exposed to the chemical or --

A. It's per month.

Q. So you see -- okay.

So it's your understanding that based on this chart, that Mr. Davis was exposed to 29,132 micrograms per liter per month of TCE, or is it cumulatively he was exposed -- if you add it --

(Simultaneous speakers - inaudible.)

THE WITNESS: I would say that's per months, yeah.

BY MS. HORAN:

Q. So you would say he -- this is stating that he was offered -- he was exposed to 29,132 micrograms per liter per month?

Sorry, I'm sorry. Sorry. On average.

A. Per month, yes.

Q. So on average, he was exposed per month to 29,132 micrograms per liter of TCE?

A. Correct.

Q. And then the same would follow for PCE, VC and Benzene?

A. Correct.

Q. Okay. And you would agree that columns 3,

4, 5 and 6, which associate with across the top charts, 1, 2, 3 and 4, are in total microgram?

A. Which columns did you say?

Q. So they're the third, fourth, fifth, sixth column over, and they're associated with charts 1, 2 3 and 4.

Those are in total micrograms?

A. Right.

Q. Okay. And so those are numbers that Mr. Davis was exposed to as an absolute amount of TCE, PCE, VC or Benzene; fair?

A. Yes.

Q. Do you have an opinion upon whether chart 1, chart 2 -- or strike that.

Do you have an opinion on whether it's most appropriate for your purposes as a specific causation expert to apply the numbers in chart 1, chart 2, chart 3 or chart 4 to Mr. Davis?

A. I would say chart 2.

Q. And why is that?

A. Well, because he was a Marine in training stationed there. And his deposition supported certain additional exposures --

Q. What are you referencing --

A. -- and this also -- sorry.

Q. I didn't mean to cut you off.
A. Chart 2.
Q. Mm-hmm. Sorry. I think you said referenced additional exposures.
What did you mean by that?
A. From his deposition.
Q. Oh, as referenced in your report?
A. Yeah. Yeah.
Q. Okay. Turning back to your report, on page 6, the numbers that you attribute to Dr. Reynolds are in micrograms.
Do you see that?
A. Yeah.
Q. And then if you turn to page 11.
A. Of my report?
Q. Of your report, yes, which is Exhibit 1.
The numbers you attribute to Dr. Reynolds are in microgram per liter month.
Do you see that?
A. Yes.
Q. What is -- is it your opinion that micrograms and micrograms per liter per month are the same unit?
A. No.
Q. What is the unit that you intended to use?

A. So -- so I can see that I inadvertently put per month. This should probably be just micrograms per liter in the -- in the report.

Q. Flipping back, I guess, to Dr. Reynolds' report, which is Exhibit 7, Appendix 17, you see Dr. Reynolds has opinions in micrograms per liter month and total micrograms --

A. Right.

Q. -- fair?

A. Right.

Q. So in your report, did you intend to use total micrograms?

A. Yes.

Q. Okay. So your opinion is based on an absolute number of exposure to the various contaminants as opposed to an average exposure over the month?

A. Yes.

Q. And why did you determine that an absolute number for amount of exposure is the proper way to assess someone's likelihood of developing NHL?

A. Well, 'cause that -- that takes into account the duration of the exposure.

Q. How does that take into account the duration of the exposure?

A. Well, it's the total amount, so it's the entire time that somebody was exposed.

Q. Sure.

But you would agree that an individual could have been exposed to 100 micrograms of TCE in one day or they could have been exposed to 5 micrograms per TCE over 20 days and both absolute would be 100 micrograms, right?

A. Yes.

Q. So turning to page 11 of your report, the second sentence says (as read):

> In my opinion, the level of exposure to TCE, PCE and Benzene that Mr. Davis experienced during his 1,000-plus days living and working on Camp Lejeune was more than sufficient to cause his NHL.

Did I read that correctly?

A. Yes.

Q. So in doing your analysis, you took into account the number of days Mr. Davis was at Camp Lejeune; fair?

A. Yes.

Q. Did you do any analysis to determine, based on the total exposure, the amount Mr. Davis

was exposed to on a daily or monthly basis?
A. No.
Q. And you don't use the numerical measure of parts per billion in your report, do you?
A. No, I don't.
Q. Is it your understanding that parts per billion is the same as micrograms?
A. I think it would be the same.
Q. What is that based off of, that you think it would be the same?
A. Well, I think they're both measures in concentration.
Q. Did you do any analysis as part of your work for this case in determining whether micrograms and parts per billion are a one-to-one ratio for conversion?
A. No, I didn't.
Q. Have you seen any published studies where they study the total exposure of an individual to a particular dose as opposed to looking at a dose over time to determine if Benzene, TCE or PCE caused NHL?
A. Can you repeat that?
Q. Sure.
Have you seen any published studies or do any come to mind where they studied a total exposure

of an individual as opposed to a dose over time that an individual was exposed to for PCE, TCE or Benzene?

A. Well, I guess I don't quite understand the distinction between the two as you -- as you asked them. Can you --

Q. Sure.

So have you seen any published studies where they studied that someone was, for example, exposed to 100 micrograms of TCE as opposed to a study that might say they were exposed to 5 micrograms of TCE over 20 months?

Does my question make more sense now?

A. I'm not sure it does.

Q. Okay. Do you recall any studies that the study looked at the -- just the absolute amount of exposure to PCE, TCE or Benzene and determined whether it caused NHL?

A. Yes.

Q. And which study was that?

A. I would have to go back and look.

Q. It would be in your report, though?

A. Yeah.

Q. Okay. You can set aside Mr. Davis' report and Dr. Reynolds' report, but if you'd like to

reference either at any point, you're welcome to.

Do you know one way or the other whether the use of total mass of ingested chemicals is a standard exposure metric in risk assessment?

A. Can you repeat that?

Q. Sure.

Do you know whether Dr. Reynolds' use and your use of the total mass of ingested chemicals is a standard exposure metric in a risk -- risk assessment?

A. I don't know.

Q. Do you know whether the United States put forward an expert in risk assessment for Mr. Davis or Mr. Howard?

Have you seen any report like that?

A. I haven't seen any reports.

Q. Okay. Do you know whether total mass ingested is generally accepted in the field of toxicology?

A. I don't know.

Q. Is total ingestion metric a methodology that you commonly use in your practice in determining a toxic exposure?

A. No, I don't commonly have patients who have toxic exposures.

Q. And when you do have a patient who has a toxic exposure, would you use the total ingestion metric as a methodology for determining their risk of developing NHL?

A. No, I wouldn't.

Q. You did not identify a threshold amount of exposure to TCE, PCE or Benzene, or a mixture thereof, whereby an individual is likely to develop NHL; fair?

A. I didn't indicate in my report. But a subsequent paper that's come to my attention by Dr. Yu indicated a threshold, a very clearly defined threshold for Benzene.

Q. Have you identified any papers or have any opinion on a threshold amount for TCE or PCE?

A. No.

Q. And it's your opinion that the Yu paper identifies a threshold whereby an individual is likely to develop NHL if exposed to Benzene?

A. Right.

Q. If a patient of you -- yours asked for a threshold amount whereby Benzene could cause their NHL, would you point them to the Yu paper?

A. I would suggest that they have an analysis done by one of these experts first.

Q. When you say, "one of these experts," are you talking about a risk assessment expert or what expert are you referencing?

A. Someone who could do an analysis related to what their Benzene exposure was.

Q. Have you ever recommended a patient get that done?

A. No.

Q. And is that because you've never had a patient who would -- had been exposed to Benzene or is that why -- why haven't you recommended someone get that done?

A. Yes. It's because I've never had a patient who had a history of exposure to Benzene where that was an issue.

Q. And then at the point that the patient got the exposure analysis or risk assessment done, would you then apply those numbers to the Yu study to determine the threshold of likelihood or what would --

A. Yes.

Q. If you could reopen Exhibit 1, which is Mr. Davis' expert report, and turn to page 11.

Your sentence that says -- that starts "in my opinion," it's the second sentence down, do you

see that?

A. Oh, yes. I'm sorry.

Q. It looks like in that sentence, you reference the level of exposure that you got from Mr. -- or Dr. Reynolds and the number of days Mr. Davis was living and working at Camp Lejeune to determine that it was more than sufficient to cause his NHL.

Other than Dr. Reynolds' numbers and the number of days Mr. Davis was there as in over 1,000 days, was there anything else that you relied on in determining it was more than sufficient to cause his NHL?

A. No.

Q. What methodology did you use to determine more than sufficient?

A. Well, the studies that I cited in the general causation statements, in addition to the fact that he had no other risk factors for developing non-Hodgkin lymphoma.

Q. So the studies in general causation and then your differential diagnosis methodology; is that fair?

A. Yes.

Q. You reference -- in your report on page 9,

you reference the MCLs.

Do you see that?

A. Yes.

Q. What is your understanding of what an MCL is?

A. So that's the contaminant level above which there is significant risk and, you know, it's recommended by agencies, such as the EPA, that contents be kept below those levels.

Q. Do you know whether the EPA uses MCLs which are maximum contaminant levels to evaluate potential risks to human health?

A. Yes.

Q. And do you know how the EPA establishes an MCL?

A. Not exactly, but I assume they review the data that had been published in scientific studies.

Q. Are you aware that MCLs are designed to be acceptable daily drinking water concentrations over a lifetime of exposure of about 70 years?

A. Yes.

Q. Are -- sorry. I didn't mean to cut you off.

Are you aware of the health protective assumptions that go into determining an MCL?

A. No.
Q. Do you know whether the EPA uses cumulative dose averaged over a lifetime to evaluate cancer risk?
A. I don't know.
Q. Would you agree that an exposure to drinking water concentration in excess of the MCL does not necessarily constitute a health risk?
A. If it's of limited frequency.
Q. Page 7 of your report, do you see the top paragraph?
A. Yes.
Q. And you offer the opinion that Mr. Davis would have been exposed through inhalation and dermal routes as well; fair?
A. Right.
Q. You did not quantify Mr. Davis' exposure via dermal or inhalation; is that fair?
A. That's fair.
Q. And why not?
A. I left that to the experts.
Q. And by the "experts," you're referencing Dr. Reynolds?
A. Yes.
Q. You say to -- okay.

The fourth sentence in that paragraph reads (as read):

> To a reasonable degree of medical probability, these additional routes would add to Mr. Davis' exposure during his day-to-day activities and add to his risk of developing NHL from exposure to the water at Camp Lejeune.

And then you have a cite to a Weisel study from 1996.

A. Right.

Q. Other than the Weisel study from 1996, do you rely on anything else for that proposition?

A. No.

Q. Other than the Weisel 1996 study, do you have -- excuse me. I'll start over.

Other than the Weisel 1996 study, are there any other studies you have in support of the proposition that dermal and inhalation routes would add to his risk of developing NHL?

A. I don't have specific studies, no.

Q. How did you determine that it would add to his risk of developing NHL?

A. Well, by absorption, as is described in this report, absorption through dermal exposure, that that will increase the concentration above and beyond what is ingested.

Q. Anything beyond that study?

A. No.

Q. If you turn to page 10 of your report, you have a section titled "Mr. Davis' time and exposure at Camp Lejeune."

A. Right.

Q. Are you aware that not all parts of Camp Lejeune have been alleged to be part of the water systems that were contaminated?

A. I believe I have seen maps showing areas that were contaminated and -- yeah. That not all areas were contaminated.

Q. Do you know one way or the other whether Camp Geiger was contaminated?

A. I don't recall. I would have to look -- look back at those reports.

Q. Underneath the chart, the first sentence reads (as read):

> During his time at Camp Lejeune, Mr. Davis would do normal day-to-day activities,

eating, drinking, showering and cleaning with the contaminated water provided by the water system. Do you see that sentence?

A. Yes.

Q. And you -- or I don't -- did you assume or was this outside -- was this Dr. Reynolds' sphere whether 100 percent of the water Mr. Davis was exposed to was contaminated or not?

A. I -- I did not assume, and I relied on the reports of the experts to define what the exposure was.

Q. Okay. So number 1 below that sentence, the first one reads (as read):

> Was on field duty for extended periods where he would live on -- in the field and get his water from what he referred to as a water bowl.

Do you see that?

A. Yes.

Q. You don't make any determination about whether the water in those water bowls was contaminated or not; fair?

A. Correct.

MR. MCGOWAN: Are you at a stopping point? I don't want to --

MS. HORAN: Yeah.

MR. MCGOWAN: -- interfere with your mojo.

MS. HORAN: Just one moment, and then I think we're good to break.

MR. MCGOWAN: That's fine.

MS. HORAN: Yeah. We're good to take a break now.

MR. MCGOWAN: Okay.

THE VIDEOGRAPHER: The time is 11:28 a.m. Pacific Time. We're going off the record.

(Whereupon, a recess was taken from 11:28 a.m. to 11:43 a.m.)

THE VIDEOGRAPHER: The time is 11:43 a.m. Pacific Time. We're back on the record.

BY MS. HORAN:

Q. Dr. Hoppe, in your expert report for Mr. Davis, which is marked as Exhibit 1 on page 7, you have a section that says "Plaintiff Cometto Davis."

Do you see that?

A. Yes, I do.

Q. And this section contains your summary of his medical history; is that fair?

A. That's correct.
Q. How did you go about developing the summary of Mr. Davis' medical history?
A. I reviewed all of the medical records that were submitted to me by the attorneys, and I think are listed in the beginning of my report.
Q. Were there any medical records you wished you had been able to see but were not provided to you?
A. No. Along the way, there may have been things that I inquired of that were then found and submitted to me.
Q. But everything that you were looking for, in terms of being able to provide this summary, was provided to you?
A. Yes.
Q. And you never spoke to any of his treating providers; fair?
A. Correct.
Q. On page 7, the third paragraph down, the fourth sentence says (as read):

> He had an uncertain history of asbestos exposure.

Do you see that?
A. Yes.

Q. Why did you include that as part of Mr. Davis' medical history?

A. Well, asbestos exposure is associated with the development of lung cancer and other cancers in the thorax, and so prior to having a biopsy diagnosis of lymphoma, one of those cancers would certainly be in the differential diagnosis based on the imaging.

Q. Is asbestos exposure a risk factor for developing NHL?

A. No.

Q. On page 8, the third paragraph down, the second sentence begins (as read):

> He tolerated chemotherapy relatively well. On July 5th, '22, he underwent an end-of-treatment PET CT. This revealed improved size of lung lesions, which remained non-hypermetabolic. The plan was to enter a surveillance phase with regular follow-up examinations. Did I read that correctly?

A. Yes.

Q. Mr. Davis has completed treatment for NHL?

A. Yes.
Q. And Mr. Davis is just being monitored for any recurrence of NHL at present, correct?
A. Correct.
Q. The bottom of page 7, the last paragraph, the fifth sentence reads (as read):
He had longstanding neuropathy in his distal lower extremities.
Did I read that correctly?
A. Yes.
Q. And then on page 8, the fifth paragraph, reads (as read):
Mr. Davis had suffered from peripheral neuropathy for more than ten years, but this appeared to be more bothersome during his chemotherapy.
Can we go off the record for a moment, please?
THE VIDEOGRAPHER: The time is 11:47 a.m. Pacific Time. We're going off the record.
(Whereupon, a recess was taken from 11:48 a.m. to 11:48 a.m.)
THE VIDEOGRAPHER: The time is 11:48 a.m. Pacific Time. We're back on the record.

BY MS. HORAN:

Q. Dr. Hoppe, we were just looking at the -- Exhibit 1, Mr. Davis' report, the fifth paragraph down, which reads (as read):

> Mr. Davis had suffered from peripheral neuropathy for more than ten years, but this appeared to be more bothersome during his chemotherapy. He was evaluated by neurology and thought to have a small fiber sensory neuropathy. Following the completion of chemotherapy on August 27th, 2022, an EMG suggesting demyelinating sensory motor axon loss. Repeat EMG on March 30th, 2023 showed a moderately severe sensorimotor mixed axonal and demyelinating polyneuropathy, chemotherapy-induced neuropathy was considered as a possible aggravating factor.
>
> Did I read that correctly?

A. Yes.

Q. Are you aware that Mr. Davis has tested

positive for HNPP?
A. No.
Q. Did you consider Mr. Davis' positive test -- strike that.
So you didn't consider Mr. Davis' positive test for HNPP as part of his medical history?
A. What do you define as "HNPP"?
Q. We'll get to that in just one second.
You would agree that NHL did not cause Mr. Davis' neuropathy?
A. Yes, I agree.
Q. And you agree that Mr. Davis had neuropathy before NHL and before chemotherapy; fair?
A. Yes.
MS. HORAN: I'm marking as exhibit -- Hoppe Exhibit 8. These are UNC Health medical records 00043_DAVIS_VC_0000000380 through -457.
(Whereupon, Deposition Exhibit 8 was marked for identification.)
BY MS. HORAN:
Q. Dr. Hoppe, have you seen these records before?
A. I looked at many medical records, including some from UNC Health, but I don't recall if these specific records I have seen.

Q. If you could turn to the Bates ending in -387.
A. The what?
Q. So the Bates number at the bottom, those are the -- the stamped copies for the litigation.
A. Oh, okay.
Q. The page ending in -387.
A. Right.
Q. And you see that this -- these are records for a visit date of March 30th, 2023?
A. Right.
Q. And your report references a March 30th, 2023 appointment; fair?
A. Yeah. So I did see these records, yes.
Q. Great.
Could you turn to the page ending in -390?
The -- do you see a section with conclusions?
A. Yes.
Q. The last two sentences read (as read):
Given the multiple entrapments, HNPP is in the differential. Superimposed chemotherapy-induced neuropathy and paraproteinemic neuropathy may need

to be considered as well.
Did I read that correct?
A. Yes.
Q. So on May 3 -- excuse me, March 30th, 2023, the differential was HNPP.
A. Okay.
Q. Do you know --
A. It was in the diff- -- in the differential.
Q. What's the distinction?
A. I'm sorry?
Q. Sorry.
I think I said is the differential, and you said is in the differential.
What distinction were you drawing there?
A. Well, HN- -- excuse me. HNPP -- and I'm not certain what HNPP is referring to, okay -- that etiology or disease was in the differential means that it was among the diseases or causations to consider, okay. And then the following sentence says (as read):
Superimposed chemo, chemotherapy, induced neuropathy and paraproteinemic neuropathy may need to be considered as well.

Q. And that's what you have in your report when you say chemotherapy-induced neuropathy was considered as a possible aggravating factor?

A. Yes.

Q. Okay. If you could turn to Bates ending in -401.

A. Right.

Q. Do you see this is notes from, again, the March 30th, 2023 appointment?

A. Right.

Q. And then if you turn to -402, under "History of Present Illness," it says (as read):

> Chief complaint is numbness, tingling and burning in his feet for the last ten years or so.

A. Yes.

Q. And then about the eighth sentence down reads (as read):

> Symptoms have been gradually progressive over the years.

A. Yes.

Q. And if you turn back to -382.

A. Okay.

Q. The top says -- the first paragraph -- strike that.

You see this is a telephone appointment note with Mr. Cometto Davis from May 9th, 2023?

A. Mm-hmm. Yes.

Q. And it says -- this is Dr. Chaudhry (as read):

> I talked with the patient. I told him that his genetic testing for a disease called HNPP is positive.

Do you see that?

A. Yes.

Q. And you're not familiar with HNPP?

A. I don't know what the abbreviation stands for.

Q. Could you turn to page -423?

A. Okay.

Q. Do you see the section -- and I apologize. This is not the clearest copy, but this is what we have.

Do you see where it says (as read):

> What is a positive PMP22 result?

A. Yes.

Q. And the last sentence in that section says (as read):

Different positive PMP22 variants can cause hereditary neuropathy with liability to pressure policies (HNPP).

A. Oh, okay.

Q. Do you see that?

A. Yes. That defines HNPP.

Q. Are you familiar with hereditary neuropathy with liability to pressure policies?

A. No.

Q. Does it suggest to you that it's a hereditary type of neuropathy?

A. Yes.

Q. So if you could turn back to -382.

A. All right.

Q. Does -- Dr. Chaudhry states in these notes with his call that he told Mr. Davis that he had HNPP, which we saw is a hereditary type of neuropathy; fair?

A. Right.

Q. You can set those aside.

And you did not take into account Mr. Davis' hereditary neuropathy in your assessment or summaries of his medical history, correct?

A. No. But I did acknowledge that he had had

peripheral neuropathy for ten years. I didn't speculate on the cause, but I didn't -- didn't hide the fact.

Q. And are you offering the opinion that Mr. Davis' neuropathy was made worse by his chemotherapy treatment?

A. I think it may very well have been made worse. In fact, in Dr. Chaudry's note on -382, he says if she -- and I --

Q. One second. Do you mind if I just get to the page? I can work with you.

You're on -382 of Exhibit 8?

A. Right.

Q. Okay.

A. His concluding sentence is (as read):

> If she needs chemotherapy, we should avoid neurotoxic drugs given his underlying neuropathy due to genetic causes.

Now, this is already after the fact. He already had received the neurotoxic drugs, but I think Dr. Chaudhry was saying if he should need chemo in the future to avoid neurotoxic drugs.

Q. Sure.

And do you know -- look at your report,

anything you need to know -- what neurotoxic drugs Mr. Davis was exposed to, if any, in his chemotherapy regimen?

A. Yes. Vincristine.

Q. Vincristine.

Any others?

A. No. Others are not associated with neurotoxicity.

Q. You think it's possible vincristine could have made Mr. Davis' neuropathy worse; fair?

A. Yes.

Q. But you have not talked to Mr. Davis about that specifically?

A. No, I haven't.

Q. Is there any medical records that you're relying on for that opinion that it may have made his chemo -- his neuropathy worse?

A. Well, it's well known medically that vincristine causes neurotoxicity. Oftentimes the drug has to be discontinued because of the severity of that neurotoxicity. And it's medically reasonable to assume that someone who already has neuropathy, it would be made worse with neurotoxic drugs, exactly as Dr. Chaudhry specifies in his note.

Q. Did you look at any records from -- strike that.

Would you agree that to the extent vincristine -- am I saying that correctly, vincristine?

A. Vincristine.

Q. Vincristine.

You agree that to the extent vincristine worsened Mr. Davis' neuropathy, any worsening would stop with the end of treatment?

A. No.

Q. What do you rely on for that?

A. Well, my experience in seeing patients who have had vincristine exposure, that the neuropathy often continues for many months after the completion of chemotherapy. And in occasional patients, they have residual neuropathy that doesn't recover.

Q. Sure.

Does the neuropathy just not recover or does the neuropathy get worse after you stopped using vincristine?

A. Some patients reported getting a little worse before it gets better. So the maximum severity is not necessarily at the very conclusion of treatment. It may get a bit worse before

potential improvement occurs.

Q. And so for how long would it get -- at what point would you stop telling a patient the vincristine is what's making their neuropathy worse?

A. Probably -- well, I would expect in a patient who doesn't have any underlying neuropathy who has been treated with vincristine who develops neuropathy that it shouldn't get any worse beyond two to four months after the completion of treatment.

Q. And then, at that point, it may get better?

A. At that point, it often gets better.

Q. And for a patient such as Mr. Davis who has a genetic neuropathy, when would you tell a patient with a genetic neuropathy that the vincristine was no longer the cause of their worsening neuropathy?

A. Well, if it's -- if it's progressing beyond a -- a couple months from the completion of treatment, I would say it's not likely that the progression is due to vincristine.

Q. And what -- did you look at any studies for that, or is that based on your experience as a physician?

A. It's based on my experience.

MR. MCGOWAN: Can we take five minutes to get this whole lunch thing squared away?

MS. HORAN: Absolutely. Can we go off the record?

THE VIDEOGRAPHER: Time is 12:05 p.m. Pacific Time. We're going off the record.

(Whereupon, a recess was taken from 12:05 p.m. to 12:09 p.m.)

THE VIDEOGRAPHER: The time is 12:09 p.m. Pacific Time. We're back on the record.

MS. HORAN: I'm marking as Hoppe Exhibit 12. This is a study by Li, 2024, that's titled "Characterizing vincristine-induced peripheral neuropathy in adults: Symptom development and long-term persistent outcomes."

(Whereupon, Deposition Exhibit 12 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, have you seen this study before?

A. No, I haven't.

Q. Could you turn -- strike that.

Are you familiar with any of the authors of this study?

A. No, I don't know any of these authors.
Q. Could you turn to --
A. They're all from Australia.
Q. -- page 8?
Do you see the second paragraph on page 8 begins "The coasting"?
Do you see that paragraph?
A. Yeah.
Q. The study says (as read):
The coasting phenomenon whereby symptoms worsen after end of treatment was also previously associated with VIPN. However, the present study incorporating multiple assessment techniques found that symptoms had reached their peak by the end of treatment. As observed in a prior study, symptom improvement occurred soon after treatment completion with significant improvements in three months post vincristine.
Did I read that correctly?
A. Yes.
Q. Do you agree with that finding of the

study?

A. Well, that was the findings that the authors present in this study. I wouldn't disagree with what they found in their study.

Q. Do you agree with it --

A. Well --

Q. -- in your professional experience and expertise?

A. I would -- I would agree also with the first sentence that you read that there's a coasting phenomenon whereby symptoms worsen after the end of treatment.

I sometimes see that in patients, or sometimes they have reached their maximum at the end of treatment.

I don't think I stated that every patient sees an increase in neuropathy after completion of treatment. Some patients do.

Q. You can set that aside.

You also include in your report Dr. Jayaram's concern that Mr. Davis was developing multiple myeloma; is that fair?

A. Yes.

Q. You're not offering an opinion or analysis in this case about whether the water at Camp Lejeune

caused Mr. Davis' multiple myeloma?
A. No.
Q. You're not, correct?
A. I am not.
Q. And you're not offering an opinion or analysis in this case on whether NHL can cause multiple myeloma; fair?
A. No, I'm not.
Q. You're not offering that opinion?
A. No.
Q. Would you agree that there's no known cause for most -- most cases of NHL?
A. Yes.
Q. What percentage of NHL cases that you've sought to understand the cause of have had an unknown cause?
A. Perhaps 10 percent.
Q. So 90 percent of the cases that you've sought to have an understanding of what caused the NHL you've been able to determine the cause?
A. I have not been able.
Q. Oh.
A. 90 percent I have not been able to.
Q. And about 10 percent of the time you have been able to --

A. Yes.
Q. -- determine the cause?
Prior to this case, have you thought -- strike that -- have you sought to understand the cause of marginal zone lymphoma in an individual?
A. Yes.
Q. How many times?
A. Most of the time that I see a patient with marginal zone lymphoma.
Q. They often ask you what the cause was?
A. No. I often consider what the cause might be.
Q. And when you consider the cause of marginal zone lymphoma in your patients, how often have you been able to determine the cause?
A. About a third of the time.
Q. And two-thirds of the time, you've not been able to determine the cause of marginal zone lymphoma?
A. Right.
Q. The one-third that you've been able to determine the cause of, what have those causes been?
A. Generally, it's been chronic Helicobacter pylori infection related to gastric marginal zone

lymphoma.

Q. Have you ever determined marg- -- the cause of marginal zone lymphoma in a patient's lung?

A. No.

Q. Do you know if there are any unique causes of marginal zone lymphoma in an individual's lung?

A. There have been some cases where there's been implication of a Cronobacter infection.

Q. And the Cronobacter infection leads to multiple marginal zone lymphoma in the lung?

A. It -- it causes inflammation that is thought to lead to marginal zone lymphoma of the lung.

Q. Anything besides the Cronobacter?

A. No.

Q. Different subtypes of NHL have different risk factors; fair?

A. Yes.

Q. Would you agree that known causes of marginal zone lymphoma do not account for the mast -- vast majority of marginal zone lymphoma found in the United States?

A. Yes.

Q. Are you familiar with the term "idiopathic"?

A. Yes.
Q. And what does that mean?
A. It means unknown cause.
Q. So if I use the term "unknown cause" throughout today, you'll understand -- or if I use the term "idiopathic," you'll understand they mean the same thing?
A. Yes.
Q. And you have done a differential diagnosis methodology for both Mr. Howard and Mr. Davis; fair?
A. Yes, I have.
Q. What is a differential diagnosis methodology?
A. Yes. Well, in this context, the differential diagnosis is considering all known risk factors for, in this case, developing non-Hodgkin lymphoma and considering each of them and ruling them in or out as potential causes.
Q. And you said, "in this case."
Is that -- did you do something in this case different than what you do in your general practice in terms of differential diagnosis methodology?
A. Well, in medical practice, differential diagnosis refers to something slightly different,

okay. It's seeing an abnormality, for example, on a chest X-ray and listing the possible findings as -- as to their significance. So it lists different entities. So it's a slightly different usage of the term compared to how it's used in this document.

Q. And how did you use it in this document?

And you pointed to your report, which is Exhibit 1?

A. Yeah. So in this document, I used it in the context that had been described to me by the attorneys as appropriate to look at for causation. And that is to examine each of the potential causes of lymphoma and to define whether Mr. Davis or Mr. Howard had that characteristic or that exposure or that history that would contribute to the lymphoma.

Q. And the methodology that you used in this case and the way you used the term "differential diagnosis methodology," do you also do that in your practice as a physician?

A. Not quite in the same formal way.

Q. What do you mean by that?

A. Well, you know, if a -- if a patient, for example, has an organ transplant, then the immunosuppression that's associated with that organ

transplant may result in the development of lymphoma. And one need not go through -- I -- I feel that, you know, all of the potential risk factors for developing lymphoma when one is obvious.

Q. As applied to this case, the differential diagnosis methodology you used requires ruling in all reasonable potential causes of the NHL; fair?

A. Yes.

Q. Did you consider an idiopathic or no known cause in your differential diagnosis in this case?

A. Well, by definition, that's unknown, so I dealt with the -- with the known risk factors.

Q. And did you consider that most NHLs have an unknown cause at all in your analysis?

A. Sure.

Q. How did you consider it as part of your analysis?

A. Well, if there was no other factor that could be identified as a risk factor, then I would have to conclude that it was idiopathic.

Q. So if there's any possible risk factor that you've not been able to rule out, then you don't -- then you would consider that to be the known cause of the ailment?

A. So if there's no known risk factor.

Q. Let me rephrase the question. There's a lot of negative.

A. Okay.

Q. If you have not been able to rule out a none risk factor that you've ruled in, then you consider that to be the cause of the ailment?

A. I'm sorry, I'm confused. I'm confused what you're saying.

Q. Sure.

So if you have a known risk factor of NHL, and you've ruled that in because it's a known risk factor --

A. Yes.

Q. -- and you haven't ruled it out for a particular patient, then you would consider that known risk factor to be the cause?

A. Yes.

Q. Okay.

A. Or a likely cause.

Q. What -- you said known or likely cause.

What's the distinction you're drawing there?

A. What was the -- what were the two --

Q. I said a known cause, and you said or a likely cause.

And I said -- and I'm just wondering what distinction you were drawing there between telling someone it was a known cause or telling it was a likely cause.

A. Oh. Well, I would have to say that if it's a known cause, that it's also a likely cause.

Q. Is it your opinion that idiopathic etiology can only exist in the absence of known risk factors?

A. It can exist -- only exist in the absence of known risk factors, but there may be factors -- risk factors that are unknown.

Q. Okay. So if you can turn to page 12 of your report. And this is the report of Mr. Davis. This is Exhibit 1. Your last paragraph reads, from the beginning (as read):

> Given the otherwise rarity of this disease, the low grade immunosuppression suffered by Mr. Davis for an extended period, the lack of any other risk factors for the development of NHL, e.g., immunosuppression, autoimmune disorders and the increased risk for marginal B-zone lymphoma in a

person -- in personnel at Camp Lejeune versus Camp Pendleton in the ATSDR report, I conclude that it is more likely than not that Mr. Davis' exposure to TCE and Benzene increase the risk of and was more likely than not a significant and substantial contributing cause of his B-cell marginal zone lymphoma.

Did I read that sentence correctly?

A. Yes.

Q. Okay. So it looks like you've identified four factors, the first being rarity of this disease.

Is that fair?

A. Yes.

Q. On page 11, the second paragraph from the bottom, discusses that it's your -- the NH- -- strike that.

You say on page 11, the second paragraph from the bottom (as read):

> Adding to my conclusion, the NHL that Mr. Davis suffered from is an uncommon one, only 8 percent of

NHL, and his presentation is uncommon for marginal zone B-cell lymphoma.

Do you see that?

A. Yes.

Q. Do you have -- where did you get the statistics in this paragraph from?

A. Well, firstly, my general knowledge, but then I likely looked up to be very specific with respect to the 8 percent number. And what -- what source I looked at for defining that must have been some large database, but I can't tell you exactly which one.

Q. Further on in the paragraph, you state (as read):

The lung is involved as a primary --

(Reporter clarification.)

BY MS. HORAN:

Q. I'll go slower.

(As read):

The lung is involved as a primary site for this lymphoma only about 1 percent of the time.

A. Yes.

Q. Do you see that?
Do you have a cite for that statistic?
A. Yeah. It was probably the same source. I'm not sure if it's addressed in one of the papers that I cite here on marginal zone lymphoma. But, again, it's my -- my general experience. I -- I see these marginal zone lymphomas of the lung very infrequently compared to marginal zone lymphomas of the skin or the stomach or the ocular adnexal structures. So they're relatively uncommon even among marginal zone lymphomas.
Q. Why does the rarity of the disease lead you to believe it's related to water exposure at Camp Lejeune?
A. Yeah. Well, I -- it's a combination of the -- the report from the ATSDR looking at the comparison of Marines stationed at Pendleton versus Lejeune where there was a remarkable increase in risk for this particular type of lymphoma. And -- and that -- which is otherwise an uncommon lymphoma. That's one of the reasons that -- that led me to that conclusion.
Q. And the study you're referencing is on page 12, the second paragraph from the bottom?
A. Right.

Q. It's that paragraph.
A. Right.
Q. And the statistics -- or the risk that you were just referencing is the 1.45 95 percent confidence interval of .92, 2.28?
A. Yes.
Q. And you would agree that the confidence interval for the ATSDR study of 2024 includes the null of 1?
A. Yes.
Q. Other than the ATSDR study and your -- of 2024 and your experience, is there anything else that led you to come to the conclusion that the rarity of the disease suggests it's related to water at Camp Lejeune?
A. No.
Q. Is the alternative true, if it's a common NHL, then it's likely not caused by Camp Lejeune water?
A. No.
Q. Why not?
A. Well, I -- I just said that the rarity was not necessarily a result of the Camp Lejeune, and then -- so it's the reverse, that the common lymphomas wouldn't necessarily be related, if I'm

understanding you correctly.

Q. Sure.

In Mr. Davis' report, you reference the rarity of the disease as a factor you thought made it more likely that it was from the Camp Lejeune water.

A. Yes.

Q. And we can go, and we can look now if you want, but I didn't see you discussing the rarity or commonality of diffuse large B-cell lymphoma that Mr. Howard as part of your analysis in that --

A. Right.

Q. -- opinion.

And so I'm wondering if -- is it just that it's a rare disease that leads you to that understanding, or is it the commonality of a disease that is somehow part of or related to Camp Lejeune?

A. I guess it's just, you know, the rare- -- rarity of the disease makes it provocative to find an explanation.

Q. The second thing you list on page 12 of your factors is the low-grade immunosuppression suffered by Mr. Davis for an extended period.

Do you see that?

A. Yes.

Q. And that -- the further explanation of that is the bottom of page 11, going to the page -- top of page 12.

A. Right.

Q. And you say at the bottom of page 11 that Mr. Davis was lymphopenic for three years preceding his diagnosis?

A. Yes.

Q. Are you offering the opinion that lymphopenia is a cause of Mr. Davis' NHL?

A. No. I would say that his lymphopenia may be a contributing factor to developing NHL.

Q. And what is that -- what studies or what is that based off of?

A. Well, that's based off of the fact that lymphocytes are important in -- as part of the immune system, including surveillance of foreign antigens or proteins, and that if you have a deficiency of lymphocytes, that may lead to the development of lymphoma or other cancers, most notably, for example, at the extreme end of the spectrum, people infected with the HIV virus where there is a severe depletion in certain subsets of the lymphocytes, you're at very high risk for developing non-Hodgkin lymphoma.

Q. Are there studies that say that -- so understanding that HIV is -- and those types of illnesses, those are extreme immunosuppressed, that low level immunosuppression would have the same -- or would have an impact on developing NHL?

A. Whether it's cause or effect is unclear. But certainly for lymphomas, if they occur in the presence of a low lymphocyte count, that's an adverse factor.

Q. And you attribute his lymphopenia to his exposure to TCE; is that fair?

A. Yes.

Q. And you say (as read):

> Studies cited above indicate that exposure to TCE may cause lymphopenia.

What studies were you referencing?

A. Whatever studies were above.

Q. So those, are they the studies found on page 2 and 3 in your TCE section, or somewhere else?

A. Yes.

Q. It looks like you might have put your finger on some -- which studies are you referencing --

A. So, for example, on page 3, the third

paragraph, Bassig, et al., 2016.

Q. Any others?

A. That's -- that's the main one here.

Q. Did you apply your differential etiology methodology for determining the cause of Mr. Davis' lymphopenia?

A. No.

Q. Did you consider any other risk factors for determining -- for the cause of lymphopenia?

A. No.

Q. Turning to page 12 again, the third thing you listed as part of your conclusion was (as read):

> The lack of any other risk factors for the development of NHL, e.g., immunosuppression, autoimmune disorders.
>
> Do you see that?

A. Yes.

Q. Do you believe that all NHLs have the same risk factors or do they differ by subtype?

A. They differ by subtype.

Q. And for Mr. Davis, did you consider only risk factors specific to marginal zone lymphoma?

A. I -- I considered the risk factors specific to marginal zone lymphoma as well as the

risk factors for lymphoma in general.

Q. How did you determine what are the risk factors for NHL in general and specifically marginal zone lymphoma?

A. From my general experience and -- and knowledge of the lymphomas.

Q. Did you run any searches in to publications or anything of that nature?

A. Regarding --

Q. Risk factors?

A. -- risk factors? No.

Q. Do you have any studies that you rely on in determining risk factors for NHL or marginal zone lymphoma?

A. Well, you know, I've read studies over the years. So it's really a combination of all of that knowledge.

Q. But sitting here today, there's not one in particular --

A. No.

Q. -- that you can point me to?

What are the risk factors in your view -- we'll do generally NHL first and then marginal zone lymphoma separately.

A. Okay.

Q. So in your opinion, what are the risk factors for developing non-Hodgkin's lymphoma generally?

A. They can include immunosuppression, infection with certain viruses, Epstein-Barr virus most notably, exposure to chemical agents. What else did I consider here? Those are the main ones.

Q. And what are the risk factors you considered for marginal zone lymphoma?

A. Chronic infection and inflammation secondary to infection.

Q. The chronic infection, is that the barium bacterium you were talking about?

A. The Cronobacter, yeah.

Q. Yeah.

The Cronobacter is the infection that you would consider?

A. Yes.

Q. Any other infections you would consider?

A. Well, for marginal zone lymphoma of different organs, there are different infectious agents.

Q. Ah, okay. So for the lung, you just considered the Cronobacter --

A. Right. Cronobacter.

Q. Sorry, could you just say it one more time?

A. Cronobacter.

Q. Cronobacter.

Any other risk factors you can think of sitting here today?

A. Not for marginal zone lymphoma, other than those that are generally contributory to non-Hodgkin lymphoma.

Q. And other than the three you've listed, immunosuppression, infection with Epstein-Barr virus and exposure to chemical agents, are there any other risk factors for developing non-Hodgkin's lymphoma?

A. Well, there may be very remote genetic predispositions. We know that from family studies. But it's not a common causality.

Q. And do you mean a family history of NHL or what are you referencing?

A. Yeah.

Q. Anything else?

A. No.

Q. Earlier today -- and I might have misunderstood -- I think you said when you were working with a patient to try to understand the cause of their ailment, you would ask them their

social history in addition to their occupational history. And the social history you would ask about would include tobacco, alcohol, drug exposures and sexual histories.

Am I remembering that correctly?

A. Yes. Yes.

Q. Are tobacco, alcohol, drug exposures and sexual histories part of the risk factors for developing NHL, or why ask those of your patients?

A. Well, they're always important with respect to tolerance for treatment, okay, or likelihood to adhere to treatments. So in -- in the general context, it's important to know about those things.

With respect to lymphoma and etiologic agents, tobacco is not associated, alcohol is not associated. If there is, you know, risk for HIV infection, for example, then that needs to be tested for, but that could -- that could be a causative factor.

Q. And you also ask about drug exposures.

Why drug exposures?

A. Well, to know whether the patient is at -- at risk for abusing agents or whether their previous history of drug use might affect their tolerance for

treatment.

Q. Okay. So drug -- prior drug exposures is not considered in your view a risk factor for developing NHL?

A. No.

Q. Is it possible that -- strike that.

Do you consider age as a risk factor for developing NHL?

A. Well, lymph- -- certainly the risk for developing lymphoma increases with age.

Q. And is that also true of marginal zone lymphoma?

A. Yes.

Q. So marginal zone lymphoma becomes more common in men as they age?

A. Yes.

Q. How much more common has marginal zone lymphoma become in men as they age?

A. Well, it -- it probably plateaus at a certain point, because certainly, you know, it's not common in men in their 20s or 30s. But it increases, probably plateaus in the 60s.

Q. You state on page 11, the fifth paragraph (as read):

The only known or potential

cause of the NHL here was the exposure to water at Camp Lejeune. Do you see that?

A. Yes.

Q. Is it possible that Mr. Davis' non-Hodgkin's lymphoma is idiopathic and there's actually no known cause?

A. Yes, it's possible.

Q. The paragraph following that sentence says (as read):

> For instance, there is no evidence of any other exposures to toxins either at home or at work, see, e.g., Davis.

And you have a cite to his deposition transcript. (As read):

> There is also no evidence of any family history that is germane.

Another cite to a deposition transcript (as read):

> No evidence of other exposures in the Marines.

Another cite transcript to his deposition (as read):

And no medical history of any NHL precursors.

And, again, you have a cite to the deposition transcript.

Do you see that?

A. Yes.

Q. So all of your understanding of the risk factors and Mr. Davis' application of them to Mr. Davis came from his deposition?

A. No. There was some statements in medical records that I reviewed.

Q. Do you recall what you saw in medical records that you reviewed as to his risk factors?

A. Well, for example, family history was noted on a number of occasions. And his past medical history was recorded on a number of occasions in his medical records.

Q. Other than his deposition and medical records, are there any other places you found relevant statements about his risk factors?

A. No, I don't think so.

Q. Did you have any risk factors that you were not able to -- that you considered that you were not able to rule out either through his medical history or his deposition?

A. No.
Q. You state no medical history of any NHL precursor.
What did you mean by "NHL precursor"?
A. So, for example, history of chronic infections that have been implicated in developing marginal B-cell lymphoma, and no medical history of prior EBV infection that might be a contributor to developing non-Hodgkin lymphoma.
Q. Other than the factors listed in the paragraph we've been referencing that begins with, "for instance," are there any other risk factors that you ruled in or ruled out as to Mr. Davis in your differential diagnosis?
A. I think that covers it.
Q. Your next paragraph says (as read):
Considering the potential causes of the NHL in this case, and the weak or absent evidence for all but the water at Camp Lejeune, I am left with the conclusion that the contaminated water was more likely than not the cause of Mr. Davis' NHL.
What did you consider as weak evidence of

other causes?

A. Well, I'd say weak or absent. And primarily, it was absent.

Q. Were there any other causes that you considered to have weak evidence?

A. No.

Q. In the paragraph with deposition citations, you reference no evidence of other exposures to toxins.

What other toxins would be of concern that you would be thinking of in assessing that?

A. Well, for example, you know, defoliants like Agent Orange.

Q. Anything else?

A. You know, herbicides that have been implicated, insecticides that have been implicated.

Q. Anything else you can think of?

A. Not offhand.

Q. You would agree that most marginal zone lymphoma patients do not have a family history of NHL?

A. Correct.

Q. You state in the paragraph with the deposition citations (as read):

No evidence of any family

history that is germane.

When you say, "family history that is germane," are you referencing family members with non-Hodgkin's lymphoma or what would be a germane family history?

A. Yeah, no -- none that is germane, meaning that none -- there's no family history of any lymphoma, like one of his parents or -- had colon cancer or some other solid tumor, but no history of lymphomas in the family.

Q. You would agree that there are marginal zone lymphoma patients with no known toxic exposures, no known germane family history and no medical history of any NHL precursor?

A. Yes.

Q. And you would agree that as to age, Mr. Davis is a typical -- is typical of patients diagnosed with marginal zone lymphoma?

A. Yes.

Q. And you -- strike that.

You determined the Camp Lejeune water caused Mr. Davis' NHL because it's the only factor left ruled in to your differential diagnosis?

A. Yes.

Q. And if you turn to page 6 of your report,

the paragraph in the middle that says, "based upon my years of experience," includes the sentence (as read):

> Based on available evidence, I also agree that TCE, PCE and Benzene all cause NHL at or exceeding the at least as likely as not standard.

Fair?

A. Fair.

Q. Have you ever assessed a risk factor for NHL before where you ruled it in as a cause that's at least as likely as not?

A. Well, I -- I hadn't used this terminology until this case came to my attention. And then I reviewed the ASTDR's recommendations -- ATSDR's recommendations for definitions.

Q. So how do you weigh a factor that's as likely as -- strike that -- that's --

How do you weigh a factor that's as least as likely as not cause in a differential diagnosis?

A. Well, by the weight of evidence from multiple studies that have looked at the relationship between exposure and development of lymphoma.

Q. And how did you weigh that against the fact that most non-Hodgkin's lymphomas and marginal zone lymphomas are of an unknown cause?

A. Well, because if I were to apply the differential diagnosis approach, in most cases of marginal B-cell lymphoma, with the exception of those that arise in the stomach, I would not be able to identify any risk factor as a causation.

Q. So in weighing your conclusion that TCE, PCE and Benzene all cause NHL at least as likely or not standard, they reach that at least, and when you weigh that against the fact that known -- most marginal zone lymphomas and NHLs in general have no known cause, without this opinion as to TCE, PCE and Benzene, you would be left with an unknown cause?

A. But we have a cause.

Q. Sure.

And I'm -- I'm trying to understand how you weigh the standard you've applied to TCE, PCE and Benzene with the known fact that most NHLs and marginal zone lymphomas have no known cause. Is it that once you've ruled it in from your general causation analysis, it's now -- do you, at that point, consider the weight of the evidence as part of your differential diagnosis or is it just fully

ruled in 100 percent?

Does that make any sense?

A. Not quite, but --

Q. Well, let me try again.

So you've ruled in TCE, PCE and Benzene causing NHL at a standard that is, at minimum, at least as likely as not; fair?

A. Yes.

Q. Okay. And when you take that ruling-in factor and you apply it to your differential diagnosis methodology, at that point, is it a binary it's been ruled in, it's been ruled out, or do you still consider the sufficiency of the evidence or the amount of evidence you've used to rule it in when you weigh it against other factors?

A. I guess I still don't understand exactly what point you're trying to make. You know, in this case for Mr. Davis, we do have his exposure history. We do have a known relationship between these exposures and the development of lymphoma in multiple studies, some of which are cited and mentioned here. So he has the risk factor.

Q. Sure.

And once you've determined he has the risk factor, do you consider that risk factor against the

evidence supporting unknown causation of most NHLs or is that no longer part of your idiopathic, no longer part of your assessment because you've ruled in a known possible risk?

A. Yes. Idiopathic is only if you've ruled out all possible risk factors.

Q. Understood.

A. So essentially it's what we call a diagnosis of exclusion.

Q. Okay. So is what you did a diag- -- what did you just call it? A diagnosis of exclusion?

A. Diagnosis of exclusion if you rule everything else out.

Q. Okay. If you turn to page 12 of your report.

You also -- your fourth factor that you considered was this -- the 2024 ATSDR report in Camp Lejeune versus Camp Pendleton --

A. Right.

Q. -- fair?

And how would you characterize the strength of the finding of ATSDR as a two marginal zone B-cell lymphoma of 1.45 with a 95 percent confidence interval of .92, 2.28?

A. So I would consider the hazard ratio of

1.45 to be quite high, and that given the small numbers of people with that diagnosis in either Camp Lejeune or Camp Pendleton, it's -- it's no surprise that the 95 percent confident -- confidence intervals are what they are.

But that's simply, you know, the 95 percent of the bell-shaped curve and that there's parts of the bell-shaped curve above and below those 95 percent estimates. And I would attach significance to the 1.45 hazard ratio. It was not a marginal hazard ratio. It wasn't 1.1 or 1.05. It was 1.45.

MS. HORAN: I'm marking as Exhibit 9.

Sorry, what?

MR. LEE: Is that Number 10 or 9?

MR. MCGOWAN: We skipped a couple.

MS. HORAN: 9. Yeah, we actually went to 12, and now we're going to go back to 9. Sorry.

BY MS. HORAN:

Q. This is the Bove study titled "Cancer Incidence Among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at U.S. Marine Corps Base Camp Lejeune: A Cohort Study." That's Hoppe Exhibit 9.

(Whereupon, Deposition Exhibit 9 was marked for identification.)

BY MS. HORAN:

Q. This is the study, Dr. Hoppe, that you reference in your report, correct?

A. Yes.

Q. Do you know whether the 2024 incident study performed any statistical significance testing?

A. Yes. The -- the numbers that they report include the confidence intervals.

Q. Do you know if the study has any individualized exposure assessment?

A. I don't think it does, no.

Q. Do you know if the study controlled or considered other potential occupational exposures among Marines or Navy service members?

A. No. I believe it assumed roughly equivalence because they were -- they just so happened to be assigned to different base camps.

Q. Turn to Table 3. It says (as read):

Comparison of cancer outcomes at Camp Lejeune versus Camp Pendleton among the Marine/Navy personnel subgroup who

began active duty and were stationed at either base between 1975 and 1985.

Do you see that?

A. Yes.

Q. All right. And if you look down, marginal zone B-cell lymphoma is maybe 20 percent up from the bottom.

A. Right.

Q. There were 43 cases.

A. Right.

Q. Do you see that?

And the adjusted hazard ratio is 1.45 and then with a confidence interval of .92, 2.28?

A. Mm-hmm.

Q. So this is where you got that statistic for your report?

A. Yes.

Q. And if you look up on this study, do you see the overall non-Hodgkin's lymphoma heading?

A. Yes.

Q. And you see there were 550 cases of non-Hodgkin lymphoma?

A. Right.

Q. And the adjusted hazard ratio is 1.01?

A. Yes.
Q. With a confidence interval of .90, 1.14?
A. Yes.
Q. And you would consider -- would you consider 1.01 to show an association or not?
A. A very weak association with the overall diagnosis of non-Hodgkin lymphoma.
Q. And then if you look a few down, do you see diffuse large B-cell?
A. Yes.
Q. And diffuse large B-cell is what Mr. Howard has, correct?
A. Yes.
Q. And you relied on this study in forming your opinions as to Mr. Howard, correct?
A. Yes. I -- I mentioned the civilian experience, not the Marine experience.
Q. Sure.
And as to Table 3, which is what you applied to Mr. Davis --
A. Yeah.
Q. -- diffuse large B-cell lymphoma has a hazard ratio of .89 and a confidence interval of .72, 1.10?
A. Right.

Q. So you would agree there's no association for diffuse large B-cell?

A. Well, again, it's that bell-shaped curve. And, you know, there's a possible association, but it's not within the 95 percent confidence interval.

Q. Would you agree that a hazard ratio of .89 shows no association?

A. Statistically, it shows no association.

Q. You mentioned a minute ago that you applied the civilian standard for Mr. Howard?

A. Yeah.

Q. Why?

A. Well, that was another exposure, another -- an exposure that supported the relationship between exposures and development of lymphoma.

Q. Mr. Howard was not a civilian, correct?

A. Correct.

Q. And so you did not cite the part of this study that does not support your opinion, but you cited a part of this about civilians?

A. I did.

Q. Could you turn to Table 5?

Do you see that this says (as read):

Cancer outcomes by duration

stationed at Camp Lejeune compared with Camp Pendleton between 1975 and 1985, Marine/Navy personnel subgroup?

A. Yes.

Q. Okay. Do you see the line for non-Hodgkin lymphoma?

A. Yes.

Q. Okay. Do you see for low duration, there is a hazard ratio of 1.02?

A. Right.

Q. And for medium duration, there's a hazard ratio of 1.01?

A. Yes.

Q. And for high duration, there's a hazard ratio of 1.0?

A. Right.

Q. So the hazard ratio is actually going down as the duration at Camp Lejeune goes up; is that fair?

A. Well, but the confidence intervals are almost the same for all of them.

So I think it's difficult to say that, you know, it's truly going down.

Q. Does this data suggest a no dose response

for NHL?

A. I would say yes.

Q. And then if you look at diffuse large B-cell, which is what Mr. Howard was diagnosed with, do you see there's -- for low duration at Camp Lejeune, it's .91 hazard ratio?

A. Yes.

Q. And for medium duration, it's .97 hazard ratio?

A. Right.

Q. And for high duration, it's .78 hazard ratio?

A. Right.

Q. So none of the hazard ratios for diffuse large B-cell show an association between any time at Camp Lejeune, low or high, and the development of diffuse large B-cell?

A. Well, statistically.

Q. Right. I'm asking statistically.

A. Yeah.

Q. And in fact, the association goes down the longer you're at Camp Lejeune; fair?

A. Well, again, the confidence intervals overlap so significantly it's hard to say one way or the other.

Q. So does this suggest there is no dose response?
A. Yes.
Q. And then if you look at marginal zone B-cell, do you see that row?
A. Yes.
(Reporter clarification.)
BY MS. HORAN:
Q. Sure.
For marginal zone B-cell, the low duration at Camp Lejeune is 1.39, correct?
A. Yes.
Q. And the medium duration at Camp Lejeune is 1.64?
A. Yes.
Q. And the high duration at Camp Lejeune is 1.57, correct?
A. Yes.
Q. Does that also suggest no dose response for the development of marginal zone B-cell dependent on the duration of time spent at Camp Lejeune?
A. Yes.
Q. So there's no dose response, according to this study, for NHL or diffuse large B-cell or

marginal zone B-cell lymphoma; fair?

A. Well, it -- it doesn't say that there isn't a threshold. And -- but beyond a threshold, these data do not support there being a dose response.

Q. And do you know if there is a threshold that's been identified for exposure to water at Camp Lejeune and the development of NHL?

A. No.

Q. And you're not offering an opinion on one, correct?

A. No.

Q. Turning back to your report, which is Exhibit 1 for Mr. Davis.

MR. MCGOWAN: Are we at a stopping point, it's 1:17?

MS. HORAN: Sure.

THE VIDEOGRAPHER: The time is 1:17 p.m. Pacific Time. We're going off the record.

(Whereupon, a lunch recess was taken from 1:17 p.m. to 1:49 p.m.)

AFTERNOON SESSION

THE VIDEOGRAPHER: The time is 1:49 p.m. Pacific Time. We're back on the record.

BY MS. HORAN:

Q. Dr. Hoppe, could you look back at Exhibit 1 and turn to page 12? And that's Mr. Davis' report.

A. Okay.

Q. The last paragraph has the four factors of rarity of the disease, low grade immunosuppression, your consideration and lack of any other risk factors for developing NHL, and the increased risk of marginal zone lymphoma from the ATSDR report.

Are there any other factors that you considered in drawing your conclusion that Mr. Davis' exposure to TCE and Benzene increased the risk of and was more likely than not a significant and substantial contributing factor for his NHL?

A. Well, I would say the studies that were discussed in the general causation.

Q. In addition, anything else other than those?

A. No.

Q. You did not conclude that PCE was the cause or a substantial contributing factor of

Mr. Davis' NHL; fair?

A. Correct.

Q. How did you rule out PCE as the cause?

A. Well, I -- I don't think I addressed it. I don't really rule it out. I just didn't address it.

Q. You say that the -- or is it fair to say that you -- it's your opinion that his exposure to TCE and Benzene increased his risk of B-cell lymphoma, the marginal zone lymphoma?

A. Yes.

Q. You didn't quantify the increased risk?

A. Sorry?

Q. You didn't quantify how much that increased risk is, correct?

A. No, I didn't.

Q. The last sentence says (as read):

> Furthermore, I conclude to a reasonable degree of scientific and medical certainty that Mr. Davis' exposure to TCE and Benzene, while stationed at Lejeune, was an independent cause of his lymphoma held to a more likely than not standard.

Do you see that sentence?

A. Yes.

Q. How did you decide to use more likely than not as your standard?

A. I thought the evidence was very substantial that that was the case.

Q. Is more likely than not the type of standard you would typically use with your patients if they asked you about what the cause of their NHL was?

A. It's not the typical terminology I would use.

Q. What is the typical terminology that you would use?

A. Well, I would say, for example, for a patient who has marginal zone B-cell lymphoma on the stomach with a -- in the setting of Helicobacter pylori infection that their lymphoma was likely caused by the Helicobacter pylori infection.

Q. So is it -- is that a stronger association than what you're offering for Mr. Davis and his association to Camp Lejeune water?

A. No, I wouldn't say. I mean, that's, you know, the clinical medical terminology that's used. And this is, you know, epidemiological legal

terminology.

Q. And if you translated that into medical terminology, what medical terminology would you use?

A. Likely.

Q. I want to turn to your section of your report that talks about prognosis, which is on page 9 of your report.

A. Okay.

Q. In the middle of the page, do you see it says (as read):

> Although Mr. Davis was without evidence of lymphoma at the time of his last followup, marginal zone lymphoma has a long, natural history. Two-thirds of patients will ultimately relapse and only half of those relapses develop within the first five years of followup.
>
> And you cite to Thieblemont 2000.
>
> Do you see that?

A. Yes.

Q. Did you rely on any other studies in determining the rate of relapse?

A. No. I -- you know, that was my general

knowledge, but I wanted to find some data that, you know, were -- that I could cite.

Q. You don't determine Mr. Davis' specific chance of relapse, but rather the population rate of relapse?

A. That's correct. Mr. Davis is part of the population.

Q. Do you have any reason to believe that Mr. Davis specifically will relapse?

A. Only within these limits.

Q. And if he does relapse, you don't know what course of treatment would be used, correct?

A. No. It depends on what the nature of his relapse was.

Q. So sitting here today, though, you can't say, if he relapses, X, Y and Z will be his treatment?

A. Correct.

Q. You say there's an 8 percent possibility that his disease could transform into a more aggressive large cell lymphoma, and you cite Thieblemont 2000 again.

A. Yes.

Q. Do you see that?

A. Yes.

Q. Does that mean there's a 92 percent chance that his disease would not transform into a more aggressive large cell lymphoma?

A. Correct.

MS. HORAN: I'm marking as Hoppe Exhibit 10. This is a study titled "Mucosa-associated lymphoid tissue lymphoma is a disseminated disease in one-third of 150 patients analyzed," and Thieblemont is the lead author.

(Whereupon, Deposition Exhibit 10 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, is this the study you relied on?

A. Yes.

Q. Could you turn to Figure 1, which is page 805?

A. Correct. Okay.

Q. Do you see Figure 1 says, as its description (as read):

> Overall survival of 158 MALT lymphoma patients, according to the stage of the disease, predicted overall survival of patients with localized and disseminated disease

was similar. 86 percent at five years and 80 percent at ten years with a median follow-up time of four years.

Did I read that correctly?

A. You read that correctly.

Q. Do you agree?

A. I agree that you read it correctly, but I do not agree that that text applies to that figure.

Q. Why not?

A. Well, because the figures are, obviously, transposed.

Q. Well, do you agree with the accuracy of the statement that I just read?

A. Yes.

Q. You just think it doesn't properly describe the figure?

A. Correct.

Q. But substantively, it's correct?

A. Yes.

Q. You can set that aside.

Okay. We're going to turn to Mr. Howard, so we can set Mr. Davis' report aside as well.

MS. HORAN: So I'm asking Dr. Hoppe to pull up Exhibit 3, which is Mr. Howard's expert

report.
BY MS. HORAN:
Q. Dr. Hoppe, have you communicated with Mr. Howard, his family or any physicians for him?
A. No, I have not.
Q. Could you turn to page 10 of his report?
According to your chart, Mr. Howard was only exposed to contaminated water at Hadnot Point; fair?
A. Yes.
Q. And underneath the chart, you have a number of -- you have a list of descriptions of Mr. Howard's exposure from his deposition transcript; is that fair?
A. That's correct.
Q. And you did not do any assessment personally to determine whether the water he was exposed to, for example, when he was drinking out of water buffaloes, was contaminated or not; fair?
A. That's correct.
Q. And you talk about him showering, laundry, swimming and mopping; is that fair?
A. Yes.
Q. And you did not attempt to quantify those exposures; fair?

A. Correct.
Q. If you turn to page 6.
A. I'm sorry?
Q. Sorry. Page 6.
At the bottom of page 6, you have Mr. Howard's exposure as attributed to Dr. Reynolds'; fair?
A. Yes.
Q. And those are absolute numbers is his total exposure, correct?
A. Yes.
Q. You have those listed as PPB?
A. Hmm. Yeah. Yeah.
Q. Do you see that?
A. Yes.
Q. Okay. And did you rely on Dr. Reynolds for Mr. Howard's exposure analysis or did you do any of your own exposure?
A. I relied on Dr. Reynolds.
Q. Okay. If you could pull back up Dr. Reynolds' report, which is Exhibit --
A. 7.
Q. 7?
And turn to Appendix 10, which is for Allan W. Howard. And I would say it's about -- it's

closer to the first third.

A. Okay.

Q. I can give you a sticky note if you want to mark that. Okay.

Comparing the numbers you attribute to Dr. Reynolds in your report on page 6 with the numbers Dr. Reynolds has in her Appendix 10 for Allan Wayne Howard, you cite to the range between chart 3 and chart 4; is that fair?

A. Yes.

Q. And those are in micrograms, correct?

A. Correct.

Q. Did you -- so you're determining that one part per billion is the same as one microgram?

A. No. I suspect that was just an oversight and that I should have used the micrograms per liter from -- from this table.

Q. Is the table micrograms or micrograms per liter?

A. Well, it's -- it's micrograms.

Q. Okay.

A. The table -- the table has both, has the micrograms per liter and then the total.

Q. Ah, so you're pointing to the second column, which is cumulative micrograms per liter

with the big M?
A. Yes.
Q. And then columns 3, 4, 5 and 6, which are associated with charts --
A. Chart 3 and chart 4, yeah.
Q. And chart 1 and chart 2?
A. Yeah.
Q. Those are all just micrograms, right?
A. Yeah.
Q. And in your report, you cite to the numbers associated with just the microgram?
A. Correct.
Q. Why offer the range between chart 3 and chart 4 as your assessment of Mr. Davis' exposure?
A. Well, I guess there was variable interpretations of exposure reflected in charts 3 and 4.
Q. How did you determine to rely on 3 and 4 as opposed to 2, chart 2?
A. So I -- I don't recall.
Q. You do not include in your report the contaminant levels for chart 1 or chart 2; is that fair?
A. That's fair.
Q. And you didn't consider them in forming

your opinion?
A. Correct.
Q. You might want to leave that open to Mr. Howard's appendix, and if you could turn back to your report, which is Exhibit 3, and turn to page 9.
A. Okay.
Q. Do you see it says (as read):

> During Mr. Howard's 449 days of exposure at Camp Lejeune, he would have likely been exposed to 5,937 parts per billion/liters-M of TCE, 251 parts per billion per liter-M of PCE, and 343 parts per billion/liter per M of vinyl chloride and 70 parts per billion/liters-M of Benzene.

Do you see that?
A. Yes.
Q. Again, Dr. Reynolds' opinions are micrograms per liter-M, not the parts per billion, correct?
A. I assume that that's also the case, from the appropriate table.
Q. You can -- if you want to look at the -- if it's Dr. Reynolds' report, Appendix 10, I believe

you used the numbers --
A. Yeah.
Q. -- from the cumulative column --
A. Right.
Q. -- correct?
A. Right. It's micrograms.
Q. And you offered this number in Mr. Howard's exposure assessment. You offer the cumulative micrograms per liter month.
What is it -- why offer it for Mr. Howard's analysis?
A. Well, it's part of the expert report.
Q. How did it play into your analysis of his exposure as it relates to your opinion?
A. Well, that would indicate a high exposure.
Q. As compared to what?
A. As compared to what one is normally exposed to outside of Camp Lejeune.
Q. Is it your opinion that the numbers on page 9 of your report, for example, the 5,937 micrograms per liter-M means that -- what does that number mean as applied to your exposure analysis?
A. Well, it's high.
Q. So -- but the parts per billion liter M,

is that unit -- what do you understand that unit to mean?

A. Well, that's the concentration of the agent in water.

Q. Is it his total concentration if you add up all the months, is it the average over every month that he was there?

Do you know what that number means?

A. Well, that -- that's -- that would be an average per month.

Q. So it's your opinion that -- or your understanding of Dr. Reynolds' opinion is that Mr. Howard was exposed to 5,937 parts per billion per liter month every month he was at Camp Lejeune?

A. On average.

Q. Okay. If you look on page 9 of your report.

A. Yes.

Q. Do you see the paragraph above that says, in the middle (as read):

Reconstructed TCE concentrations at Hadnot Point drinking water reached a maximum level of 546 micrograms per liter during December of 1978?

A. Yes, I see that.
Q. Okay. So the maximum level he was exposed to was 546 micrograms per liter per -- assuming the average is a range?
A. Okay. So that level was reached during December 1978, yeah.
Q. So turning back to the next paragraph, could you turn to Dr. Reynolds' report again, and to Appendix 10? And then do you see -- I apologize. I don't know why this happened. If you turn to the page after the summary charts. I apologize, it looks like some of the headings got cut off in the printing.
But do you see that that first chart has the 5,937, 251, 343 and 70?
A. Yes.
Q. Do you know or have you done any analysis to determine if those numbers are the addition of all the numbers above them?
A. Well, as I look at this table, I assume that they are sums of those numbers above.
Q. Okay. So if 5,937 is the sum of the TCE values per month for the time that Mr. Howard was at Camp Lejeune, does that make sense to you?
A. Well, I think so, but it would be nice to

see the headings of these columns.

Q. Yeah. I don't know why they got cut off when we printed. I apologize for that.

A. So I don't know if these are measurements on different days or -- I simply don't know.

Q. Okay. If I represent to you that these are the concentrations of the addition of the months that Mr. Howard was there, how does the -- if you add up the month that Mr. Howard was there and the exposure that was estimated for each month, how does that cumulative number -- what does that tell you for your specific causation analysis?

A. Well, that -- that value of 5,937 is a very high value. And so, you know, I would consider it very highly as a causation.

Q. Does the cumulative exposure column, if you turn back to the page before it, suggest that Mr. -- that Dr. Reynolds took into account Mr. Howard's use of the water in any way when she determined the cumulative column?

A. Well, I can't speak to what she did. But I assume that that was the cumulative exposure.

Q. That Mr. Davis -- strike that.

That Mr. Howard was exposed to, including his unique interactions with the water?

A. Yes, if I understand you correctly.
Q. Okay. So if someone were living in a place where there were 20 micrograms per liter of TCE in the water, would it be important to know how they interacted with the water in order to determine their exposure?
A. Sure.
Q. And would you agree that just knowing that there were 20 micrograms per liter of TCE or any other chemical in the water would not allow you to, in and of itself, determine the risk of any particular individual without also assessing how they uniquely consumed or interacted with the water; fair?
A. Fair.
Q. You included in your report for Mr. Howard the units of parts per billion.
And I think you -- you agree that Dr. Reynolds used micrograms?
A. Correct.
Q. Okay. It wasn't intentional on your part and you didn't do any analysis to convert micrograms --
A. No.
Q. -- to parts per billion?

A. No.
Q. Do you know if the published literature looking at exposure uses micrograms or parts per billion?
A. I think some looks at part per billion and some looks at micrograms.
Q. Do you know if the literature uses parts per billion per liter a month as a unit to assess or micrograms per liter month?
A. Yeah, I can't say that I've seen that per month utilized in papers I reviewed.
Q. If you turn back to page 9 --
A. Right.
Q. -- you state that the -- I think you said earlier that -- when I was asking you about how these numbers played into your analysis, I believe you testified they were high?
A. Right.
Q. What analysis are you doing or -- to determine that they are high?
A. Well, I look at the maximum contaminant levels that have been established, I think, by the EPA for these agents, and I think they're all, you know, at the five micrograms per liter level.
Q. Anything else?

A. That's the main thing.
Q. Turning to page 11 of your report, you, at the top, provide again Dr. Reynolds' numbers.
And those are the same numbers that you provided on page 6, correct?
A. Okay. Right.
Q. And you say (as read):
> In my opinion, the level of exposure to TCE, PCE and Benzene that Mr. Howard experienced during his 449 days living and working on Camp Lejeune was more than sufficient to cause his NHL and was substantial.

Did I read that correctly?
A. Correct.
Q. What -- how did you determine the level of exposure was sufficient?
A. Well, basing it on the expert report from Dr. Reynolds, and also the literature that I cited and referred to.
Q. And we -- I believe you testified previously that you hadn't determined a threshold amount of TCE, PCE or Benzene.
And that's also true for Mr. Howard; you

don't have that opinion in his report either, correct?

A. Correct.

Q. As to exposure, other than the numbers of absolute exposure and the micrograms per liter/month that you have from Dr. Reynolds and the number of days that Mr. Howard was living at Camp Lejeune, are there any other factors of exposure you considered in forming your opinion?

A. No.

Q. If you turn back to page 9, right under the parts per billion per liter month sentence, you have references to a study from 19- -- from the 1990s in New Jersey.

A. Right.

Q. I believe it's the next two paragraphs.

A. Yes.

Q. Why did you include that study?

A. Well, I thought it was perhaps the most relevant because, you know, it related to groundwater contamination and it was a large study. They -- so I felt that their conclusions were valid.

Q. Do you know if this study is a cohort study or an ecological study?

A. Let's see. This one, this was not a

cohort study, so I would assume that it's an ecological study, if it's one or the other.

MS. HORAN: I'm marking as Hoppe Exhibit 11.

(Whereupon, Deposition Exhibit 11 was marked for identification.)

BY MS. HORAN:

Q. This says (as read):

Drinking Water Contamination and the Incidence of Leukemia and Non-Hodgkin's Lymphoma.

And this is the Cohn study.

I apologize, there's some random highlighting on this. I don't know where that came from. But I'm not going to ask you about it, and I'd just ask that you please ignore it.

MS. HORAN: Oh, maybe yours doesn't have it.

Is there no highlighting on your copy?

MR. MCGOWAN: No.

MS. HORAN: Oh, great. I take that back, then.

BY MS. HORAN:

Q. Dr. Hoppe, having had a chance to review this study, do you have any opinion on what type of

epidemiological study this is?

A. Well, it doesn't specify that it's a cohort study.

Q. And I think you testified earlier, you're not particularly familiar with the term "ecological study"?

A. Correct.

Q. Okay. Do you agree that this study, the Cohn study, did not consider duration or extent of exposure?

A. I believe that's the case.

Q. Okay. You can set that aside.

Dr. Hoppe, do you treat kidney cancer?

A. No, I don't.

Q. And you're not offering any opinions in this case on the cause, treatment and/or prognosis of Mr. Howard's kidney cancer?

A. That's correct.

Q. Turning to your medical history of Mr. Howard, prior to June of 2023, when Mr. Howard was brought into the ER, there were no signs of lymphadenopathy, correct?

A. Umm, no.

Q. That's incorrect?

A. That's -- that's incorrect.

Q. Okay. Why is that incorrect?
A. Well, I -- included in my summary here, I noted that on November 30th, 2016, an MRI at OSU Wexner Medical Center noted that there are small to prominent multiple noticeable mesenteric lymph nodes in the central abdomen, which are nonspecific, confluent mesenteric node in the left abdomen, measures 1.3 by 2.5 cm in size. No definite retroperitoneal adenopathy, no discreet enlarged pelvic or inguinal nodes. Essentially stable since the prior comparison CT dated 2014.
Q. And then on April 18th, 2019, a computed tomographic scan of the thorax revealed no mediastinal or auxiliary --
(Reporter clarification.)
MS. HORAN: Sure. Mediastinal.
THE WITNESS: Mediastinal.
BY MS. HORAN:
Q. Mediastinal or auxiliary lymphadenopathy.
Dr. Hoppe, how do you say that word?
A. Lymphadenopathy.
Q. Lymphadenopathy.
Okay. So after 2016, didn't the next records suggest there was, again, no lymphadenopathy?

A. Well, that was an examination of his chest only, whereas the previous exam that I noted was a scan that included his abdomen, and it was in his abdomen that these lymph nodes were reported to be present.

Q. Okay. And then in August of 2020, the second sentence says (as read):

> Imaging was performed at that time, including a CT of the chest, abdomen and pelvis, no lymphadenopathy was reported.

Did I read that correctly?

A. Yes, you did.

Q. So in August of 2020, when his abdomen underwent a CT, there was no lymphadenopathy?

A. According to the report, yes.

Q. Do you have reason to doubt the report?

A. I didn't look at the images myself so I have to go by the report.

Q. Did you look at the images of the 2016 --

A. No, I did not.

Q. So there were -- in 2016 -- strike that.

In November of 2016, you think there were signs of the lymphadenopathy, but then they -- when they tested again in August of 2020, there were

none?

A. According to the reports, yes.

Q. Is that common or what does that tell you?

A. Well, there are two possibilities, or at least two possibilities, you know. One is that the lymph nodes that were identified in 2016 were potentially infection-related or some other inflammatory stimulus that was not present in 2020, right. Is that what it -- yeah.

That's one possibility. Another possibility is that he could have had lymphoma at that time that -- with some regression that can happen spontaneously, that became more evident at a later date.

A third possibility is that given the circumstances in August 2020 that this patient was scanned following a bicycle accident and traumatic injuries, there's a tendency for radiologists to focus on the acute problem and not to pay too much attention to some enlarged nodes that might be present, especially if they're only marginally enlarged. That's why I said I have to rely on the report since I didn't see the images myself.

Q. Do you have any reason to believe that the enlarged nodes that were seen in November of 2016

are related to Mr. Howard's subsequent development of non-Hodgkin's lymphoma?

A. I can't be absolutely certain. I would say there -- given that they were not seen subsequently per reports, more likely they were not related to lymphoma.

Q. And in 2016, they were not tested -- or no tests were done to determine if they were non-Hodgkin's lymphoma?

A. Correct.

Q. And to the best of your knowledge, there was no investigation by the physicians at that time, or consideration of them, that the enlarged nodes were non-Hodgkin's lymphoma; fair?

A. Correct.

Q. And then in June of 2023, which begins kind of towards the end of the page --

A. Yeah.

Q. -- Mr. Howard was brought into the ER; fair?

A. Right.

Q. And the last sentence reads (as read):

> He biked 30 to 40 miles a day, but was complaining of some fatigue and body aches.

Correct?

A. Correct.

Q. And then in October of 2023, Mr. Howard was diagnosed with diffuse large B-cell lymphoma; fair?

A. Right.

Q. And he began chemotherapy on October 16th of 2023?

A. Yes.

Q. Give you a minute. All set?

A. Yeah.

Q. Okay. Would you agree that diffuse large B-cell lymphoma is the most common lymphoma in the United States?

A. It's one of the two most common.

Q. What's the other common?

A. Follicular lymphoma.

Q. And you agree that Mr. Howard's treatment for his diffuse large B-cell lymphoma was appropriate?

A. Yes.

Q. And you agree that Mr. Howard tolerated chemotherapy well for his non-Hodgkin's lymphoma?

A. Yes.

MS. HORAN: I'm marking as Hoppe

Exhibit 13. These are medical records of Mr. Howard with the Bates 00490_HOWARD_KH_0000000334 and it goes through -339.

(Whereupon, Deposition Exhibit 13 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, have you seen these medical records before?

A. Yes, I believe I have.

Q. And the first page is for a date of encounter from October 24th, 2023?

A. Yes.

Q. And this is for a follow-up appointment after his first round of chemotherapy; fair?

A. Right.

Q. And it says (as read):

Still lives an active lifestyle, states he rode 50 miles on his bike the day after treatment.

Do you see that?

A. That's great. Yes.

Q. And it says (as read):

Patient is very happy to report lymphadenopathy has

significantly decreased. Appetite is good, no nausea, no issues with constipation or diarrhea, no chest pain or shortness of breath, no fever or chills.

Do you see that?

A. Yes.

Q. And you would agree that that's a patient who is tolerating chemotherapy very well; fair?

A. Yes.

Q. You can set that aside.

Following the two cycles of chemotherapy, a PET scan showed that Mr. Howard had a complete metabolic response to his treatment, correct?

A. Correct.

Q. That means there was no clear evidence for residual or recurrent disease; fair?

A. Well, it's likely that he still had disease, it just didn't show up on imaging.

Q. Oh, okay. I think I might have -- okay.

So is it fair to say that there was no clear evidence for residual or recurrent disease that was seen?

A. Or you could say more precisely, he had a complete metabolic response.

Q. Okay. And he completed his treatment for non-Hodgkin's lymphoma on February 19th of 2024?

A. Right.

Q. And as of today, there's no evidence of any recurrence of Mr. Howard's NHL; fair?

A. Right.

Q. Turning to your Section 11, which is page 11 of Mr. Howard's report, that's the title -- or the section with a title "Differential Diagnosis, Methodology to Determine the Etiology of the NHL."

Do you see that section?

A. Yes.

Q. And if you turn to the last page, page 12, you say (as read):

> Given the lack of any other risk factors for the development of NHL, e.g., immunosuppression, autoimmune disorders, HIV infection, EBV infection, hepatitis C virus infection, organ transplantation, familial history or exposure to herbicides and the increased risk for lymphoma among individuals exposed to TCE, PCE and Benzene, I conclude that it is more

likely than not that in Mr. Howard's case, the additive exposure to TCE, PCE and Benzene increased the risk of and was more -- and was likely a significant contributing cause of his diffuse large B-cell lymphoma. Did I read that correctly?

A. Yes.

Q. So the only causal explanation you identify for Mr. Howard's NHL is the water at Camp Lejeune; fair?

A. Yeah. All other risk factors were absent.

Q. And you used the same differential diagnosis methodology that you used for Mr. Davis, correct?

A. Correct.

Q. So we went through for Mr. Davis the risk factors for NHL generally and the risk factors for marginal zone lymphoma.

And for NHL generally, you mention risk factors of immunosuppression, infection with EBV, which I believe is Epstein Bar Virus, exposure to chemical agents and genetic family, familial relations.

Is the general list the same for Howard of risk factors?

A. The general risk factors, yes. But you didn't mention all of them. You didn't mention, like, transplantation.

Q. Oh, the paragraph -- the parentheses are the list of risk factors you considered for Mr. Howard?

A. Yeah.

Q. Okay. And are these general to NHL or are any of these specific to diffuse large B-cell lymphoma?

A. Well, for example, the organ transplantation, most of the secondary lymphomas among patients with organ transplantation are diffuse large B-cell, although occasionally, you will find others.

Q. Any other specific to --

A. HIV infection.

Q. Okay.

A. Immunosuppression in general.

But, you know, they -- most of the secondary lymphomas in those situations are diffuse large B-cell, but you can say other lymphomas as well.

Q. And when you say, "secondary lymphomas," do you mean they've already developed a lymphoma and this is the --

A. No.

Q. -- second one?

A. No. No. It's secondary to the risk factor.

Q. Okay. So they have already developed some type of immuno- -- or have some type of immunosuppression --

A. Right.

Q. -- and then they developed diffuse large B-cell --

A. Right.

Q. Okay.

(Reporter clarification.)

BY MS. HORAN:

Q. What studies or your experience, what did you rely on in determining what the risk factors you should consider for Mr. Howard were?

A. My general knowledge of risk factors in patients who develop lymphoma.

Q. Did you look for any studies or anything to determine or to confirm your experience?

A. No.

MS. HORAN: I'm marking as Hoppe Exhibit 14. This is a study, "Epidemiology and the etiology of diffuse large B-cell lymphoma" by Sofia S. Wang.

(Whereupon, Deposition Exhibit 14 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, have you seen this study before?

A. No.

Q. Could you turn to page 258?

Do you see Table 4, which is in the top right, says (as read):

Summary of risk associations of established risk factors for diffuse large B-cell lymphoma?

A. Right.

Q. And you would agree that that chart does not include TCE, PCE or Benzene as a risk factor; fair?

A. It does not list those.

Q. And you would agree that chart does not list exposures to any toxic chemical agent as a risk factor?

A. I agree.

Q. Is exposure to TCE, PCE and Benzene commonly considered by practitioners when assessing the cause of diffuse large B-cell lymphoma?

A. Well, I would only say that if they are faced with a patient who has those exposures, they would consider it. In my practice, exposure to those agents is, you know, very rare. And so I wouldn't have reason for most patients to ask about that specific exposure.

Q. You can set that aside.

Oh, actually, do you agree with the established risk factors for diffuse large B-cell lymphoma in Table 4, do you generally agree with that table, that those are established risk factors?

A. I agree with these.

Q. You can set that aside.

Do you agree that most cases of diffuse large B-cell lymphoma are of unknown cause?

A. Yes.

Q. Did Mr. Howard's hypothyroidism factor into your differential diagnosis at all?

A. I don't recall when his hypothyroidism developed. But I would say hypothyroidism is not a predisposing cause for lymphoma. It's not a risk factor for lymphoma, even, you know, in the list

that you provided from Dr. Wang.

Q. So you did not consider it as part of your differential diagnosis methodology then?

A. No, I would not have because it's not a risk factor.

Q. Is it possible that Mr. Howard's diffuse large B-cell lymphoma is idiopathic?

A. I would only consider it idiopathic if there was no other explanation.

MR. ELLIOTT: Go off the record.

MS. HORAN: Can we go off the record?

THE VIDEOGRAPHER: The time is 2:53 p.m. Pacific Time. We're going off the record.

(Whereupon, a recess was taken from 2:53 p.m. to 3:04 p.m.)

THE VIDEOGRAPHER: The time is 3:04 p.m. Pacific Time. We're back on the record.

BY MS. HORAN:

Q. Dr. Hoppe, you -- on page 11 of Exhibit 3, which is your report for Mr. Howard, if you could turn to page 11.

A. All right.

Q. And you have, just a little bit past the midway of the page, it says -- the paragraph starts, "For instance."

Do you see that paragraph?

A. Yes.

Q. And it includes a number of citations to Mr. Howard's deposition transcript?

A. Right.

Q. Other than his deposition transcript, did you also -- or what other documents did you review to determine what risk factors were relevant to your differential diagnosis?

A. Only what I saw in the medical record.

Q. Anything other than medical records and his deposition?

A. No.

Q. Were there any risk factors that you were not able to rule in or rule out based on the deposition and medical records?

A. No.

Q. The next -- or at the end of page, there's a paragraph that begins "In a Bove, et.al., study in 2024."

Do you see that?

A. Yes.

Q. And that is the 2024 Bove study that we looked at earlier that's been marked as Exhibit -- Exhibit 9?

A. Right.
Do you want me to --
Q. Sorry, I just want to confirm that it's the same study.
A. It is the same study, yeah.
Q. And I think you actually mentioned this already, but you cited the statistics for civilian workers in Mr. Howard's report?
A. Yes.
Q. Why?
A. Well, because they were there.
Q. And you did not cite the statistics for Marines even though they were also in the study, correct?
A. That's correct.
Q. And you didn't cite the statistics as they applied to Marines that showed that there was no association; fair?
A. No reported association, correct.
Q. You did not do a quantitative risk assessment for Mr. Howard, correct?
A. What do you mean by "quantitative risk assessment"?
Q. Did you take any effort to quantify the risk -- the increased risk of Mr. Howard developing

diffuse large B-cell lymphoma in light of his exposure to TCE, PCE and Benzene?

A. Well, I -- perhaps not quantitative, but qualitative.

Q. And what -- other than the term "increased risk," is there any more specific assessment you did to qualitatively determine his risk -- increased risk?

A. By reviewing the literature related to exposure to TCE, PCE and Benzene in the non-Hodgkin lymphomas.

Q. Did you assess what Mr. Howard's risk for developing non-Hodgkin's lymphoma or diffuse large B-cell lymphoma was regardless of his exposure to TCE, PCE and Benzene?

A. You mean his --

Q. I mean the general population --

A. His general population --

Q. Yeah.

A. -- risk?

No, I didn't.

Q. Was that not necessary for your assessment of the increased risk in light of these exposures?

A. Well, I believe that the data are what they are relative to the studies that had been

published indicating an increased risk. And those were the numbers that I gave my opinion on.

Q. If you turn to the last page, page 12, your concluding paragraph includes a conclusion that it was TCE, PCE and Benzene that made it -- that -- strike that.

You conclude that (as read):

> It's more likely than not that in Mr. Howard's case, the additive exposure to TCE, PCE and Benzene increased the risk of and was likely a significant contributing cause of his diffuse large B-cell lymphoma. Furthermore, I conclude to a reasonable degree of scientific and medical certainty that Mr. Howard's exposure to TCE, PCE and Benzene while stationed at Lejeune was an independent cause of his lymphoma.

Do you see that?

A. Yes.

Q. So you -- for Mr. Howard, you opined that TCE, PCE and Benzene were all a significant contributing -- were all the cause of his

non-Hodgkin's lymphoma; fair?

A. Yes.

Q. And if you look back to Exhibit 1 for Mr. Davis, your concluding paragraph for Mr. Davis includes only PCE and Benzene.

Is there a reason why there's a discrepancy between those two?

A. No specific reason. I just didn't address the PCE issue in as much detail in the Davis case versus the Howard case.

Q. Turning to his prognosis, on page 8, you say that Mr. Howard's likelihood of relapse is 10 percent.

What did you rely on for that figure?

A. Those are data that have been published related to the International Prognostic Index and its value in predicting outcome for patients treated with contemporary therapy for diffuse large B-cell lymphoma.

Q. And you also offer two late effects from treatment he received, including a cardiac effect from doxorubican [sic] and impaired immune health from rituximab?

A. Rituximab.

Q. Rituximab.

Is that fair?

A. Yes. Potential.

Q. Potential?

A. Potential late effects.

Q. So Mr. Howard has not experienced any cardiac effects from doxorubican [sic]?

A. Not that I'm aware of.

Q. And you're not aware that Mr. Howard has experienced any impaired immune health from Rituximab?

A. Not that I'm aware of.

Q. It's speculative if any of these late effects will impact Mr. Howard; fair?

A. Well, there are established risks. I wouldn't call it speculative. It's unknown.

Q. It's unknown.

Turning first to the cardiac effects, you agree that it's a known risk that doctors control for by limiting the cumulative dose of doxorubican [sic]?

A. Yes.

Q. And Mr. Howard's physician limited his cumulative dose of doxorubican [sic]?

A. Yes, he did because beyond a certain point, the -- the risk of those late effects

increases substantially, but the risk is there even with the conventional doses of chemotherapy, including doxorubicin.

(Reporter clarification.)

THE WITNESS: Cin, C-I-N.

MS. HORAN: I'm marking as Hoppe Exhibit 15. This is Zduniak article from 2022 titled "Cardiovascular outcomes of patients treated for non-Hodgkin lymphoma with first line doxorubicin-based chemotherapy."

(Whereupon, Deposition Exhibit 15 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, this is a study you cite to in your report, correct?

A. Yes.

Q. And you cite to it for the proposition that the cumulative incidence of cardiovascular events at five years among patients treated with standard chemotherapy for diffuse large B-cell lymphoma is 11.4 percent, correct?

A. Correct.

Q. And that 11.4 percent includes the first six months, one year and over one year, correct?

A. Correct.

Q. And Howard is now past one year since his treatment?
A. I believe so. Yes.
Q. And the study only found that 31 percent of cardiac events were after the first year?
A. Are you saying 31 percent of patients or ...
Q. Yes.
A. 'Cause I'm looking at the curves in Figure 2, and it looks like the majority of events occur beyond 12 months.
Q. So if you look at Table 2.
A. Okay.
Q. Actually, strike that.
You see the -- across the top, it says (as read):
Early onset CV event N equals 23.
A. Correct.
Q. Is that your understanding that there were 23 events that were early onset --
A. Right.
Q. -- CV?
A. Right.
Q. And there were 12 that were subacute CV

events?
A. Right.
Q. And 16 that were late CV events?
A. Right.
Q. And so of the population, roughly 31 percent were late events?
A. Okay.
Q. So the risk to Mr. Howard would be that 11.4 percent multiplied by that 31 percent; fair?
A. Well, the one thing I'm looking for here is what the -- the median followup was of these patients. Because the true risk of the late events would depend on the duration of followup.
So, yeah, they say here the median followup was five years, which is not very late in terms of potential toxicity for the agents that we use for cancer treatment.
Q. And what's your opinion that that's not very late based off of?
A. My general experience and knowledge that late effects can occur many years after -- after primary treatment.
Q. And that's based on your practice as a treating physician?
A. Yes.

Q. Do you have any studies or anything that has looked beyond five years?
A. I would have to -- I'd have to search on that. I'm sure there is -- there are some, but off the top of my head, I can't quote them.
Q. Turning back to your report.
A. Yes.
Q. So back to Exhibit 3, which is Mr. Howard's report, you state that Rituximab is a known immunosuppressive therapy, it may result in long-term impaired immune health, correct?
A. Yes.
Q. And then you cite to a study Shree 2020?
A. Right.
MS. HORAN: I'm marking as Exhibit -- Hoppe Exhibit 16. This is "Impaired immune health in survivors of diffuse large B-cell lymphoma," by Shree.
(Whereupon, Deposition Exhibit 16 was marked for identification.)
BY MS. HORAN:
Q. Dr. Hoppe, is this the study you cite to in your report?
A. Yes.
Q. And you cite to the study for the

proposition that Rituximab may impact immune health?
A. Yes.
Q. On the first page, do you see the section that says "results"?
A. Yes.
Q. The last sentence reads (as read):
The elevated risks could not be explained by exposure to chemotherapy, stem cell transplantation or Rituximab except for IRRs for humoral deficiency, which were consistently higher after the incorporation of Rituximab into DLBCL treatments.
Do you see that sentence?
A. Yes.
Q. Would you agree that the study concluded that elevated risks of various immune-related conditions could not be explained by exposure to Rituximab?
A. Well, it's a little confusing, this sentence, taking it out of context, because it says (as read):
Except for IRRs for humoral deficiency, which were consistently

higher after the incorporation of Rituximab into DLBCL treatments.

So it doesn't absolve Rituximab completely of accounting for these side effects.

Q. But it also doesn't attribute them definitively to Rituximab; fair?

A. Can you repeat that question?

Q. Sure.

The study is not definitively attributing the impacts to Rituximab; fair?

A. Not to Rituximab directly.

Q. I want to turn next, Dr. Hoppe, unless you have something else on that exhibit.

A. No.

Q. I want to turn next to what we've been referring to as your general causation opinions.

And these are the same as my understanding from you today for both Mr. Howard and Mr. Davis.

So do you have a preference on which report we use --

A. No.

Q. -- to go through them?

Okay. Turning to page -- we'll use your Davis report, then, which is Exhibit 1. And this might be easier just 'cause the citations are in the

text.

On page 6 of your report, you have a couple of sentences on the standards for evidence for causation for TCE, PCE and Benzene.

Do you see that at the top of the page?

A. The very -- very first paragraph on page 6?

Q. Yes.

A. Yes.

Q. And you're equating equipoise and above evidence with the standard at least as likely or not -- as not; fair?

A. Yes.

Q. The top of page 6, the first paragraph, the standard sufficient evidence equipoise and above and sufficient evidence that you use for TCE, PCE and Benzene, those studies came from the 2017 ATSDR study?

A. Correct.

Q. Do you see those standards regularly in your practice?

A. Well, as I -- I think I said earlier, I don't use this terminology in my practice.

Q. And you said earlier, I believe, that you've never read the Camp Lejeune Justice Act?

A. You know, I -- I don't think so. If I did, it was very early in the materials that were -- were given to me.

Q. But you're not attempting to interpret legal language or legal causation standards in your report; fair?

A. I don't think so.

Q. And you didn't feel it was necessary to review statutory language in order to offer your opinions in this case?

A. No.

Q. Other than the ATS- -- 2017 ATSDR report, have you seen any published guidance on how one applies the equipoise and above or as likely as not standard?

A. I -- I think I've seen it referred to in some other publications, but I can't -- I wouldn't be able to pinpoint where.

Q. Turning to page 3, so the first part of this page is about TCE, correct?

A. Yes.

Q. And you opine that TCE can cause immune system dysfunction?

A. Yes.

Q. And the two -- or the human studies you

cite is the two Bassig studies on the Chinese factory worker, the Bassig 2013 and 2016?

A. Yeah. And also the -- the Lash study.

MS. HORAN: Marking as Hoppe Exhibit 17. This is "Occupational Exposure to Trichloroethylene and Serum Concentrations of IL-6, IL-10 and the TNF Alpha." The author is Bassig, and it was published in 2013.

(Whereupon, Deposition Exhibit 17 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, this is the Bassig 2013 study that you cite to?

A. Yes.

Q. Could you turn to page 6, please? The last two sentences, which are at the top of page 6, read (as read):

> Given that immunologic alterations are suspected to play a role in lymphomagenesis, and IL-10 plays an important role in immunologic processes, our findings provide additional evidence that TCE is immunotoxic in humans and some support for the biologic

plausibility that TCE may be associated with NHL. However, our findings require replication in larger studies and in other exposed populations.

Do you see that?

A. Yes.

Q. You agree the study did not find that TCE causes NHL?

A. No. It demonstrated effects on the immune system that would potentially be causative for NHL.

Q. Sure.

So it did not find a causative relationship between TCE and NHL; fair?

A. No. But I'm not sure that was what they were trying to do. They were trying to demonstrate that exposure to this agent affected immune markers.

Q. And so it offers just some support for the biologic plausibility that TCA may -- TCE may be associated with NHL?

A. Yes.

Q. And that's the limit of their findings; fair?

A. Yeah.

MS. HORAN: I'm marking as Hoppe

Exhibit 18. This is a "Comparison of hematological alterations and markers of B-cell activation in workers exposed to Benzene, formaldehyde and trichloroethylene." And this is a Bassig study from 2016.

(Whereupon, Deposition Exhibit 18 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, you also cite to this study in your report; fair?

A. Yes.

Q. In this study, were the immune markers of the exposed workers within normal range?

A. What do you include under "immune markers"?

Q. What would you include as an immune marker for this study?

A. Well, I would include, for example, chromosome loss, which is demonstrated to be affected by Benzene exposure.

Q. What page are you looking at?

A. That is page 697.

Q. You're looking at the charts?

A. Right. Or in Figure 2, certain subsets of lymphocytes being significantly affected by exposure

to TCE.

Q. Anything else?

A. Well, those are -- those are examples, yeah. So, you know, I would -- I think your statement was that there was no -- no association --

Q. Oh, no, I asked if the immune markers of the exposed workers were within normal range.

A. I'm not certain if they were within normal range, but they were significantly decreased.

Q. And would you agree that changes in immune markers in the normal range would not be clinically significant, even if there were changes?

A. No. I wouldn't agree that would not be clinically significant.

Q. So you would -- it's your opinion that if there's a change in the immune marker, but it remains in the normal range, that's clinically significant?

A. Yes.

Q. Do you know if these workers were followed to determine if NHL developed?

A. If --

Q. NHL --

A. Well, again, I think that this study was not intended to look at the absolute linkage to NHL.

Q. You can put that aside.

The -- the two studies we just looked at, which were marked as Exhibit 16 and 17 -- 17 and 18, those studies involved comparison for the measured outcomes between TCE exposed and controlled or unexposed factory workers, correct?

A. Correct.

Q. So you -- in order for these studies to be relevant to Camp Lejeune, you have to adopt the assumption that the exposures to TCE for the workers in these studies was sufficiently similar for TCE exposure for Camp Lejeune subjects; fair?

A. Well, I think this relates more to mechanisms of -- that might predispose one to the development of lymphoma. You know, and, you know, that's the entire reason for including them.

Q. Ah.

So you included the Bassig 2013 and 2016 studies in order to, in your review of the mechanisms, the biologic mechanisms under which TCE can impact the immune system?

A. Right.

Q. Do you know if the Chinese factory workers in the studies that you cited to were exposed to TCE via contaminated water?

A. I don't know that they weren't.
Q. Do you know what the source of their TCE exposure was?
A. So it was occupational exposure. Exactly what the occupations were, I'm uncertain. They were factory workers.
Q. So because you limit the utility of these studies to biologic mechanisms for the interaction between TCE and the immune system, does the frequency, intensity and duration of TCE exposure, does it matter if it's the same between the Chinese factory workers and the Camp Lejeune individuals for your -- for the purposes of your analysis?
A. I would say no. It's simply an explanation of the mechanisms, potential mechanisms related to development of lymphoma.
Q. Is it your opinion that subjects with lower but normal lymphoid subpopulation counts are immunosuppressed relative to subjects with higher but normal numbers of lymphoid cell populations?
A. I would say that for certain subpopulations of lymphocytes, if you look at the total population of lymphocytes, it would be hard to say that in the lower part of the normal range there's any greater risk than in the higher part of

the normal range.

Q. The immune markers looked at the studies -- looked at in the studies, are those immune markers considered to be validated, reliable and relevant markers of cancer risk?

A. Yes.

Q. And why is that?

A. Well, because effects on lymphocyte subpopulations can affect what's referred to as immune surveillance, where certain subsets of lymphocytes recognize foreign antigens, foreign proteins or -- that may be products of -- of neoplasm or cancer. And if subpopulations of lymphocytes are reduced, then there may be a decreased ability for immune surveillance.

Q. In your report on page 3 -- excuse me -- after the Bassig studies, you cite to, in the following paragraph, the Karami 2013 study and the Scott Jinot 2011 study?

A. Right.

Q. Excuse me.

Are there any other meta-analysis you looked at related to TCE other than Karami and Scott and Jinot?

A. Those are the two main ones that I recall.

MS. HORAN: I'm marking as Hoppe Exhibit 19. This is the "Occupational trichloroethylene exposure and risk of lymphatic and hematopoietic cancers: a meta-analysis." This is Karami 2013.

(Whereupon, Deposition Exhibit 19 was marked for identification.)

THE WITNESS: Oh, sorry.

BY MS. HORAN:

Q. Dr. Hoppe, this is the study you relied on, correct?

A. Yes.

Q. Did you evaluate the underlying studies?

A. The -- each individual study included in the analysis --

Q. Yes.

A. -- no.

Q. Did you look at any of them?

A. Yes. Well, I can say I recall looking at the Cocco study.

I'm not sure about any others.

Q. Why didn't you include the Cocco study in your report?

A. Well, I didn't look upon this as an extensive general causation analysis. So, you know,

I -- the Karami study and the Scott Jinot study were meta-analyses, and so they included studies like the Cocco study in their -- in drawing their conclusions.

Q. When you say you didn't view this as an extensive general causation analyses, what did you view your general causation analyses to be?

A. Well, I knew that there were general causation experts, and I was a specific causation expert. That was my understanding, at least, to -- to include some general considerations, which I did. But not to be -- not to extensively review the literature.

Q. Why didn't you submit your opinions -- your general causation opinions at the same time as the other general causation experts?

Do you know why?

A. I was told I had a certain deadline, you know, for preparing my report.

I had no idea when the general causation deadlines were.

Q. Fair enough.

If you look at Karami, which is Exhibit 19, do you see the abstract, the second sentence reads (as read):

We conducted a meta-analysis of published cohort and case controlled studies exploring occupational TCE exposure in relation to five different lymphatic and hematopoietic cancers.

Do you see that?

A. Yes.

Q. And that's your understanding of what Karami did?

A. Yes.

Q. And all the studies and the analyses were classified as those that assessed either occupational TCE exposure or mixture of chlorinated solvent exposure studies; fair?

A. Yes.

Q. And if you turn to page 2, do you see the section that says "Methods"?

A. Yes.

Q. The very end of that page in the first column, it says (as read):

As a result, we excluded community-based studies of TCE exposure from drinking water since

the level and route of exposure differ from those found in occupational settings. Did I read that correctly?

A. Yes.

Q. Do you agree with the authors' decision to exclude drinking water studies?

A. Well, they -- that was their choice. They wanted to have one less variable in their analysis. So whether I agree or disagree with them, this -- that was their decision.

Q. Does their decision to exclude drinking water studies impact the applicability of this study to your findings related to drinking water at Camp Lejeune?

A. No. I -- you know, because as they explained the level and route of exposure was different, and they just didn't want to confuse their -- their data analysis by having those variables.

Q. Sure.

So they did not include in their analysis the route of exposure relevant to this case; fair?

A. They excluded studies of drinking water.

Q. And your specific causation opinions in

this case are exclusively related to exposure of drinking water; fair?

A. Well, there were other exposures in addition to drinking that were outlined.

Q. In your report, you're saying?

A. Yeah.

Q. But Dr. Reynolds and her exposure assessment considered only drinking water --

A. Okay.

Q. -- is that fair?

A. Yeah.

Q. Do you agree that the level and route of TCE exposure from drinking water is different from the level and route of TCE exposure in an occupational setting?

A. Certainly the route is different.

Q. Do you agree that the level is also different?

A. It may be, but I don't know in which direction.

Q. Do you believe that TCE in a residential water is comparable or similar to TCE in the ambient air in an occupational setting?

A. I would guess that it's more hazardous -- hazardous in water than in the occupational setting.

Q. And why is that?
A. Well, I just think if you're ingesting something, it's more hazardous than simply inhaling some volatile components of that chemical.
Q. And I think you started out your answer with it's just a guess; is that fair?
Or do you have any studies that set forth that?
A. No, I don't have any studies that report it.
Q. Have you seen any other epidemiology studies where this type of distinction is made?
A. I may have in the papers that I've looked at, but I don't recall specifically.
Q. How do you compare contaminant levels in drinking water to an occupational setting?
A. Well, they're different in the sense that the route of the chemical from entering the body is different.
Q. And how do you compare them?
How do you take a study on occupational setting and apply it to a case on drinking levels -- drinking water?
A. Well, the exposure is there. And if the exposure is significant, then -- then it's

comparable.

Q. Can you turn back to the abstract. The second-to-last sentence says (as read):

> Summary estimates for occupational TCE exposure were not associated with risk of HLMM leukemia.

A. Leukemia.

Q. Oh, leukemia, or CLL, SLL.

Do you see that sentence?

A. Yes.

Q. How do you explain the finding of a raised summary estimate for NHL, but a null result for CLL?

A. I'm not an expert on CLL, so ...

Q. Well, let me ask a different question, then.

Does that suggest TCE exposure does not have the same effect on all NHL subtypes?

A. Well, just that they were not able to demonstrate a relationship, and I don't know what their exact results were for CLL, but they did not see an increased risk.

Q. Sure.

And CLL is a subtype of NHL; fair?

A. Yes.

Q. And so the fact that they found an association for NHL and no association for CLL suggests that TCE exposure doesn't have the same effect on all subtypes of NHL; fair?

A. Yes.

Q. Could you turn to page 8?

Do you see it says (as read):

> Our meta-analytical findings for TCE exposure and NHL risk still warrant further exploration given the limited dose response patterns observed in our review and the recent conclusion by IARC that the carcinogenic evidence for TCE and NHL is limited.

Did I read that correctly?

A. Yeah. It took me awhile to find it, but I think so, yes.

Q. Okay. Have you had a chance to review that second-to-last --

A. Yeah.

Q. -- sentence now?

A. Yeah.

Q. Do you agree with that second-to-last sentence?

A. Well, I agree that it warrants further exploration.

Q. Do you know one way or the other whether IARC concluded that the carcinogenic evidence for TCE and NHL is limited?

A. No.

Q. You don't know one way or the other?

A. I -- I was not aware of that, no.

MS. HORAN: We've been going about an hour. Do you mind if we take a break, and I might be able to streamline the end of this?

THE WITNESS: Sure.

MS. HORAN: Go off the record.

THE VIDEOGRAPHER: The time is 4:00 p.m. Pacific Time. We're going off the record.

(Whereupon, a recess was taken from 4:01 p.m. to 4:12 p.m.)

THE VIDEOGRAPHER: The time is 4:12 p.m. Pacific Time. We're back on the record.

MS. HORAN: I'm marking as Exhibit 20. This is a study called "Long-term exposure to low level ambient BTEX and site-specific cancer risk: A national cohort study in the UK Biobank," by Yu and it's a 2025 study.

/ /

(Whereupon, Deposition Exhibit 20 was marked for identification.)

BY MS. HORAN:

Q. Dr. Hoppe, I don't believe this is in your report, but I believe you've mentioned this a couple of times today; is that fair?

A. That's correct.

Q. And this study focuses on low-level ambient exposure to BTEX; fair?

A. Yes.

Q. Meaning it studied lower exposure doses than occupational studies would?

A. Yes.

Q. It also evaluates long-term exposures; fair?

A. Yes.

Q. Do you know if the Yu study controls for co-exposures?

A. For?

Q. Co-exposures.

A. Meaning, like, occupational exposures or --

Q. Sure.

Being just exposed to more than just one, one thing?

A. I don't think so.
Q. This study has no data on NHL subtypes, correct?
A. That's my recollection.
Q. This study evaluates all NHLs as a whole?
A. Right.
Q. Do you know -- and I think you've testified that you don't -- you're not familiar with a term of an ecological study, so you don't know one way or the other whether this study is an ecological study; fair?
A. I don't know, but by the nature of the analysis, I suspect it may be ecological.
Q. And it didn't have individual exposure data for participants; fair?
A. Fair.
Q. If you could turn to page 5. The second column, the top paragraph, the last two sentences reads (as read):

> The exposure assessment based on residential address could not capture activity patterns of individuals, thus, potential exposure misclassification might exist. Moreover, despite the

adjustment of a series of confounders, we could not rule out residual confounding by other unmeasured factors that might affect the exposure and cancer incidents. Finally, indoor emissions are an important source of BTEX. The lack of data on individual indoor exposure is a common limitation in environmental epidemiological research and the results should be interpreted with caution.

Did I read that correctly?

A. Yes.

Q. So there's no way to know which study participants were exposed to what levels of contamination; fair?

A. That's true. But, you know, what -- what you quote is, you know, every, you know, current scientific paper mentions the strengths of their studies and the weaknesses of their studies. And, you know, I wouldn't -- I wouldn't interpret this extraction of data to, you know, reflect on the -- the overall manuscript. But yes, you are correct.

Q. Do you agree that the results should be interpreted with caution, as they state?
A. Sure.
Q. The study found a positive association between every cancer and every chemical it evaluated; fair?
A. Yes.
Q. You can set that aside.
MS. HORAN: I'm marking as Exhibit -- Hoppe Exhibit 21. This is a "Retrospective cohort study of cause-specific mortality and incidence of hematopoietic malignancies in Chinese Benzene-exposed workers." And it's Linet 2015.
(Whereupon, Deposition Exhibit 21 was marked for identification.)
BY MS. HORAN:
Q. Dr. Hoppe, you cite to this study in your report, correct?
A. Yes.
Q. And that's on page 4 of your report as it relates to Benzene?
A. Yes.
Q. Could you turn to page 2 of this study?
Do you see where it says "study population"?

A. Yes.
Q. Roughly in the middle of that paragraph, there's a section -- or a sentence that begins (as read):

> Briefly Benzene-exposed workers.

Do you see that?
A. Yes.
Q. Okay. And that reads (as read):

> Briefly Benzene-exposed workers in the spray and brush painting coatings, rubber chemical, including pharmaceutical manufacturing, shoe making and other, including printing and insulation industries were identified from those employed any length of time during 1972 to 1987 to 1,427 work units, departments, and 672 factories in 12 Chinese cities. See Table 1.

Do you see that sentence?
A. Yes.
Q. Do you agree that factory workers in China between 1972 and 1987 in coatings, chemical, shoe

making, printing and insulation industries were exposed to higher levels of Benzene than individuals at Camp Lejeune through the water between 1953 and 1987?

A. I have no idea how to compare those two.

Q. If you're not clear -- strike that.

If you're not sure how to compare the population in Linet with the population at Camp Lejeune's exposures, how does Linet factor into your analysis?

A. Well, that is simply exposure to those agents, including Benzene, resulted in an increased risk for non-Hodgkin lymphoma in this population.

Q. Sure.

And how would you take that finding and apply it to this case, which is Camp Lejeune water?

A. Well, the mechanisms of injury are the same whether you ingest it or inhale it.

Q. And is there any way for you to compare the levels of exposure between those two populations?

A. No. I think that's what I was trying to get at.

Q. Turning to your report, you also cite the Rana 2021 study?

A. Yes.
Q. You would agree that the Rana study found that the risk of developing NHL differed by subtype of NHL; fair?
A. I don't recall offhand. I have to take -- I would have to take -- I would have to take a look at that.
Q. Well, just looking at your report, you state (as read):
They reported increases in the risk for a wide variety of lymphomas --
A. Oh, yeah.
Q. (As read):
-- and specifically a doubling of the risk --
A. Yeah.
Q. (As read):
-- of diffuse large B-cell --
A. Right.
Sorry.
Q. So you agree that the Rana study found that the risk of developing NHL differed by subtype?
A. Yes.
Q. Turning to your section on PCE, every

study that you cite to includes the null value in their confidence interval, suggesting no association; fair?

A. That's correct.

Q. And you're familiar with IARC?

A. I forget what IARC stands for.

Q. International Agency for Research on Cancer.

A. Okay.

Q. You cite to IARC as a reliable source for Benzene?

A. Yes.

MS. HORAN: So I'm handing you what's been marked as Hoppe Exhibit 22.

(Whereupon, Deposition Exhibit 22 was marked for identification.)

MS. HORAN: "Trichloroethylene, tetrachloroethylene and some other chlorinated agents," and this is by IARC.

BY MS. HORAN:

Q. Have you seen this before?

A. This report is from -- I saw some part of it. I don't think I saw this entire report.

Q. Could you please turn to page 329?

MR. MCGOWAN: Can I just hand him this

one?

MS. HORAN: That's fine with me.

MR. MCGOWAN: This is page 329.

THE WITNESS: I got it.

MR. MCGOWAN: All right.

THE WITNESS: Okay.

BY MS. HORAN:

Q. And do you see where it's 6.3 overall evaluation?

A. Yes.

Q. And it says (as read):

Tetrachloroethylene is probably carcinogenic to humans, group 2A.

A. Yes.

Q. And if you turn to page 326.

A. Okay.

Q. Do you see a 5.2.2, other cancer sites?

A. Correct.

Q. Do you see it says (as read):

Several studies evaluated exposure to tetrachloroethylene and the risk of cancers at other sites, including esophagus, kidney, cervix and non-Hodgkin's lymphoma, no

consistent patterns were seen across studies.

A. Yes.

Q. Do you agree with that assessment?

A. Well, that's their assessment -- assessment based on the studies they reviewed, yes.

Q. And based on the studies you've reviewed, do you agree with that assessment?

A. I would have to look at their -- at the sources that they use to make this conclusion.

Q. Could you turn to page 327?

A. Yeah.

Q. So right above 5.3, there's a sentence that starts "For non-Hodgkin lymphoma."

Do you see that?

A. Yes.

Q. It says (as read):

> For non-Hodgkin lymphoma, three cohort studies showed an increased risk based on small numbers and the largest study with the best control of potential confounders did not. Case control studies on non-Hodgkin lymphoma did not find significant associations.

Did I read that correctly?
A. Yes.
Q. IARC is a respected organization; fair?
A. Yes.
Q. You can set that aside.
Do you see Hoppe Exhibit 23? It says (as read):
ATSDR assessment of the evidence -- sorry.
Let me start over.
MS. HORAN: I am marking as Exhibit 23, Hoppe Exhibit 23, "ATSDR assessment of the evidence for the drinking water contaminants at Camp Lejeune and specific cancers and other diseases."
And it's dated January 13th, 2017.
(Whereupon, Deposition Exhibit 23 was marked for identification.)
BY MS. HORAN:
Q. Dr. Hoppe, this is the ATSDR study that you discuss on pages 5 and the top of 6 of your expert reports; fair?
A. Yes.
Q. Have you read Dr. Bove's deposition transcript?
A. No.

Q. Do you know how long an epidemiological study takes to plan and perform?

A. Depending on the nature of the epidemiological study, I would say at least three or four years if they are retrospective.

Q. Did you know that Dr. Bove performed ATSDR systematic review of four chemicals and 16 health outcomes at Camp Lejeune in six weeks?

A. Well, he completed that analysis.

Q. In six weeks, were you aware of that?

A. No.

Q. And do you know if he had any help in performing the analysis?

A. No.

Q. Are you aware that Dr. Bove testified that the 2017 assessment's purpose was to add diseases to the VA presumption list?

A. No.

Q. The 2017 assessment did not use significance testing to assess the evidence for causality; fair?

A. I'm sorry, can you repeat that?

Q. The 2017 assessment did not use significance testing to assess the evidence for causality; fair?

A. I don't recall. I mean, it looked at multiple studies and recorded whether they were significant or not.

Q. Do you see on page 8, the last paragraph on page 8 says (as read):

> In our assessment, we did not use confidence intervals to determine whether a finding was statistically significant, nor did we use significance testing to assess the evidence for causality.

Did I read that correctly?

A. Yes.

Q. So you agree that ATSDR did not use significance testing to assess the evidence?

A. Yes.

Q. Instead, it looked at the ratio of the upper end of the confidence interval to the lower end, correct?

A. Are you reading something?

Q. I believe that's what it says in the paragraph just above.

A. Okay.

Q. Are you aware of whether it's generally acceptable in epidemiology to use a confidence

interval ratio?

A. I'm not aware.

Q. In the sentence -- the last sentence of the second-to-last paragraph says (as read):

An effect estimate --

A. Sorry, where are you reading from?

Q. Page 8 still.

A. Okay.

Q. The second-to-last paragraph. It's a short one.

A. Yeah.

Q. Begins "In the disease."

A. Yeah.

Q. The second sentence says (as read):

An effect estimate, e.g., risk ratio, odds ratio or standardized mortality ratio was considered to have good precision or less uncertainty if the ratio of the upper limit to the lower limit of its 95 percent confidence interval was less than or equal to 2.

A. Okay.

Q. Have you ever used this parameter before?

A. No.

Q. Have you ever seen an authority that suggests that this is a common or a standard methodology?

A. No. Again, I'm not an epidemiologist, so I don't travel in those circles.

Q. If you turn to page 6 or 7 -- or 6. Sorry. This classification scheme categories section, which runs from 6 to 7, references an epidemiological study considered to be of high utility.

And I believe you referenced that language in your report as well.

A. Yes.

Q. How do you define "high utility"?

A. Can you show me again where that phrase is used?

Q. So it's used at least on page 7, number 2.

A. Oh, okay, I was looking at page 6.

Q. Partially down the middle, but it might be -- it's also in, I believe, number 3. I think they use it a couple times.

A. Well, I would say high utility would mean a very useful study.

Q. And how would you determine whether a study was very useful?

A. Well, that would be based on -- in my mind, on number of patients, duration of followup, the confidence related to exposure and exposure duration.

Q. Anything else you can think of right now?

A. The elimination of other -- other effects that might influence the development of endpoint.

Q. Anything else?

A. Not offhand.

Q. In rendering your opinion, did you rely on high utility epidemiological studies as you've defined the term?

A. I believe so.

Q. And what studies -- what high utility epidemiological studies did you rely on that you would consider high utility as you just defined it?

A. The -- well, essentially all of the studies that I included in my report.

Q. All of those studies, in your opinion, would qualify as high utility as you've just defined the term?

A. Yes.

MS. HORAN: I think I am wrapped up. Do you mind if we go off the record for one or two minutes and I can confer with my colleague?

MR. ELLIOTT: Absolutely.
THE VIDEOGRAPHER: The time is 4:36 p.m. Pacific Time. We're going off the record.
(Whereupon, a recess was taken from 4:36 p.m. to 4:38 p.m.)
THE VIDEOGRAPHER: The time is 4:38 p.m. Pacific Time. We're back on the record.
MS. HORAN: Dr. Hoppe, thank you very much for your time today. I don't have any further questions for you.
I will just note, I don't believe we have materials considered list that includes Dr. Ambinder's reports. We'll check our files again, but we'll follow up with you because I believe Dr. Hoppe testified that he has read and reviewed those. So we'll go through our files, and we'll follow up with you afterwards, but I just wanted to note that for the record.
MR. MCGOWAN: Okay. All right. We're going to take five minutes ourselves. We'll be right back.
MS. HORAN: Sure.
THE VIDEOGRAPHER: Time is 4:39 p.m. Pacific Time. We're going off the record.
(Whereupon, a recess was taken from

4:39 p.m. to 4:45 p.m.)

THE VIDEOGRAPHER: The time is 4:45 p.m. Pacific Time. We're back on the record.

EXAMINATION BY MR. MCGOWAN

BY MR. MCGOWAN:

Q. Doctor, could you describe briefly for the judges who will read this one day what your day-to-day work entails outside of any litigation context?

A. Sure.

That's nearly all of my time. And as I indicated earlier, about 60 percent of my time is in clinical practice, and that includes seeing new patients with cancer, specifically lymphoma, since my practice is limited to lymphoma, seeing those patients in consultation, treating them when appropriate, and also following them after the completion of therapy in long-term followup.

That's 60 percent of my time.

The other 40 percent includes teaching. We have teaching programs with residents and fellows and medical students who are learning the disciplines of cancer treatment. And also, clinical research. I'm involved in a number of clinical trials related to the treatment of patients with

lymphoma. And the followup of long-term complications of lymphoma, lymphoma treatment.

And then an additional proportion of my time is spent in administration. I currently chair the department committee for appointments and promotions.

Q. How many years have you been treating, teaching and researching cancer, lymphoma in particular?

A. So I've been on the faculty at Stanford for 49 years, and I would say that 47 of those I've been limited to the -- to lymphoma, my practice. And throughout that time, I've been involved in teaching and research.

Q. Okay. Is it accepted in your field that PCE, TCE and Benzene can cause NHL?

A. Yes.

Q. And the exposure numbers in your report, were those cumulative numbers?

MS. HORAN: Objection to form.

THE WITNESS: Sorry?

MS. HORAN: You can answer. I object, Dr. Hoppe, but you should answer the question. I just said objection, form.

THE WITNESS: Okay. Can you repeat the

question?
BY MR. MCGOWAN:
Q. Yeah. The exposure numbers in your report, were those cumulative?
A. Yes.
MS. HORAN: Same objection.
BY MR. MCGOWAN:
Q. All right. I want to draw your attention to the Bove 2024 study that we talked about earlier, some hours ago.
And do you remember the .89 hazard ratio for DLBC?
A. Right.
Q. Now, if we just take that on its face, would that -- could you -- could someone misrepresent that to say, hey, look, because it's .89, these chemicals are protective or preventive of that disease?
A. No. I wouldn't draw that conclusion.
Q. Right.
Are there limitations on that study and every study?
A. Yes.
Q. All right. Do scientists who actually want to know the truth cherry-pick a single study or

do they look at the body of science as a whole in making determinations and judgments?

A. They look at the overall body of data.

Q. Okay. And does a single study make the body of science in any given topic?

A. No.

Q. Now, the IARC meeting, the publication that was put in front of you, do you recall that?

A. Yes.

Q. Are you aware that that meeting was held in 2012 and it was published in 2014?

A. I did notice that it was published in 2014.

Q. And today is now 2025, so we have 11 years more of experience and research between then and now?

A. Yes.

Q. All right. We keep hearing this throughout this case about the confidence interval including the null. Okay.

That -- the fact that a confidence interval includes the null does not dictate or suggest that there's no association; is that true?

It's just a mathematical question?

MS. HORAN: Objection. Form.

THE WITNESS: Yes. That's -- that's correct. It does not mean there's no association. This is statistics and confidence intervals reflect the statistics.

BY MR. MCGOWAN:

Q. Okay. And is it -- is it correct and fair to say that a study with a confidence interval includes the null does not show an association?

Is that -- is that a true statement? Is that a fair statement?

MS. HORAN: Objection. Form.

THE WITNESS: No.

BY MR. MCGOWAN:

Q. Okay. And last, is it -- is it true that when we're talking about water concentrations, micrograms equals parts per billion, that's just math?

MS. HORAN: Objection. Form.

THE WITNESS: Yes.

MR. MCGOWAN: Okay. That's all I have for you, Doctor. Thank you.

MS. HORAN: I just have one question.

FURTHER EXAMINATION BY MS. HORAN

BY MS. HORAN:

Q. I think earlier today you testified that

you did not assess whether micrograms were the same as parts per billion, and I believe you just testified that they are the same.

What changed?

A. I checked on that.

Q. And what did you look at?

A. Well, I -- I asked the attorneys.

Q. And they told you that?

A. Yeah.

MS. HORAN: No further questions.

THE VIDEOGRAPHER: This concludes today's testimony given by Dr. Richard Hoppe. Going off the record at 4:51 p.m. Pacific Time.

(Whereupon, the deposition concluded at 4:51 p.m.)

--oOo--

I declare under penalty of perjury the foregoing is true and correct. Subscribed at _____, _____,
CITY STATE

this ___ day of _____, 2025.

_____
WITNESS SIGNATURE

CERTIFICATE OF REPORTER

I, Kathleen A. Maltbie, Certified Shorthand Reporter licensed in the State of California, License No. 10068, the State of Nevada, CCR 995, and the State of Texas, CSR 12212, hereby certify that deponent was by me first duly sworn, and the foregoing testimony was reported by me and was thereafter transcribed with computer-aided transcription; that the foregoing is a full, complete, and true record of proceedings.

I further certify that I am not of counsel or attorney for either or any of the parties in the foregoing proceeding and caption named or in any way interested in the outcome of the cause in said caption.

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In witness whereof, I have hereunto set my hand this day:

_____ Reading and Signing was requested.

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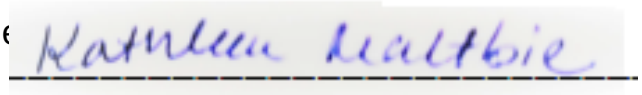
___x___ Re  uested.

_____

KATHLEEN A. MALTBIE
RPR-RMR-CRR-CCRR-CLR-CRC-RDR
California CSR 10068, Nevada CCR 995
Texas CSR 12212

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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

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