Exhibit 602

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

IN RE: CAMP LEJEUNE WATER LITIGATION,	)	
This Document Relates To:	)	No. 7:23-CV-897
ALL CASES	)	

Video Deposition of HOWARD HU, PH.D., taken at Weitz & Luxenberg, 1880 Century Park East, Suite 700, Los Angeles, California, commencing at 9:10 a.m., on Wednesday, July 23, 2025, reported stenographically by Lisa Moskowitz, California CSR 10816, Certified Realtime Reporter, Nevada CCR 991, Washington CCR 21001437, Illinois CSR 084.004982, RPR, CLR, NCRA Realtime Systems Administrator.

GOLKOW, a Veritext Division
877.370.3377 ph | 917.591.5672 fax

APPEARANCES:

On behalf of Plaintiff:
WEITZ & LUXENBERG
BY: ROBIN L. GREENWALD
rgreenwald@weitzlux.com
1880 Century Park East
Suite 700
Los Angeles, California 90067
(310) 247-0921

On behalf of Defendant:
U.S. DEPARTMENT OF JUSTICE
BY: GIOVANNI ANTONUCCI
giovanni.antonucci@usdoj.gov
BY: AMY KLIMEK
amy.a.klimek@usdoj.gov
1100 L Street NW
Washington, DC 20005
(202) 514-2000

ALSO PRESENT VIA ZOOM:

ALLISON O'LEARY
MATTHEW ELLIOTT
MARCUS TUBIN
DIANA GJONAJ

VIDEOGRAPHER:

TORR PIZZILLO,
GOLKOW TECHNOLOGIES

I N D E X

DEPOSITION EXHIBITS

(STENOGRAPHER'S NOTE: All quotations from exhibits are reflected in the manner in which they were read into the record and do not necessarily denote an exact quote from the document.)

QUESTIONS NOT ANSWERED

PAGE	LINE
312	20

WEDNESDAY, JULY 23, 2025
LOS ANGELES, CALIFORNIA
9:10 A.M.

THE VIDEOGRAPHER: We are now on the record. My name is Torr Pizzello. I'm a videographer for Golkow, a Veritext division. Today's date is July 23, 2025, and the time is 9:10 a.m. This video deposition is being held at 1880 Century Park East, Los Angeles, California in re Camp Lejeune Water Litigation versus United States of America for the U.S. District Court for the Eastern District of North Carolina, Southern Division. The deponent is Dr. Howard Hu.

Counsel will be noted on the stenographic record. Your court reporter is Lisa Moskowitz and will now introduce herself and swear in the witness.

THE CERTIFIED STENOGRAPHER: My 00:-2625 name is Lisa Moskowitz. I'm a

California certified stenographic reporter. My CSR license number is 10816.

00:-26 5

HOWARD HU, PH.D.,

called as a witness,

was examined and testified as follows:

EXAMINATION

BY ATTORNEY ANTONUCCI:

Q. Good morning.

A. Good morning.

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

A. Howard Hu.

Q. Could you please state your current business address?

A. 1845 North Soto Street, Los Angeles, California 90089.

Q. Dr. Hu, my name is Giovanni Antonucci. I'm an attorney for the Department of Justice. I represent the United States in the Camp Lejeune Water Litigation that's currently pending in the Eastern District of North Carolina.

Dr. Hu, you've had your deposition taken before; is that correct?

A. Correct.

Q. And you've had your deposition taken before many times; is that right?

ATTORNEY GREENWALD: Objection to form.

THE WITNESS: A bunch of times.

BY ATTORNEY ANTONUCCI:

Q. Could you please estimate approximately how many times you've been deposed?

A. Lifetime?

Q. Yes, sir.

A. I'd say maybe 30 or 40.

Q. Okay. So, Dr. Hu, you know the rules. I'm just going to go over some of the basics for the record.

You took an oath before we started this morning; is that correct?

A. Correct.

Q. And you understand the nature of that oath; right?

A. Yes.

Q. Dr. Hu, as you can see, a court

reporter is taking down everything we say. Because she can only record words, it's important that you answer questions verbally. For example, you must say yes or no instead of nodding or shaking your head. Okay?

A. Yes.

Q. Only you are testifying today, Dr. Hu. You must answer to the best of your ability, and you may not ask others for their help.

Do you understand?

A. I do.

Q. If you don't understand a question, please let me know, and I will try to clarify. If you don't ask for clarification, I will assume you understood the question.

Is that fair?

A. Yes.

Q. During the deposition, you may hear your attorney say: Objection. Unless she instructs you not to answer the question, please answer the question after the objection has been made.

Do you understand?
A. I understand.
Q. Is there any reason why you're unable to give your most truthful and accurate testimony today?
A. No.
Q. Is there any reason why your memory might be impaired?
A. No.
Q. Dr. Hu, you may ask for breaks at any time. Please just answer any questions I've asked before we go take a break.
Are you all right with that?
A. Yes.
Q. Am I correct, Dr. Hu, that you've been retained by the plaintiffs' leadership group as an expert witness in the in re Camp Lejeune water litigation?
A. Yes.
Q. And you were retained as an expert witness in general and specific causation; is that correct?
A. Correct.
Q. Okay. Were you retained for both at the same time?

A. I actually don't recall.
Q. Before being retained, had you heard about Camp Lejeune?
A. Yes.
Q. What did you know about Camp Lejeune prior to being retained?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: In the 2010s, I was a member of the Board of Population and Public Health Practice of the National Research Council of the United States, and part of our oversight was to commission the study of Camp Lejeune done by the Centers for Disease Control.
BY ATTORNEY ANTONUCCI:
Q. Are you referring to the 2009 study on the assessment of the evidence of health effects at Camp Lejeune?
A. I think it was a study after that. Or maybe -- actually, I think we were there to review the study. It was commissioned by the board before I became a member of the board, but then our charge was to oversee

the conduct of the study and the progress made.

Q. Other than overseeing the conduct of the study and progress made, did you have any other involvement with the NRC study published in 2009?

A. I did not.

Q. When you say that you were charged with overseeing the conduct of the study, what do you mean by that?

A. As a board, our charge was just to listen to the progress that was being made, look at any questions that arose, and use the expertise of the board to provide any guidance or feedback or criticisms.

Q. Did you have any role in the peer-review process for that report?

A. No.

Q. Did you read the report prior to it being released publicly?

A. Not that I recall.

Q. Did you perform any kind of scientific evaluation of the 2009 NRC report on Camp Lejeune?

A. I think I may have read it, but

other than that, I don't recall.
Q. And you read it after it was released publicly; is that correct?
A. Correct.
Q. Do you have any opinions on the quality of the 2009 NRC report on Camp Lejeune?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: Not as I sit here today.
ATTORNEY ANTONUCCI: I am marking for identification Exhibit 6.
BY ATTORNEY ANTONUCCI:
Q. This is your amended deposition notice.
(Exhibit Number 6 was marked for identification.)
BY ATTORNEY ANTONUCCI:
Q. Dr. Hu, have you seen this document before?
A. Yes.
Q. I will represent to you that this is the amended deposition notice for the deposition we're currently sitting at; is

that right?
A. Yes.
Q. And you can put that to the side now. I appreciate that.
I'm now marking as Exhibit 7 the initial deposition notice and subpoena.
(Exhibit Number 7 was marked for identification.)
BY ATTORNEY ANTONUCCI:
Q. When you've had a chance to look through that, please look back up at me.
Dr. Hu, I will represent that that is the original deposition notice and subpoena for your testimony prior to this deposition being rescheduled.
Do you have any of the materials mentioned in the subpoena in your custody or control?
A. At home, not here.
Q. Which of the materials mentioned in the subpoena are in your custody or control?
A. It's a long list, Counsel. You really want me to go through it?
Q. I'll represent to you that your counsel has objected to the subpoena and

stated that you have no responsive materials in your custody or control.

Do you disagree with that?

A. Let's see. I'm sorry. I thought this also included all materials considered in my reports. This is actually something else. Emails, letters, correspondence, text messages to these particular people and the plaintiffs.

Well, I mean, this also states all bills, invoices, or documents reflecting compensation; so I have copies of that and so does plaintiffs' counsel.

Q. Okay. So you've provided copies of bills and compensation to attorneys for the plaintiffs; is that correct?

A. I think I did, yeah. I'm just about done, but may I ask you a question? On page 5, number 12, a copy of any studies related to idiopathy.

Can you define what you mean by that?

Q. Dr. Hu, I think I'd actually like to move on from our discussion of the subpoena. We've received the objections

from your counsel.

A. Okay.

Q. You can put that to the side, please.

I'm going to hand you what I've marked for identification as Exhibit 8.

(Exhibit Number 8 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. I represent to you this is the copy of your CV that was produced to the United States on June 12, 2025. I'd like you to please turn to the last page Bates-numbered CL_PLG_expert_hu_202.

A. Yes.

Q. This document is titled Consulting Rates As of July 1, 2024; is that right?

A. Correct.

Q. Are these your current litigation consulting fees?

A. Yes.

Q. Okay. You can put that to the side. Thank you, Doctor.

Dr. Hu, are you a hematologist?

A. No.

Q. And you're not an oncologist; is that correct?
A. Correct.
Q. So you are not a medical or radiation oncologist; correct?
A. Correct.
Q. Have you ever treated a patient for non-Hodgkin's lymphoma?
A. Yes.
Q. In what capacity have you treated a patient for non-Hodgkin's lymphoma?
A. As a resident in internal medicine at Boston City Hospital.
Q. Did your residency include an oncology rotation?
A. It did.
Q. And that's the capacity in which you treated a non-Hodgkin's lymphoma patient?
A. Yes.
Q. How long was your oncology rotation during your residency?
A. Three months.
Q. Did you only treat the one non-Hodgkin's lymphoma patient during that

time?

A. I think there were several. There was a cancer ward at University Hospital of Boston University.

Q. Okay. Other than the several non-Hodgkin's lymphoma patients you treated as a resident in internal medicine, have you otherwise treated patients for non-Hodgkin's lymphoma?

A. Consulted on but not treated.

Q. When you say "consulted on," what do you mean by that?

A. During my practice as an occupational environmental medicine physician, I'm sometimes asked to consult on patients who develop non-Hodgkin's lymphoma with respect to understanding the etiology.

Q. And in your treatment of patients with non-Hodgkin's lymphoma as a resident, did your treatment depend on the subtype of NHL that the patient presented with?

A. Well, this is in the 1980s. It's really too far ago for me to recall.

Q. Okay. Do you recall whether you've ever diagnosed a case of non-Hodgkin's

lymphoma?

A. As a first diagnosis, not that I recall.

Q. Dr. Hu, you are not a geneticist; is that correct?

A. Correct.

Q. You are not an immunologist; correct?

A. Correct.

Q. And you are not a toxicologist; correct?

A. Correct.

Q. You are not an expert in exposure assessment; is that correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I would disagree with that. I spent a lot of my training in occupational environmental medicine and environmental epidemiology reviewing, learning, studying, and doing research on exposure assessment.

So it's a discipline that I use a lot in my own profession as an

environmental epidemiologist; so I would disagree with that characterization.

BY ATTORNEY ANTONUCCI:

Q. Okay. Do you have any certification in exposure assessment?

A. No.

Q. Have you ever been a principal investigator for an exposure assessment study?

A. Yes.

Q. Approximately how many exposure assessment studies have you served as a principal investigator for?

A. Maybe 10 or 12.

Q. Have you ever taught a course on exposure assessment?

A. It was part of my environmental epidemiology course that I taught.

Q. Are you an expert in risk assessment?

A. I would not say that's true.

Q. Okay. Have you ever performed a human health environmental risk assessment?

A. Not per se.

Q. Dr. Hu, you've never published peer-reviewed literature regarding the effects of TCE on non-Hodgkin's lymphoma or any of its subtypes; correct?

A. Correct.

Q. You've never published peer-reviewed literature regarding the effects of PCE on non-Hodgkin's lymphoma or any of its subtypes; correct?

A. Correct.

Q. You've never published peer-reviewed literature regarding the effects of vinyl chloride on non-Hodgkin's lymphoma or any of its subtypes; correct?

A. Correct.

Q. You've never published peer-reviewed literature regarding the effects of benzene on non-Hodgkin's lymphoma or any of its subtypes; correct?

A. I believe I published a case report of benzene and multiple myeloma, and multiple myeloma has been included by some as a subtype of non-Hodgkin's lymphoma.

Q. Other than the case report on multiple myeloma, have you published any

other peer-reviewed literature regarding the effects of benzene on non-Hodgkin's lymphoma or any of its subtypes?

A. No.

Q. In your practice as an environmental epidemiologist or emergency medicine physician, have you ever treated individuals with diseases that were exposed to the water at Camp Lejeune?

A. I'm not an emergency physician, but apart from that, I have not treated anybody or evaluated anybody from Camp Lejeune, as far as I know.

Q. Thanks. I'm sorry about that.

A. It's all right.

Q. Dr. Hu, you're aware that one of the plaintiffs in the Camp Lejeune water litigation is Mr. Robert Kidd; is that correct?

A. Yes.

Q. Have you ever examined Mr. Kidd?

A. I have not.

Q. Have you ever communicated directly with Mr. Kidd?

A. No.

Q. Have you ever communicated indirectly with Mr. Kidd?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No.

BY ATTORNEY ANTONUCCI:

Q. You're aware that Ronald Carter is one of the plaintiffs in the Camp Lejeune water litigation; is that correct?

A. Yes.

Q. Have you ever examined Mr. Carter?

A. I have not.

Q. Have you ever communicated directly with Mr. Carter?

A. No.

Q. Have you ever communicated indirectly with Mr. Carter?

A. I might have posed a question for attorneys to pose to his widow, I believe.

Q. And Mr. Carter's widow is named Frances Carter; is that right?

A. I believe so.

Q. Are the -- is the result of the question you posed to the widow of Frances Carter in your report?

A. It would have been related to history that would have been summarized in my summary of his case.

Q. Okay. I've premarked several exhibits that I'd just like to show you to confirm that they are your reports. So this first one is Exhibit 1.

(Exhibit Number 1 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. I'll represent to you that this is your general causation report and materials considered lists from February 14, 2025, and the supplement of April 29, 2025.

Dr. Hu, this is the general causation report that you issued in the Camp Lejeune water litigation; correct?

A. Correct. But you mentioned it also contains my supplement. I'm just looking for that to make sure that it does.

Q. I apologize. It contains the materials considered list supplements. The January 31, 2025, supplement will be a separate exhibit.

A. Okay.

Q. So you can confirm this is your general causation report, excluding the January 31, 2025, supplement?

A. Yes.

Q. Okay. So the next exhibit is your January 31, 2025, supplement. This is Exhibit 2.

(Exhibit Number 2 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, can you please confirm that Exhibit 2 is your Phase 2 general causation report supplement, dated January 31, 2025?

A. Confirmed.

Q. Thank you.

Next is Exhibit 3. I will represent to you that this is the Phase 3 specific causation initial report that you disclosed for Mr. Robert Kidd on June 12, 2025.

(Exhibit Number 3 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Can you please confirm that Exhibit 3 is your specific causation report

for Mr. Robert Kidd?
A. Confirmed.
Q. Thank you.
Exhibit 4 is the specific causation report that you disclosed for Frances Carter for the estate of Ronald Lee Carter in the Camp Lejeune water litigation on June 12, 2025.
(Exhibit Number 4 was marked for identification.)
BY ATTORNEY ANTONUCCI:
Q. I ask that you please review this and confirm that it is your initial specific causation report for Mr. Carter.
A. Confirmed.
Q. And now Exhibit 5 is your rebuttal report to Dr. Richard F. Ambinder for Robert Arnold Kidd, dated May 16, 2025.
(Exhibit Number 5 was marked for identification.)
BY ATTORNEY ANTONUCCI:
Q. Please review this and confirm that it is your rebuttal report to Dr. Ambinder for Mr. Kidd.
A. Confirmed.

Q. Great.

I'm handing you what I've marked as Exhibit 9. This is the rebuttal report to Dr. Richard F. Ambinder for Ronald Lee Carter, dated May 16, 2025.

(Exhibit Number 9 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Please take a look and confirm that Exhibit 9 is that report.

A. Confirmed.

Q. I'm now handing you what I've marked for identification as Exhibit 10. This is your rebuttal report of Dr. Lisa A. Bailey for Robert Arnold Kidd, dated May 16, 2025.

(Exhibit Number 10 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Please take a look at Exhibit 10 and confirm it is that report.

A. Confirmed.

Q. I'm now handing you what I've marked for identification as Exhibit 11.

///

(Exhibit Number 11 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. This is the rebuttal report to Dr. Lisa A. Bailey for Ronald Lee Carter, dated May 16, 2025, that you disclosed in this case. Please take a look at Exhibit 11 and confirm it is that report.

A. Confirmed.

Q. Thanks, Dr. Hu.

Did you speak with any other retained experts in this case while preparing your reports?

A. Which one?

Q. Did you speak with any other retained experts in this case while preparing any of your reports?

A. Oh. I think at some point I chatted with Dr. Felsher in the presence of counsel.

Q. And you reviewed the expert report of Dr. Kelly Reynolds disclosed on February 7, 2025; is that correct?

A. Yes.

Q. And you also reviewed the expert

report of Mr. Morris Maslia disclosed October 25, 2024; is that correct?

A. Yes.

Q. Did you review the entire expert report of Mr. Maslia or just portions of it?

A. I skimmed it and then reviewed specific portions.

Q. Which specific portions of Mr. Maslia's expert report did you review?

A. Things that related to his estimation of likely what are contaminant levels.

Q. You're aware that Mr. Maslia is a civil engineer; correct?

A. Yes.

Q. Are you also an expert in civil engineering?

A. I am not.

Q. Are you aware that Mr. Maslia's report offers several opinions on ATSDR's effort to use computer modeling to calculate average monthly concentrations of contaminants in drinking water?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I don't recall that specific activity.

BY ATTORNEY ANTONUCCI:

Q. Have you read any of ATSDR's water modeling reports related to contamination at Marine Corps base Camp Lejeune?

A. Nothing separate from ATSDR's draft and final report on Camp Lejeune.

Q. When you say that you reviewed ATSDR's draft report on Camp Lejeune, what are you referring to?

A. At some point they had a report that was in draft form before it was finalized.

Q. Dr. Hu, you're aware ATSDR has released several reports on water contamination at Marine Corps base Camp Lejeune; correct?

A. I'm referring to their -- it's in my materials considered list. I can't remember the title of the report.

Q. Do you recall approximately when you reviewed a draft report of Marine Corps base Camp Lejeune?

A. Maybe two-and-a-half or three years

ago.

Q. So is it possible that you reviewed the 2024 cancer incident study published by Frank Bove prior to its publication?

A. That's separate from what I'm talking about. I'm talking about a ATSDR monograph about Camp Lejeune. At least that's what I thought you and I were talking about.

Q. Okay. We'll come back to that.

Dr. Hu, in your reports, you stated that you relied on the general causation reports which you also authored, dated December 9, 2024, and January 31, 2025; is that correct?

A. Yes.

Q. And that the general causation report and supplement are included in your materials relied on list and hereby incorporated by reference in their entirety as if fully set forth herein; correct?

A. Correct.

Q. What does that mean?

A. It means that the report that I authored on general causation and its

addendum remains, in my view, my opinions, and I stand by them.

Q. Okay. Dr. Hu, I'd like you to please turn to Exhibit 1. That is your general causation report. And I'd like you to look, please, to page 7.

A. Okay.

Q. So at the bottom of page 7, there's a section titled Standard Applied, and, again, this is Exhibit 1; is that right?

A. Yes.

Q. Okay. And in this section of page 7 of Exhibit 1, you quote portions of the text of the Camp Lejeune Justice Act of 2022; correct?

A. Correct.

Q. Dr. Hu, how does your background qualify you to review and interpret the Camp Lejeune Justice Act of 2022's legal causation standard?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I am not a lawyer, but I think that the text here speaks for itself.

BY ATTORNEY ANTONUCCI:

Q. What do you mean by "the text speaks for itself"?

A. Well, as I quoted here: The plaintiffs must show one or more relationships between the water at Camp Lejeune and the harm.

To me, that means that they have to -- if they're alleging that the water was responsible for a particular cancer, they need to show what was in the water that may have caused cancer, what cancer are we talking about, what's the evidence that a particular substance could cause that cancer, and then what's the evidence that that particular plaintiff had the kinds of exposure and the kinds of exposure characteristics, timing, and other characteristics that would tie them towards the cancer that they developed.

That second quote: To meet the burden of proof, parties should produce evidence showing that the relationship between exposure to water at Camp Lejeune and the harm is -- it states: Several

conditions sufficient to conclude that a causal relationship exists.

I think I address that in my reports. I think looking at causal relationships and opining about them is part of my profession. That's a part of what I've done over many years.

Condition B, or -- I'm sorry.

Or condition B: Sufficiently conclude that a causal relationship is at least as likely or not is a particular condition that seems to be specific for the Camp Lejeune Justice Act, and I interpreted it as best as I could in my evaluations.

Q. Okay. Dr. Hu, you just said that you interpreted the "at least as likely as not" standard to be -- as best as you could; is that right?

A. Correct.

Q. In your view, what is your interpretation of the "at least as likely as not standard" in the Camp Lejeune Justice Act?

A. My interpretation is that there's a body of evidence that may not make the more

likely than not but at least as likely or not is similar. Could be thought of as a 50 percent, you know, probability. Some call it equipoise, but it's, I guess you could say, one notch below more likely than not.

Q. Dr. Hu, on page 8 of Exhibit 1, you also quote ATSDR's 2017 assessment of the evidence for the drinking water contamination at Camp Lejeune and specific cancers and other diseases; is that right?

A. Yes. And that's the report I thought we were discussing earlier in our deposition.

Q. Understood. My apologies for the confusion there.

So the ATSDR's 2017 assessment of the evidence for drinking water contamination at Camp Lejeune and specific cancers and other diseases is the ATSDR report that you reviewed a draft of; is that correct?

A. I think so. Yes.

Q. Why did you review a draft of that report?

A. It was furnished to me.
Q. Was this furnished to you in your capacity as a litigation consultant or as part of your professional work?
A. That's a good question. I really don't recall. I think it was probably because of my membership on one of the National Research Council expert committees. And now that I think about it, I think earlier in the deposition, I said I was part of the board of Population and Public Health Practice. I think it was part of this other board I served on called the board of Environmental Studies and Toxicology.
Q. Did you serve as a peer reviewer on the ATSDR's 2017 assessment of the evidence?
A. Not formally. I was just a member of the board trying to provide oversight on the progress of the study.
Q. Did you provide criticism or feedback on the study to ATSDR?
A. I'm sure I made some comments, but I don't recall the nature of those comments currently.
Q. Do you recall whether you reviewed

those as a Microsoft Word document?
A. No.
Q. Do you have your comments on the ATSDR's 2017 assessment of the evidence in your possession?
A. No.
Q. Okay. Could you find your comments on ATSDR's 2017 assessment of the evidence if you were asked to?
A. You'd have to look -- consult the National Research Council. I'm sure they took minutes at the meetings, and they could find me as a member and find out where they have documented comments. I don't have them.
Q. Do you recall whether you ever provided written comments on ATSDR's 2017 assessment of the evidence?
A. Not that I recall. I think it was verbal at meetings.
Q. Okay. So on page 8 of Exhibit 1, your general causation report, you comment on ATSDR's use of four categories to classify the strength of the evidence for a causal relationship between chemicals in the

water at Camp Lejeune and various harms.

Do you see where I'm reading from there?

A. Yes.

Q. Do you know where ATSDR got their equipoise and above standard from for their 2017 assessment of the evidence?

A. I don't specifically. I mean, I say in my report here that they employ the same language as the Camp Lejeune Justice Act. So my presumption, sitting here, is it probably comes directly from the Act.

Q. Okay. Dr. Hu, are you aware that the ATSDR's assessment of the evidence was published several years before the Camp Lejeune Justice Act was enacted?

A. No, I'm not -- I don't follow the litigation on this; so I was not aware of that.

Q. Okay. So on page 8 of Exhibit 1, you note that ATSDR, in their 2017 assessment of the evidence, used equipoise and above as a category of classification; is that correct?

A. Yes.

Q. How do you understand the term "equipoise and above"? What does that mean to you?

ATTORNEY GREENWALD: Objection. Asked and answered.

THE WITNESS: Well, as I explained here, equipoise and above: The evidence is sufficient to conclude that a causal relation is at least as likely as not but not sufficient to conclude that a causal relationship exists. That's how I interpreted it.

BY ATTORNEY ANTONUCCI:

Q. Okay. Also, in Exhibit 1 on page 8, you state that you expressed some of your opinions in your report under a more likely than not standard; is that correct?

A. Correct.

Q. Could you define more likely than not, please?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: A notch above equipoise and above. It's, you know -- I'm not sure how you get

beyond more likely than not as expressed. It's more likely than not. Seems rather obvious to me. Some would say, you know, 51 percent probability or above. But I think that's a sufficient way to characterize it quantitatively. But I think other than that, I think more likely than not is a relatively self-explanatory term.

BY ATTORNEY ANTONUCCI:

Q. Okay. And of the more likely than not and at least as likely as not standards, which did you apply in your specific causation report?

A. Well, I applied either, whichever made sense to me.

Q. Is that the same for your general causation reports?

A. Correct.

Q. And is the standard you applied in your specific and general causation reports based on the text of the Camp Lejeune Justice Act of 2022?

A. It's based on essentially what I

wrote here.

Q. Dr. Hu, I ask now that you put that to the side. We'll be coming back to it; so please try to keep that binder clipped.

A. Sure.

Q. I appreciate that.

I'd like for you to turn to Exhibit 4. That's your specific causation initial report for Mr. Carter.

ATTORNEY GREENWALD: That's one you didn't give me. I didn't bring it in paper, and I can't get on my computer; so do you mind handing it to me?

ATTORNEY ANTONUCCI: Not at all.

ATTORNEY GREENWALD: I had all the other ones, but that was the one I didn't bother.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, could you please turn to page 14 of Exhibit 4. The paragraph above the line of asterisks on page 14 of Exhibit 4 starting with the word "thus."

Do you see that?

A. Yes.

Q. That reads: Thus given my general causation assessment and the factors reviewed above, it is my opinion, to a reasonable degree of medical certainty, that the combination of Mr. Carter's exposures to TCE, PCE, and benzene from Camp Lejeune more likely than not was a substantial contributing factor to the causation of his mantle cell lymphoma.

Did I read that correctly?

A. You did.

Q. I'd appreciate if you could put that to the side and please look at page 11 of Exhibit 3. That's your specific causation initial report for Mr. Kidd.

Are you looking at Exhibit 3, Dr. Hu?

A. Oh, wait a minute. Oh, we're going to the -- sorry. Here we go. 11, you said?

Q. Yes, sir.

All right. So, again, we are on Exhibit 3, your initial specific causation report for Mr. Kidd. Page 11 above the asterisk line that says: Thus given my general causation assessment and the factors

reviewed above, it is my opinion to a reasonable degree of medical certainty that Mr. Kidd's combination of exposures to TCE, PCE, and benzene from Camp Lejeune more likely than not was a substantial contributing factor to the causation of his non-Hodgkin's lymphoma.

Did I read that correctly?

A. Yes.

Q. Dr. Hu, what is a substantial contributing factor?

A. A factor that played a significant role in the multi-step process that is typically associated with the carcinogenic process, initiation promotion, and eventual manifestation of cancer.

Q. Dr. Hu, can you define substantial contributing factor without using the term in your definition?

A. Substantial to me means meaningful. It is clear. It is something that, if it had not been present would have led to less likelihood of a cancer being caused or caused at that time.

Q. So in your view, if something is a

substantial contributing factor, that means but for that factor, their disease wouldn't have occurred?

ATTORNEY GREENWALD: Objection. Form. That mischaracterizes his testimony.

THE WITNESS: But for that factor, it is less likely it would have occurred or would have occurred at a later time period.

ATTORNEY ANTONUCCI: Counsel, I ask you please limit your objections to form and foundation.

BY ATTORNEY ANTONUCCI:

Q. In your view, if X is a substantial contributing factor to Y, did X cause Y?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: It is -- I can't -- I can't express it more accurately or precisely than substantial contributing factor. I don't consider cancer in most circumstances as something that only has one single factor; so when you say is that

equivalent, substantial contributing factor means it is the cause of cancer. I consider that a false comparison because I don't consider most cancers as a single-cause disease.

BY ATTORNEY ANTONUCCI:

Q. If something is a substantial contributing factor to cancer, is it fair to say that it is a cause of that cancer?

A. I think that's more or less similar. I haven't really thought of whether it's completely equivalent. I think that gets into semantics, which I feel is not productive.

Q. Can there be more than one substantial contributing factor to a given outcome?

A. Yes.

Q. So in your reports -- and I believe you still have the Mr. Kidd specific causation initial report in front of you.

That's Exhibit 3; is that correct?

A. Yes.

Q. You wrote that: The combination of

exposures to TCE, PCE, and benzene from Camp Lejeune more likely than not was a substantial contributing factor to the causation of his non-Hodgkin's lymphoma.

Is that correct?

A. Correct.

Q. What do you mean when you say that: Exposure to these contaminants is more likely than not a substantial contributing factor to non-Hodgkin's lymphoma?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, we just discussed at length what I mean by substantial contributing factor. The combination of exposures, what I mean by that is his history of being exposed to drinking water that was contaminated at times by all three of these carcinogens; trichloroethylene, perchloroethylene, and benzene.

BY ATTORNEY ANTONUCCI:

Q. I apologize if my question wasn't clear. I was referring to the combination of the terms "more likely than not" and

"substantial contributing factor."

In your opinion, does that change the meaning of any of the terms?

ATTORNEY GREENWALD: Objection. Form, asked and answered.

THE WITNESS: No.

ATTORNEY ANTONUCCI: I am marking for identification Exhibit 12.

(Exhibit Number 12 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. So, Dr. Hu, I handed you Exhibit 12. That is the document with Bates range CL_PLG-expert_HU_204 through 215; is that right?

A. Yes.

Q. Could you please turn to the last page with Bates number ending in 215.

A. Okay.

Q. This is an invoice dated May 1, 2025; is that correct?

A. Yes.

Q. And this invoice is for services rendered between March 1 and April 30, 2025; correct?

A. Correct.

Q. The fourth bullet point on this invoice states: Review of expert reports by Madigan, Savitz, Felsher, Bailey, Ambinder; Nix versus Chemours and DuPont; Yates vs Ford Motor Corp; Wang, et al., 2023 UK Biobank Study; Yu, et al., 2025 pre-print UK Biobank study, and review of transcripts of Mallon and Gondek depositions, 9.5 hours; is that correct?

A. Yes.

Q. So, Dr. Hu, is it fair to say that you reviewed the materials listed here for the first time after March 1, 2025?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, it's the first time I billed for it. I couldn't say for absolute sure that it only occurred after March 1.

BY ATTORNEY ANTONUCCI:

Q. Okay. Do you recall whether you reviewed the Wang, et al., 2023 UK Biobank Study prior to issuing your general causation report?

A. I did not, if I recall correctly.
Q. Did you review the Wang, et al., 2025 UK Biobank Study prior to issuing your supplemental general causation report?
A. I did not, as I recall.
Q. Did you review the Yu, et al., 2025 UK Biobank Study prior to issuing your general causation report?
A. 2025 did you say?
Q. Yes, sir.
Had you reviewed the Yu, et al., 2025 UK Biobank Study prior to issuing your general causation report?
A. No.
Q. Had you reviewed the Yu, et al., 2025 UK Biobank Study pre-print prior to issuing your general causation report?
A. No.
Q. Had you reviewed the Yu, et al., 2025 UK Biobank Study prior to issuing your supplemental general causation report?
A. Not that I recall.
Q. Dr. Hu, how did you become aware of the Wang, et al., and Yu, et al., studies?
A. I believe counsel pointed out that

it was -- well, was it -- as I sit here today, Counsel, I can't remember whether plaintiffs' counsel pointed out the existence of these studies or whether I came across it through my own search means. I just simply can't recall.

Q. Okay. So also on page 215 of Exhibit 12, you note that you reviewed Nix versus Chemours and DuPont; is that right?

A. Correct.

Q. What is Nix versus Chemours and DuPont?

A. It's a litigation case that involved consideration of general causation, and I can't remember, as I sit here today, what the specific chemicals were involved. I recall just skimming it and not reading it in terrible detail.

Q. Okay. So when you say that Nix versus Chemours and DuPont was a litigation case, do you mean that it's an opinion from a judge?

A. Oh, gosh. Honestly, I don't even remember.

Q. Okay. Do you recall why you

reviewed Nix versus Chemours and DuPont?

ATTORNEY GREENWALD: Objection to form.

THE WITNESS: I recall the plaintiffs' attorney suggested that might be a case that would be --

ATTORNEY GREENWALD: I just want to remind you not to talk about our conversations, Dr. Hu.

THE WITNESS: Okay.

ATTORNEY GREENWALD: That's privileged.

THE WITNESS: A case that might be pertinent. That's all.

BY ATTORNEY ANTONUCCI:

Q. Did you consider Nix versus Chemours and DuPont in rendering your opinions in this case?

A. No.

Q. Did you modify your opinions in any way to be consistent with the ruling in Nix versus Chemours and DuPont?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No.

BY ATTORNEY ANTONUCCI:

Q. Okay. Dr. Hu, what is Yates versus Ford Motor Corp?

A. Same response actually. Something related to litigation and some kind of exposure.

Q. Did you consider Yates versus Ford Motor Corp in rendering your opinions in this case?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No.

BY ATTORNEY ANTONUCCI:

Q. Did you modify your opinions in any way to be consistent with the ruling in Yates versus Ford Motor Corp?

ATTORNEY GREENWALD: Same objection.

THE WITNESS: No.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, I'll represent to you that Nix versus Chemours and DuPont and Yates versus Ford Motor Corp are not on your materials considered list.

Is that because you did not

consider them in rendering your opinions in this case?

A. Correct.

ATTORNEY GREENWALD: Objection. Asked and answered.

THE WITNESS: Sorry. Correct.

BY ATTORNEY ANTONUCCI:

Q. Okay. Dr. Hu, you authored reports on the specific causation of the non-Hodgkin's lymphoma of Mr. Kidd and Mr. Carter, and those are Exhibits 3 and 4; correct?

A. Correct.

Q. If I refer to non-Hodgkin's lymphoma during this deposition as NHL, will you understand what I mean?

A. Yes.

Q. Non-Hodgkin's lymphoma, or NHL, arises from a mutation in the DNA of a lymphocyte; correct?

A. Correct. Well, hold on a second. It arises from a mutation in a stem cell that typically results in -- is projected to become a lymphocyte.

Q. Okay. If a mutation had occurred

in a stem cell that was projected to become a leukocyte, the resulting cancer would not be NHL; correct?

A. Correct.

Q. Lymphocytes are a type of white blood cell; correct?

A. Yes.

Q. So non-Hodgkin's lymphoma is a group of blood cancers; correct?

A. It's a group of cancers that arise from cells that typically become constituents of blood.

Q. It is a group of cancers and not one single type; correct?

A. Correct.

ATTORNEY GREENWALD: Objection. Form.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, there are over 60 different subtypes of NHL; correct?

A. My recollection is over 40, but I have no reason to disagree with you.

Q. Okay. To your knowledge, Mr. Carter was diagnosed with diffuse large B-cell lymphoma; correct?

A. Correct.
Q. Diffuse large B-cell lymphoma is a subtype of NHL; correct?
A. Correct.
Q. If I refer to diffuse large B-cell lymphoma as DLBCL, will you understand what I mean?
A. Yes.
Q. DLBCL is the most common subtype of NHL; correct?
A. Correct.
Q. DLBCL accounts for about a third of NHLs; correct?
A. I don't recall what the latest proportion is under -- using the Seer data, but that would be the source of data that I would use to make that estimate.
Q. Mr. Carter was diagnosed with mantle cell lymphoma; right?
A. Correct.
Q. Mantle cell lymphoma is a type of B-cell lymphoma; isn't it?
A. Type of what?
Q. B-cell lymphoma.
A. Yes.

Q. And mantle cell lymphoma is a subtype of NHL; correct?
A. Correct.
Q. And if I refer to mantle cell lymphoma as MACL, will you understand what I mean?
A. Yes.
Q. MACL is a rare subtype of NHL; correct?
A. More or less.
Q. MACL accounts for about 5 percent of all NHLs; correct?
A. More or less, correct.
Q. Are you familiar with the term "hematopoietic malignancies"?
A. Yes.
Q. The term "hematopoietic malignancies" encompasses blood cancers; correct?
A. Correct.
Q. The term "hematopoietic malignancies" encompasses NHL; correct?
A. I'd agree with that.
ATTORNEY ANTONUCCI: Okay. I'd like to take a five-minute break.

THE VIDEOGRAPHER: We are off the record at 10:15 a.m., and this concludes the end of media Unit Number 1. We are now going off the record.

(Recess taken from 10:15 a.m. to 10:37 a.m.)

THE VIDEOGRAPHER: We are now back on the record. This is the beginning of media Unit Number 2, and the time is 10:37 a.m.

BY ATTORNEY ANTONUCCI:

Q. Okay, Dr. Hu, we are back on the record.

You understand that you're still under oath; correct?

A. Correct.

Q. Did you discuss the substance of your testimony with counsel during the break?

A. Not at all.

Q. Okay. Dr. Hu, I'd like for you to turn to Exhibit 1, please. That is your Phase 2 general causation report.

A. Thank you.

Q. And if you could, please flip to page 9 of Exhibit 1.

A. Okay.

Q. Under subheading 2, Non-Hodgkin's Lymphoma, do you see where I am on the page?

A. Yes.

Q. That reads: Non-Hodgkin's lymphoma NHL is a type of cancer that starts in lymphocyte cells which are part of the body's immune system.

Did I read that correctly?

A. You did.

Q. So, Dr. Hu, you agree that NHL subtypes are cancers of lymphocytes?

A. Yes.

Q. Dr. Hu, you agree that multiple myeloma is a cancer of a plasma cell; correct?

A. Yes.

Q. And you agree that plasma cells are not a type of lymphocyte, correct?

A. I agree with that.

Q. Dr. Hu, you agree that lymphocytes are not a type of plasma cell; correct?

A. Correct.

Q. Dr. Hu, you never mention multiple myeloma in any of your reports; correct?
A. I did not.
Q. And you're not offering opinions about multiple myeloma in this case; is that correct?
A. Correct.
Q. Dr. Hu, did I understand you correctly that you said all NHL subtypes arise from mutations in stem cells that will become lymphocytes?
A. I don't think I meant to say all, but I meant to say that they can start in mutations in the pluripotential stem cells.
Q. What is the basis of that opinion?
A. It's based on my understanding of current cancer biology as it relates to hematopoietic cancers as well as some of the writings that have appeared in peer-reviewed journals by my colleague, Dr. Bernard Goldstein, who's a hematologist and environmental health expert and who has studied in depth the subject of environmental carcinogenesis and hematopoietic cancers. I quoted some of his

work in one of my reports.

Q. Are you referring to the study Benzene As a Cause of Lymphoproliferative Disorders by Bernard D. Goldstein, published in Chemico-Biological Interactions in 2010?

A. That's one of them, yes.

Q. Are there any others?

A. I believe I quoted another one, which was a chapter in an IARC publication that stands to -- I'm sorry, International Agency For Research on Cancer. That was published in 2019.

Q. Other than the IARC chapter and Benzene As a Cause of Lymphoproliferative Disorders By Dr. Goldstein, are there any other peer-reviewed sources that support your opinion?

A. Well, none that I quoted in my reports and that I'm in a position to quote today.

Q. Okay. Dr. Hu, is it your understanding that mutations associated with NHL subtypes do not occur after stem cells begin differentiating into different types of cells?

A. My opinion is that they can occur at each stage of a cell's differentiation process leading up to its differentiation until the specific cell type of cancer that becomes manifest.

Q. In reaching your conclusions, you performed a literature review on the topic of associations between PCE, TCE, benzene, and NHL; is that correct?

A. Correct.

Q. And you did that for your general causation and specific causation conclusion; is that correct?

A. Yes.

Q. In analyzing the epidemiologic and toxicologic literature on an association, a literature is a key step.

Do you agree?

A. Yes.

Q. And do you agree that a search should be crafted to produce both positive and negative results?

A. Yes.

Q. Otherwise, you risk forming an unbalanced opinion; correct?

A. I would say that's correct.
Q. What databases did you use to perform your literature search?
A. Mostly PubMed.
Q. Other than PubMed, did you use any others?
A. You know, I would use Google Scholar particularly to see if there's any so-called gray literature that's not published in the peer-reviewed literature. It includes things like monographs and books.
Q. Other than PubMed and Google Scholar, did you use any other research database for your literature review?
A. No.
Q. For your specific causation conclusions, what were your search terms?
A. Well, my specific causation reports were based first on my general causation reports in which I used the searches I just explained to you. And then the rest of it was mostly dependent on the specifics of the plaintiff themselves, and sometimes I would review the literature further if there are

nuances of their particular case that needed further explanation.

Q. Okay. So for your general causation opinions, what were your search terms?

A. Search terms? I mean, I can remember some, but I know that I can't remember all of them. You know, certainly cancer, neoplasm, using the medical subject headings for each of the cancers and for each of the exposures of interest, and then once I located specific references of interest, I would look at the PubMed list of similar articles.

I would look at the bibliographic entries of each of the references to see if there were other references that might be useful. So it's sort of like a tree. It may start with PubMed, but then as you pick up different references in PubMed, or you look in the bibliographies of the papers themselves, create other leads to follow, and those are the strategies I employed.

Q. You just mentioned that you used the medical subject heading for each cancer

as your search terms.

Did I understand that correctly?

A. Yes.

Q. What is a medical subject heading?

A. These are specific bibliographic tools that the National Library of Medicine uses so that when you say something -- here's an example.

A lead is a toxicant that I've studied a lot. If you just put "lead" in a search, then you'll get everything that relates to lead. But if you put lead and then you put MESH, which is the medical subject heading in brackets, then it understands what you mean is the lead toxicant.

And that's an example of how the medical subject heading allows you to be more specific in your search even though, you know, a particular word may have multiple meanings.

Q. Did you use the medical subject heading for NHL in your search?

A. I think I did. I can't specifically recall, as I sit here today,

but I think I did.

Q. Did you use the medical subject heading for DLBCL in your search?

A. I don't remember.

Q. Do you remember if you used the medical subject heading for MACL in your search?

A. I don't remember.

Q. Are your search terms included in your reports?

A. Not that I recall, no.

Q. Why not?

A. That's just a detail that I didn't feel was absolutely necessary.

Q. Do you believe that it's necessary for scientific research to be reproducible?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, of course. And, you know, I was not in the mode of producing a systematic review for publication. As I actually explained in one of my reports, to do a systematic review formally requires a very intensive process. It's

typically done by a team of researchers, and it takes a long time. So that was not my charge.

My charge was to do the best scientific review that I could. And I also relied quite a bit, as my reports reveal, on peer-reviewed published, systematic reviews and systematic reviews and meta-analyses that appeared in the literature.

BY ATTORNEY ANTONUCCI:

Q. Is it your opinion that systematic reviews are a high quality piece of evidence that can be used in determining causation of a disease?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, they're a methodology that helps take a -- if used properly, helps take a broad systematic and rigorous review of the literature, and if they're joined with a meta-analysis, it is additionally enhanced by a quantitative approach towards determining the relationship

between an exposure of interest and outcome of interest.

BY ATTORNEY ANTONUCCI:

Q. So in Phase 2, general causation of this litigation, you provided a report which reviewed the associations between TCE, PCE, benzene, and NHL; is that correct?

A. Yes.

Q. Did you offer any opinions on associations between vinyl chloride and NHL?

A. No.

Q. Did you offer any opinions on associations between DCE and NHL?

A. DCE?

Q. 1,2 trans trichloroethylene.

A. Yeah, no.

Q. So to be clear, you did not offer any opinions on the associations between 1,2 trans trichloroethylene and NHL?

ATTORNEY GREENWALD: Objection. Form, asked and answered.

THE WITNESS: I did not.

BY ATTORNEY ANTONUCCI:

Q. Are you aware of any consensus scientific organizations like IARC that

agree with you that TCE, PCE, and benzene more likely than not can cause NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I don't believe that they've considered that subject recently, and I don't -- I think as I wrote in my report, there was a subgroup of their working group that felt the evidence was sufficient for one of the causal connections that you mentioned.

I think it was benzene and NHL, but it wasn't the entire working group.

BY ATTORNEY ANTONUCCI:

Q. Are you aware of any consensus scientific organizations like IARC that agree with you that exposure to TCE more likely than not can cause DLBCL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Not that I recall.

BY ATTORNEY ANTONUCCI:

Q. Are you aware of any consensus

scientific organizations like IARC that agree with you that PCE more likely than not can cause DLBCL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Not that I'm aware of.

BY ATTORNEY ANTONUCCI:

Q. Are you aware of any consensus organizations like IARC that agree with you that benzene more likely than not can cause DLBCL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Not that I'm aware.

BY ATTORNEY ANTONUCCI:

Q. Are you aware of any consensus scientific organizations like IARC that agree with you that TCE, PCE, or benzene more likely than not can cause MACL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Not that I'm aware. I'm not sure it's ever been considered.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, in your specific causation reports for Mr. Kidd and Mr. Carter, you considered the latency periods or the length of time between the plaintiffs' first exposure to contamination at Camp Lejeune and when they developed NHL; correct?

A. Yes.

Q. And as part of your efforts in reaching your opinions, you compared the latency periods of Mr. Kidd and Mr. Carter to those from published studies; is that correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Correct.

BY ATTORNEY ANTONUCCI:

Q. Okay. For Mr. Carter, you cited the study Evidence of Long Latency Periods Prior to Development of Mantle Cell Lymphoma by Racke, et al., published in 2010; is that correct?

A. Forgive me. I'll have to consult my report to jog my memory.

Q. Sure. So it might help if you look

at page 13 of Exhibit 4.

A. I'm there. Okay. Can you repeat your question, please?

Q. You cited Racke, et al., 2010 Evidence of Long Latency Periods Prior to Development of Mantle Cell Lymphoma in your report for Mr. Carter; is that correct?

A. I cited Racke, et al, and also Olsson and Brandt.

ATTORNEY ANTONUCCI: Okay. I am marking for identification Exhibit Number 13.

(Exhibit Number 13 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. I will represent to you that Exhibit 13 is the Racke, et al., 2010 study.

A. Yes.

Q. Okay. So if you look at the second page of Exhibit 13, Dr. Hu --

A. Yes.

Q. -- the last sentence of the only paragraph on this page says: Taken together, the data presented strongly suggests that a long latency period

following the initiating IGH/CCN D1 translocation may occur in MCL with patients harboring in situ lesions for years prior to the development of clinical disease.

Did I read that correctly?

A. Yes.

Q. And to make this finding, Racke, et al., identified seven patients that were diagnosed with mantle cell lymphoma and who had previous pathological material available that contained lymphoid tissue, and that was unrelated to the subsequent mantle cell lymphoma; is that right?

A. Correct.

Q. All seven specimens that Racke, et al., studied showed evidence of mantle cell lymphoma, and the oldest specimen was taken 15.5 years prior to the diagnosis of mantle cell lymphoma; correct?

A. I think what you said is the pathologic specimens revealed mantle cell lymphoma, and that's not what the investigators said. In situ mantle cell lymphoma, which is basically a preliminary

stage prior to actual manifestation of the disease.

Q. Okay. I am reading from the sentence starting with the word "pathological." That's also on page 2 of Exhibit 13, Racke, et al.

A. I see it.

Q. Pathological specimens range from 2.1 to 15.5 years prior to the diagnosis of MCL. In all seven specimens, cyclin D1 positive collections of lymphocytes were identified, five with distinct homing to mantle zones, one with follicular colonization, and one with a diffuse distribution.

Did I read that correctly?

A. You did.

Q. Racke, et al., there is saying that the oldest specimen was taken 15.5 years prior to the diagnosis of mantle cell lymphoma; correct?

A. Correct.

Q. And that all seven specimens showed evidence of in situ 2 mantle cell lymphoma; correct?

A. Correct.
Q. Do you know if any of the study participants in Racke, et al., were exposed to any toxicants?
A. I don't think that was ever considered.
Q. Do you know if the latency periods discussed in Racke, et al., is at all related to their exposure to toxicants?
A. There's no information about that.
Q. Do you know whether or not Mr. Carter had evidence of in situ mantle cell lymphoma before 2010?
A. He's never had a lymph node biopsy, as far as I know, prior to the manifestation of his disease.
Q. Okay. You can put that to the side. Thanks very much.
So, Dr. Hu, earlier you mentioned you also cited Olsson and Brandt; is that right?
A. Correct.
ATTORNEY ANTONUCCI: I'm going to mark Olsson and Brandt as Exhibit 14.
///

(Exhibit Number 14 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, I just handed you Risk of Non-Hodgkin's Lymphoma Among Men Occupationally Exposed to Organic Solvents by Olsson and Brandt published in 20 -- 1988, excuse me.

Is that correct?

A. Yes.

Q. The Olsson and Brandt study was a case-controlled study; correct?

A. Correct.

Q. Okay. And if you look at page 249 of Exhibit 14, Olsson and Brandt, on the second column, paragraph starting about halfway up the page with the word "there were large."

Do you see that?

A. Yes.

Q. Olsson and Brandt wrote: There were large variations in the length of periods from the start of exposure to the diagnosis of non-Hodgkin's lymphoma 2 to 60 years with a median of 21 years.

Did I read that correctly?

A. You did.

Q. Dr. Hu, how does this study support your conclusion that MACL has a long latency period?

A. Well, this sentence basically says that the latency period, which is the length of period from exposure to diagnosis of the disease, in the cases varied from 2 to 60 years with a median that is the 50th percentile of 21 years.

So that, I believe, supports my contention here that the latency period is long and that it is -- includes the apparent latency period for Mr. Carter.

Q. Thanks, Dr. Hu. You can put that study aside.

You performed a differential etiology for Mr. Carter and Mr. Kidd; is that correct?

A. Correct.

Q. And to do that, you ruled in the relevant risk factors for developing each of the diseases experienced by Mr. Carter and Mr. Kidd; correct?

A. Well, I considered them.
Q. Okay. And you also considered known risk factors for NHL that were not applicable to each plaintiff; correct?
A. That's correct.
Q. And you ruled those out; correct?
A. Well, I considered them.
Q. Okay. Is there a reason that you didn't rule out risk factors that were not applicable to each plaintiff?
A. Well, when I say I considered them, that also includes whether I -- you say rule them out. I say I discount them, and it's a somewhat equivalent term. Rule out really is a bit more absolute, I guess, and I hesitate to use a term like that unless there's absolute certainty that, you know, the factor was not present.
So, for instance, if there's no documentation that someone had been tested for a particular thing, you know, I -- may not necessarily be ruled out, but I discounted it because there was no evidence that they had something else.
Q. In conducting the differential

etiologies for Mr. Kidd and Mr. Carter, did you rule out any risk factors?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: All right. Let's -- you said both Mr. Kidd and Mr. Carter?

BY ATTORNEY ANTONUCCI:

Q. Let's start with Mr. Kidd.

A. Okay.

Q. Did you rule out any risk factors for NHL in performing your differential etiology for Mr. Kidd?

ATTORNEY GREENWALD: Same objection.

THE WITNESS: Let me go to that report.

BY ATTORNEY ANTONUCCI:

Q. Differential etiology is discussed on page 11 of your initial report for Mr. Kidd, and that is Exhibit 3.

A. I'm there.

Q. Okay.

A. Okay. So, first of all, I considered other risk factors as they're articulated by the American Cancer Society.

Risk factors, in those cases, are things that, through epidemiology, have been found to have, in some study or another, a association with -- between the exposure and, in this case, non-Hodgkin's lymphoma. He was white, as it turns out. NHL is more common among whites; so he meets that criteria.

However, he does not have family history of a first-degree relative with NHL. There's no previous treatment for cancer chemotherapy drugs. He did not have evidence of a weakened immune system. He did not have evidence of inherited syndromes associated with immunodeficiency. He did not have evidence of autoimmune disease, previous lymphoma, or chronic infections with Helicobacter pylori, Chlamydophila, Siddiqi, Campylobacter jejuni, or hepatitis C, and no history of breast implants.

So I considered all those things that had been identified as risk factors by the American Cancer Society. There is no evidence of that.

So just to go back to your

terminology, I guess you could say I ruled them out. Although I hesitate to use that terminology because that has a certain amount of absolutism associated.

Is it possible he might have had actually one of these conditions? It just was never tested or never documented? It's possible. But without any evidence, I effectively considered them in my differential diagnosis. Differential etiology. Sorry.

Q. So for Mr. Kidd, for example, you concluded that he had no clear evidence of any known risk factor for DLBCL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, I did discuss the fact that he had some X-rays, which I would say the vast majority of the Americans have X-rays at some point in their life. But I did not see any evidence of an unusually amount of exposure to X-rays that would make him stand out as somebody who -- for whom radiation exposure

would be considered a risk factor for NHL.

And as I also said, you know, there was no -- in my view, none of these risk factors can negate the additional contribution of his exposure to TCE, PCE, and benzene.

BY ATTORNEY ANTONUCCI:

Q. Okay. I'd like to read the last sentence of the paragraph 5 on page 11 of your specific causation report for Mr. Kidd, Exhibit 3. That says: Overall, Mr. Kidd had no clear evidence of any known risk factor for NHL and certainly no risk factor that would negate the contribution of his exposure to TCE, PCE, and benzene as discussed above.

Did I read that correctly?

A. You did.

Q. What does "negate the contribution of his exposure to TCE, PCE, and benzene" mean?

A. It means that this goes back to the discussion earlier in our deposition, which is the prevalent view of carcinogenesis is

that it's a multi-factor disease where, for most cases of cancer, there's multiple causes, if you will, or factors that together initiate and promote and end up causing the manifest cancer.

So in this particular case, unless there was something that was an overwhelming risk factor like some of the hereditary cancer syndromes, the contribution of his exposures which are known to be carcinogenic, and at a fairly high level in my view, make that more likely to be a substantial contributing factor, and without any of these other risk factors, it stands out.

Q. So considering whether or not Mr. Kidd had any clear evidence of a known risk factor allowed you to conclude that his exposure to TCE, PCE, and benzene at Camp Lejeune more likely than not was a substantial contributing factor to the causation of his NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, it's just one

of the factors. I mean, you know, I went through general causation. I went through his whole history. I looked at latency. I looked at the degree of exposure. I looked at all sorts of other factors in my approach to try and understand the specific causation question.

BY ATTORNEY ANTONUCCI:

Q. Okay. Do you employ differential diagnosis as part of your medical practice?

A. Yes.

Q. Do you employ differential etiology as part of your medical practice?

A. I mean, I typically do because I'm a public health physician, and I'm always interested in prevention, but no, I mean, if I'm treating somebody with diabetes, I don't necessarily, especially if they're type 2 diabetes and they've been having it for a long time, I don't necessarily go through a whole differential etiology as part of the workup and the treatment and management.

Q. Okay. I'd like you to turn to page 9 of Exhibit 3. That is your specific

causation initial report for Mr. Kidd.

A. Okay.

Q. Under subheading B, the second paragraph begins with "In reaching conclusions."

Do you see where I am?

A. Yes.

Q. Here you wrote: In reaching conclusions regarding causation, practitioners in the medical and scientific fields in which I specialize must acknowledge and account for certain inherent limitations to understanding the etiology of an individual's cancer. There is nothing specific about cancer, e.g., its clinical presentation or its pathology, when it develops in an individual that definitively indicates or proves its cause. There are no tests that have yet been developed that can definitively identify cause in such a manner. Methods have been developed aimed at quantitatively estimating the contribution to the causation of an individual's disease by an individual's exposure to an associated risk factor;

however, as with most cases of cancer induced by environmental causes, making such precise estimates is not possible in this case.

Did I read that correctly?

A. You did.

Q. Dr. Hu, it's your opinion that there's no test one can administer to determine whether a person's NHL was caused by exposure to PCE; right?

A. Correct.

Q. It's also your opinion that there's no test one can administer to determine whether a person's NHL was caused by exposure to TCE; correct?

A. Correct.

Q. And it's your opinion that there's no test one can administer to determine whether a person's NHL was caused by exposure to benzene; correct?

A. Correct.

Q. It is your opinion that there are no biomarkers for exposure to any of those three chemicals; correct?

A. That's not true.

Q. Okay. I'll have you turn to your general causation report. That's going to be Exhibit 1, page 24.

Under subheading F, you wrote: There are no tools, e.g., biological markers --

A. Wait, wait, wait. You said page 24 of my general causation report?

Q. Yes, sir.

A. And you said -- oh, yeah. Okay. I'm sorry. I'm looking at the wrong subheading.

Q. Subheading F, the sentence that starts "In conclusion."

A. Yeah.

Q. I'm reading from the third line down from that paragraph. It says: There are no tools (e.g., biological markers) with which to assess historical and/or cumulative exposures to TCE.

Did I read that correctly?

A. That's correct. That's historical or cumulative. That's not the same thing as recent or ongoing.

Q. Okay. I'll have you turn to

page 34. That's Exhibit 1. Under subheading H.

A. Yes.

Q. Third line down you wrote: There are no tools (e.g., biological markers) with which to assess historical and/or cumulative exposures to PCE.

Did I read that correctly?

A. That's correct.

Q. All right. And page 40, Exhibit 1, under subheading E. In the fourth line from the beginning of that paragraph you wrote: There are no tools (e.g., biological markers) with which to assess historical and/or cumulative exposures to benzene.

Did I read that correctly?

A. You did.

Q. What tools are available to assess recent exposures to PCE?

A. I don't remember specifically, but typically for volatile organic compounds, you can either measure the compound in blood itself or in urine or its metabolites. But typically these are -- these are chemicals that are not persistent in a person's blood

or urine for more than a certain defined period of time. Sometimes it's hours. Sometimes it's days. Maybe weeks, but certainly not what I would have considered as historical or cumulative exposure.

Q. Okay. And neither Mr. Kidd or Mr. Carter were exposed to water at Camp Lejeune within the past few weeks; is that right?

A. What do you mean by that? Not exposed in the past few weeks. Well, one of them has passed away. What are you talking about?

Q. Could you use, for example, the blood test you were just describing to quantify the exposures of Mr. Kidd or Mr. Carter?

A. Well, certainly if their exposures by history had been years ago, no.

Q. Dr. Hu, it's your opinion that the clinical presentation of NHL does not change depending on whether it was caused by exposure to TCE, PCE, or benzene; correct?

A. As far as I'm aware, you are correct.

Q. Does that change depending on the subtype of NHL that a patient presents with?

A. Can you repeat the original question?

Q. It's your opinion that the clinical presentation of NHL does not change depending on whether the NHL was caused by exposure to TCE, PCE, or benzene; correct?

A. Correct. And then you asked is that also true -- I'm sorry. You're going to have to repeat the question. I got a little bit confused.

Q. Is that also true of NHL subtypes?

A. So certainly within NHL, if you're comparing subtype to subtype, they have different clinical presentations. They may occur at different times of life, in general. They may have different ways of presenting in terms of the symptoms and what are typically done in terms of the workup when you look at the radiological imaging, et cetera.

So I'm not -- I'm not sure if I answered your question, but that's, I think, the answer to what you posed.

Q. Dr. Hu, are you familiar with the NHL subtype MALT lymphoma?
A. Which one?
Q. MALT lymphoma?
A. Is that an acronym, or is that the name?
Q. MALT lymphoma is an acronym for mucosa-assisted lymphoid tissue lymphoma.
Are you familiar with it?
A. No.
Q. Is there any way to tell from the clinical presentation of a person's NHL whether it was caused by exposure to one of PCE, TCE, or benzene?
A. No --
ATTORNEY GREENWALD: Object --
THE WITNESS: Not that I'm aware of.
BY ATTORNEY ANTONUCCI:
Q. And so the only way to determine whether an individual's NHL was caused by exposure to TCE, PCE, or benzene is through toxicological and epidemiological analysis coupled to evidence of exposure?
ATTORNEY GREENWALD: Objection.

Form.

THE WITNESS: Well, in my view, it can only be done by the process that I went through, which is first consider the general causation and then consider all the specific information about the particular patient in terms of their patient history, their family history, their dietary history, their infectious disease history, the clinical presentation of the disease, the data on exposure to the various things that allegedly may have caused their cancer, the length of exposure, the level of exposure, on and on and on.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, you based your opinions in part on Dr. Kelly Reynolds' report of February 6, 2025; is that correct?

A. Yes.

Q. And to the extent that you opined that exposure to contaminated water at Camp Lejeune caused Mr. Kidd and Mr. Carter's cancers, is that opinion based on

Dr. Reynolds' exposure calculations?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I incorporated her calculations in my analysis.

BY ATTORNEY ANTONUCCI:

Q. How did you incorporate Dr. Reynolds' calculations in your analysis?

A. Well, as I expressed in my reports, I read the report and focused in on her summation of the total concentration time product of exposure to each one of those contaminants, and then I used that information to estimate what the average exposure was to each of those contaminants during their time at Camp Lejeune.

Q. Why did you calculate average exposure during the time the plaintiff spent at Camp Lejeune?

A. Because that allowed me to consider the overall degree of exposure in the process of doing the evaluation.

Q. Does the cumulative microgram per liter month calculation Dr. Reynolds did not allow you to consider the overall degree of

exposure?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I mean, that's another metric that may have some value. But it is not, you know, typically referable or comparable to the known or the kinds of measurements that are used to express risk.

BY ATTORNEY ANTONUCCI:

Q. Okay. So rather than use the cumulative microgram per liter month calculations, you added the microgram per liter months of exposure for each month that a plaintiff was exposed, then divided by the number of months in which a plaintiff was exposed?

A. Correct.

Q. Okay. And that is how you arrived at your average exposure during a plaintiffs' time at Camp Lejeune; correct?

A. That's correct.

Q. Okay. So it's true that your calculation of time-weighted average for each plaintiffs' exposure is based on

Dr. Reynolds' exposure calculation; correct?
A. Yes.
Q. Do you agree that your opinions on Mr. Kidd's and Mr. Carter's exposures caused by their NHL are, therefore, only as good as Dr. Reynolds' numbers?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: Well, as I also discussed in my report, I compared the experience of Mr. Carter with some of the scenarios that appeared in the ATSDR report, and one of the scenarios in particular which I reproduced in my specific causation report, was very similar to the exposure scenario for Mr. Carter.
I also found one that was very similar to the exposure scenario for Mr. Kidd, and ATSDR went through the process of taking these illustrative exposure scenarios and then calculating the lifetime cancer risk by age group based on the years of exposure, whether they worked on base

or lived off base, et cetera.

And, like I said, I found these scenarios that are very similar to each of these plaintiffs. And those cancer estimates were also part of my basis for my conclusions.

BY ATTORNEY ANTONUCCI:

Q. Did you afford equal weight to the time-weighted average for each plaintiffs' exposure that you calculated using Dr. Reynolds' numbers and ATSDR's exposure scenarios?

A. I mean, they both -- I found both of them useful and compelling. I'm not sure I would try to sort of weigh one more than the other. They were both contributory.

Q. Okay. Did you validate or test Dr. Reynolds' exposure calculations?

A. Well, I looked at her report, looked at her methodology, felt that was compatible with what I consider as the proper approach to exposure assessment, but I did not redo her calculations.

Q. Do you know how Dr. Reynolds determined when and where Mr. Carter was at

Camp Lejeune?

A. Well, I remember from her reports that she used multiple sources of history, history taking, recordkeeping, et cetera, to come up with those parameters.

Q. Do you know if Dr. Reynolds used deposition testimony to determine where and when Mr. Carter was at Camp Lejeune?

A. I'd have to go back and look at the records to see -- to answer that truthfully.

Q. Are you aware that Mr. Carter's widow, Mrs. Frances Carter, did not know precisely when and where he worked on the base during her deposition?

A. I have no knowledge of that.

Q. Are you aware that Mr. Carter's employment records do not state where he worked at the base throughout his career?

A. I have no knowledge of that.

ATTORNEY ANTONUCCI: I'm going to introduce Dr. Reynolds' report. This is going to be Exhibit 15, I believe. Yes, 15.

(Exhibit Number 15 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. And this version of the report has the written portion of her report and the appendices for Mr. Kidd and Mr. Carter. I've omitted the appendices for the other 23 plaintiffs.

A. Great.

ATTORNEY GREENWALD: What number was this? I'm sorry.

ATTORNEY ANTONUCCI: 15.

ATTORNEY GREENWALD: Thank you.

BY ATTORNEY ANTONUCCI:

Q. Okay. Dr. Hu, if you could please turn to Exhibit 15, appendix 19, page 1. You should be looking at the summed variable totals for Mr. Kidd.

A. Well, page 1 of -- oh, which appendix?

Q. Appendix 19.

A. 19. Okay.

ATTORNEY GREENWALD: You don't mind if I find this; right? Okay. Let me just grab it from you. I found it; so I'll get it for you.

THE WITNESS: There it is.

ATTORNEY GREENWALD: It's right here.

THE WITNESS: All right. Thanks.

BY ATTORNEY ANTONUCCI:

Q. Okay. For the record, we are looking at Exhibit 15, appendix 19, page 1. This shows summed variable totals for plaintiff Robert Kidd; is that correct?

A. Yes.

Q. So in this table, Dr. Reynolds provided several different summed exposure totals; correct?

A. Yes.

Q. The first column on the far left lists the contaminants of concern in this case; TCE, PCE, vinyl chloride, and benzene; right?

A. Yes.

Q. The next column over shows the cumulative microgram per liter month calculation; correct?

A. Yes.

Q. And Dr. Reynolds calculated this value by adding mean monthly concentrations of contaminants in drinking water for each

month the plaintiff was exposed; correct?
A. Yes.
Q. Have you ever seen an exposure assessment presented in terms of cumulative microgram per liter month before?
A. Microgram per liter month. I probably have. I've seen a lot of exposure assessments, but I can't remember which ones or when.
Q. Okay. Now, if you look at the next column over, it's labeled Chart 1: 1L, for one liter.
Do you see that?
A. Yes.
Q. And this shows cumulative monthly total contamination exposure for each of the volatile organic compounds at issue in this case; right?
A. Yes.
Q. And this was calculated by multiplying the number of exposure days by 1 liter per day by the summed monthly average concentrations; is that right?
A. Yes.
Q. Okay. And chart 2, which is

labeled ATSDR, shows the same data as chart 1, but instead of 1 liter, Dr. Reynolds used ATSDR's ingestion estimates of 6 liters per day three days per week and 3 liters per day three days per week; correct?

A. I don't remember the assumptions, and they're not stated on the table, but I have no reason to disagree with you.

Q. Okay. And chart 3 shows the same data as chart 1, but instead of 1 liter, Dr. Reynolds used deposition-informed activity ratios; is that correct?

A. Something that referred to the deposition, yes.

Q. Okay. And chart 4 shows the same data as chart 1, except instead of 1 liter, Dr. Reynolds combined deposition-informed activity ratios with averaged ingestion volumes from four military field manuals; correct?

A. I don't see the field materials and the description of them listed on the table, but like I said before, I don't have any reason to disagree with you.

Q. Okay. Of all of the five columns

that show some variable totals here, you only use the cumulative microgram per liter month in your reports; is that correct?

A. That's correct.

Q. Okay. Why didn't you use any of the other summed variable totals in your reports?

A. I felt that was the best estimate that was given by Dr. Reynolds.

Q. You felt that the cumulative microgram per liter month was the best estimate given by Dr. Reynolds?

ATTORNEY GREENWALD: Objection. Form, asked and answered.

THE WITNESS: Yes.

BY ATTORNEY ANTONUCCI:

Q. Why is it that cumulative microgram per liter month is the best estimate?

A. I mean, I think that was what she put forth as, you know, her best estimates. You know, I don't have any other rationale for focusing on that, and I rely on her judgment as to what are -- the best estimates might be.

Q. And I apologize if I already asked

this, but you modified cumulative microgram per liter months as presented in Reynolds' report to calculate a time-weighted exposure average for each plaintiff's time spent on base; right?

A. Correct.

Q. But you also testified that Dr. Reynolds' calculations and methodology is reasonable; correct?

A. Yes.

Q. If it was reasonable, why didn't you use her calculations as presented?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I did use them as the ones that showed up on the following page.

BY ATTORNEY ANTONUCCI:

Q. Okay. So you're referring to the -- I'm sorry.

What are you referring to, Dr. Hu?

A. Okay. These are double-sided pages. Hold on a second. This.

ATTORNEY ANTONUCCI: Okay. So for the record, Dr. Hu is indicating to

the chart table Finished Water Concentration Micrograms Per Liter Kidd Model Cumulative in Exhibit 15. It is also labeled page 1.

THE WITNESS: Yeah. It's a little confusing.

BY ATTORNEY ANTONUCCI:

Q. Okay. When you say you used these values, which values are you referring to, Dr. Hu?

A. The ones at the very bottom.

Q. Okay. So this is the total microgram per liter month for each contaminant; correct?

A. That's correct.

Q. Okay. And for the purposes of your report, you divided that by the number of months each plaintiff was on base; correct?

A. That's correct.

Q. And you did that because it allowed you to compare them to known measurements.

Am I correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: To concentrations

that have appeared in, for instance, the ATSDR CREG and the EPA maximum contaminant list.

BY ATTORNEY ANTONUCCI:

Q. Okay. So it follows that Dr. Reynolds' cumulative microgram per liter months cannot be compared to ATSDR's CREGs without dividing them by the number of months the plaintiff was on base; right?

A. That's correct. Yes.

Q. Dr. Reynolds based her calculations of each patient's cumulative exposure on the data in Mr. Maslia's report.

Is that your understanding?

A. I'd have to go back and look at her report to see if -- I mean, she clearly used that report, but whether she used other things as well, I don't recall.

Q. So you agree that, at least in part, Dr. Reynolds based her calculations on the data presented in Mr. Maslia's report; correct?

A. Yes.

Q. Hypothetically, if Mr. Maslia's data were incorrect, would Dr. Reynolds'

calculations be impacted?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, if I recall, she also referred to the ATSDR's work, but I don't recall how much that depended on Dr. Maslia's reports. I just don't recall the whole choreography of exposure assessments and what was dependent on what.

BY ATTORNEY ANTONUCCI:

Q. So, Dr. Hu, all else being equal, if Dr. Reynolds' cumulative exposure numbers decreased by 15 percent, would that impact your opinions on the causation of Mr. Kidd's NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: 15 percent? Probably not.

BY ATTORNEY ANTONUCCI:

Q. Okay. If Dr. Reynolds' cumulative exposure numbers decreased by a factor of 15 percent, would that impact your opinions on the causation of Mr. Carter's NHL?

A. Probably not.
Q. How much would Dr. Reynolds' cumulative exposure numbers have to decrease to impact your opinions on the causation of the plaintiffs' NHL?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: I don't know. I haven't considered that, and I'm not able to give you an opinion on that right now.
BY ATTORNEY ANTONUCCI:
Q. Would it be more than 50 percent?
A. I don't know.
ATTORNEY GREENWALD: Objection. Form.
BY ATTORNEY ANTONUCCI:
Q. So to be clear, you haven't considered whether or not -- if Dr. Reynolds' numbers were too high or too low, if that would impact your final conclusions; is that correct?
ATTORNEY GREENWALD: Objection to form.
THE WITNESS: That's not an

exercise I performed.
BY ATTORNEY ANTONUCCI:
Q. Okay. And you testified that 15 percent would be too low to change your opinions but that you weren't aware whether 50 percent would be too high; is that right?
ATTORNEY GREENWALD: Objection to form. Asked and answered.
THE WITNESS: That's what I said, and that's, you know, again, off the top of my head, but I hesitate to say anything with any more certitude given that I haven't really considered the question.
BY ATTORNEY ANTONUCCI:
Q. Okay. Dr. Reynolds did not perform a risk assessment for either plaintiff, did she?
A. Not that I'm aware.
Q. She did not characterize the plaintiffs' risk from exposure to contamination; correct?
A. That's another way of saying the same thing. I'm not aware of it.
Q. And she didn't calculate their

excess lifetime cancer risk based on their exposure to contamination?

A. Not that I'm aware of.

Q. Dr. Reynolds didn't calculate Mr. Kidd's or Mr. Carter's margins for exposure, did she?

A. Define margins of exposure.

Q. Dr. Reynolds didn't calculate the ratio calculated by determining a level of exposure in which harm to human health is not expected to occur than dividing by an estimated level of human exposure for Mr. Kidd or Mr. Carter; correct?

A. Correct.

Q. Dr. Reynolds did not consider exposure information from animal or epidemiology studies in her report; correct?

A. Correct.

Q. Rather she just quantified exposure; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: More or less, yes.

BY ATTORNEY ANTONUCCI:

Q. And you did not perform a risk

assessment either; correct?
A. Correct.
Q. And you didn't use Dr. Reynolds' exposure calculations to calculate lifetime cancer risk; correct?
A. I did not.
Q. You didn't calculate a margin of exposure for Mr. Kidd or for Mr. Carter; correct?
A. Correct.
Q. You relied instead on ATSDR's 2017 Public Health Assessment lifetime cancer risk estimates; is that correct?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: I used that, yes.
BY ATTORNEY ANTONUCCI:
Q. Okay. I'd like you to turn to Exhibit 4. That's the initial specific causation report for Mr. Carter.
A. Okay.
Q. And if you could, please go to page 9.
A. All right.
Q. Under subheading 3, Exposure

Assessment, Risk of Cancer, Ronald Carter, the end of the first paragraph you wrote: Accounting for Mr. Carter's workweek, vacations, et cetera, Dr. Reynolds estimated Mr. Carter had a total exposure time of 6,865 days, which is equivalent to 229 months. One can consequently calculate Mr. Carter's time-weighted average exposure levels of PCE, TCE, and benzene by totaling his microgram per liter months of exposure to each chemical from Hadnot Point, Tarawa Terra, and Midway Park and dividing by 229 months. This yields time-weighted average exposure levels of 34.5 micrograms per liter or parts per billion of PCE, 113 micrograms per liter or parts per billion of TCE, and 1.7 micrograms per liter or parts per billion of benzene.

Did I read that correctly?

A. You did.

Q. So in order to calculate the time-weighted average in micrograms per liter, you divided for TCE 2,000 -- 25,877 by 229; is that correct?

A. Say that one more time.

Q. I'll break that down a little bit.
Mr. Carter's total exposure was calculated as being 229 months; correct?
A. Yes.
Q. And his cumulative micrograms per liter month of exposure to TCE as calculated by Dr. Reynolds was 25,877 micrograms per liter; correct?
You may need to refer to the appendices of Dr. Reynolds' report to find that out.
A. Of TCE?
Q. Correct.
A. I have 25,603 in my paragraph. That's a little bit different than the figure you just quoted.
Q. That is for the Hadnot Point Water Treatment Plant; correct?
A. Yeah.
Q. And for the Tarawa Terrace Water Treatment Plant for TCE, it was 225 micrograms per liter month; correct?
A. Yes.
Q. For PCE, Dr. Reynolds calculated -- strike that. I'm going to move on.

You compared the time-weighted exposure levels that you calculated with ATSDR's CREG and EPA's MCL; correct?

A. Correct.

Q. And for both plaintiffs, you noted that the time-weighted exposure levels you calculated exceeded the CREGs for those contaminants; correct?

A. That's one of the comments I made.

Q. So, for example -- I believe we're in the Carter report. So that's Exhibit 4, page 7, note 17.

A. Page 4 of the specific causation report on Carter?

Q. Page 7 of Exhibit 4.

A. Okay.

Q. If you look at note 17 of Exhibit 4, you wrote that: CREG cancer risk evaluation guide concentrations represent concentrations of cancer-causing substances unlikely to result in an increase of cancer risk in an exposed population above a target risk level of one excess cancer per 1 million exposed people. They are derived by ATSDR using USEPA cancer slope factors,

default exposure assumptions (regarding ingestion and body weights).

Did I read that correctly?

A. Yes.

Q. CREG values are estimated contaminant concentrations unlikely to result in more than one excess cancer in a million persons during their lifetime; correct?

A. Right.

Q. That's over an average of 78 years; correct?

A. Okay. I think it's 70, not 78.

Q. Okay. So it's over an average of 70 years --

A. Yes.

Q. -- is that right?

CREG values are developed for drinking water to identify a concentration at the tab representing one in a million or 1 times 10 to the negative 6 risk; is that correct?

A. Yes.

Q. And that's based on EPA's oral cancer slope factor; right?

A. Yes.
Q. And that's when consumed by a 70-kilogram human drinking 2 liters of such water per day for a 70-year lifetime; correct?
A. Is it 70 or 75? I don't remember the exact parameters, but I have no reason to disagree with you, Counsel.
Q. And you're aware that an MCL, or maximum contaminant level, is the highest level of a contaminant that's allowed in drinking water as determined by the EPA; correct?
A. Yes.
Q. EPA takes health risks into consideration when setting MCLs; is that right?
A. Correct.
Q. They also take available water treatment technology into account when setting MCLs; correct?
A. They also take into account what?
Q. When setting MCLs, EPA also takes into account available water treatment technology.

A. I don't know the answer to that.
Q. When setting MCLs, EPA takes into account costs; is that right?
A. I knew the answer to that at one time, but I don't now. I'm not sure, Counsel.
Q. And MCL is not the same thing as a reference dose; correct?
A. Correct.
Q. And then MCL is not the same thing as a threshold dose; correct?
A. Correct.
Q. And I believe you wrote in your rebuttal reports that comparing the CREG and the MCL to ATSDR's -- excuse me. Strike that.
You wrote in your rebuttal reports that comparisons to time-weighted averages of exposures to micrograms per liter to MCLs and CREGs was not a methodology for quantitating risk; is that right?
A. Right.
Q. So why did you make those comparisons?
A. It's simply to give a sense of the

degree of exposure of the individual.
Q. Do you agree that an exposure exceeding the CREG provides basis only to conclude that there's an increased risk of more than one in a million for any type of cancer?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: I would agree with that.
BY ATTORNEY ANTONUCCI:
Q. And do you agree that exceeding the MCL is not a basis for any conclusion about the risk of an individual's cancer?
A. I would agree that it's not useful for that purpose.
Q. Okay. When you calculated the time weighted cumulative microgram per liter calculations, did you take into account Mr. Kidd or Mr. Carter's body weight?
A. No, because I think in terms of risk, it doesn't matter whether he's 150 kilograms or only 50 kilograms if he's drinking water at a certain concentration. He's being exposed relatively to the same

amount. Presumably someone who's 150 kilograms is drinking a lot more water than someone who's 50 kilograms.

So what really matters is the dose that is referent to the individual, but that's not going to change based on the concentration of a particular contaminate in water.

Q. So the reason that you didn't take into account their body weight is because you assumed that they consumed more water if they weigh more?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No, no, no. It's just that I think it's -- it's not -- you know, his body weight is not relevant to the question of whether drinking water with a pollutant at a certain level is a risk for cancer.

BY ATTORNEY ANTONUCCI:

Q. Do you consider body weight when dosing your patients' medications?

ATTORNEY GREENWALD: Objection to form.

THE WITNESS: Absolutely, but that's when you're trying to calculate a dose, not a concentration. A dose. A dose is the concentration and the weight of the pill or the weight of the solution that you're giving; right.

So you're trying to obtain the same level of a medicine, in your example, in blood, and that -- that's where the dose matters. And you have to calculate the weight of the person in trying to determine how much medication to give.

But in this case, we're talking about a contaminant in water where everybody titrates their own dose because they're drinking whatever water their body needs. So it's a bit of comparing apples to oranges. Like I said, to understand the contaminant level in water is sufficient to understand the risk.

BY ATTORNEY ANTONUCCI:

Q. Okay. So you don't need

information about the dose of contamination each plaintiff was exposed to in order to render the opinions you're rendering?

A. No.

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No, no, no. You're drawing an incorrect conclusion from what I just said.

She calculated -- I'm sorry. I used her data to calculate the average concentration in water of what he was drinking. That's not a dose. That's just the concentration. Dose would require taking the information and then multiplying it, like you said, by body weight, this, that, and the other. But that's irrelevant in this particular case because he's drinking that, and the dose is whatever he takes in to drink.

And the dose presumably will be very similar to a 150-kilogram individual dose in terms of body weight versus someone else who's only

50 kilograms drinking the same concentration of the pollutant.

BY ATTORNEY ANTONUCCI:

Q. So please help me understand.

A. Sure.

Q. Where in your opinion does dose come into account?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Dose presumably was taken into account when the ATSDR made these lifetime projections. Okay? That's what I relied on to give some sort of quantitative sense of what his risk for cancer was from drinking this.

Now, if you'd like to dissect that, we should go back to the ATSDR methodology and go exactly over how they went over their calculation of lifetime risk.

BY ATTORNEY ANTONUCCI:

Q. Based on your understanding, did Dr. Reynolds quantify dose?

A. Dr. Reynolds simply quantified the

level of contaminant that was being consumed by each individual and the length of time in particular periods of time. That's it.

Q. Why is it that you used concentration per month, meaning micrograms per liter months, rather than total mass consumed or microgram months?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Rather than total micrograms. Wait. Say that again. You confused me.

BY ATTORNEY ANTONUCCI:

Q. Sure.

In your report, you considered the microgram per liter month calculation, which is a concentration; correct? Why didn't you look at the total mass of exposure micrograms per month?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: So I'm not sure I'm following exactly what you're trying to get at. But my methodology was simply aimed at trying to estimate the

likely overall concentration of pollutant in water that was consumed by the individual during their time at Camp Lejeune, period.

And that was a way of allowing me to compare his experience with those that were projected by the ATSDR in their life and their excess cancer estimates.

ATTORNEY ANTONUCCI: Okay. I'm going to mark for identification Exhibit 16.

For the record, this is ATSDR's Public Health Assessment for Camp Lejeune Drinking Water, U.S. Marine Corp base, Camp Lejeune, North Carolina, January 20, 2017, and this has the Bates range CLJA_health effects-11 through 212.

(Exhibit Number 16 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, ATSDR calculated three-year rolling average concentrations of contaminants in drinking water for this

report; is that right?
A. I'm not sure, as I sit here, but I have no reason to disagree with that.
Q. Okay. So on page 7 and 8 of your initial report on Mr. Carter, you wrote that: ATSDR calculated three-year rolling average concentrations of contaminants in drinking water.
Would you like me to find that and point it out for you?
A. No. I see it at the top of page 8.
Q. Right.
So do you agree that's what they did?
A. Yes.
Q. And this was based on ATSDR's historical reconstruction and carried out by Mr. Maslia; is that right?
A. That's correct.
Q. Do you know how ATSDR calculated that three-year rolling average?
A. I don't know the specifics, no.
Q. How did ATSDR perform the exposure assessment for the public health assessment?
A. I read at one point that the --

their description methodology, but I don't sit here -- as I sit here today, I don't recall the specific methodology.

Q. Did you have any opinions on ATSDR's exposure assessment for the public health assessment of 2017 as you read it?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: All I can say is that I had no reason to disagree with their methodology.

BY ATTORNEY ANTONUCCI:

Q. Okay. So on -- and, Dr. Hu, you can put that aside for the moment. I'm sorry.

A. I'm just curious to see something in it that relates to what I extracted from it, but please go ahead and question me whatever you need.

Q. So on your initial report for Mr. Kidd and, again, that's Exhibit 3 on page 8 --

A. We're back to Kidd?

Q. Yes, sir.

A. Okay. Page 8.

Q. Correct.
A. All right. Hold on. Okay.
Q. So this is Exhibit 3, page 8. You wrote that: Mr. Kidd's status as a Marine living at Camp Lejeune and the timing of his exposure profile very closely aligns with the exposure profile (discussed in 2E above) of a Marine who trained and lived on base for three years exposed to drinking contaminants from the Hadnot Point WTP (i.e., PCE, TCE, and benzene) that ATSDR estimated was associated with a lifetime cancer risk beginning in the early 1970s of over 1 per 10,000 until around 1983 with a peak of 3.5 per 10,000 for exposures surrounding 1981.

Is that right?

A. Correct.
Q. And then just to clarify, one of the acronyms you wrote, the Hadnot Point WTP.

What does that mean?

A. Water treatment plant.
Q. And then you wrote something very similar in your report for Mr. Carter.

That's Exhibit 4 on page 10.

Here, you wrote: In addition, I note that Mr. Carter's status as a civilian worker at Camp Lejeune and the timing of his exposure profile very closely aligns with and, in fact, exceeds the exposure profile (discussed in 2G above) of 15 years of exposure to workers on base who lived off base to all chemical contaminants from Hadnot Point (i.e., PCE, TCE, and benzene) that ATSDR estimated was associated with a lifetime cancer risk of over 1 per 10,000 for exposures between the mid-1960s to around 1982 with a peak of 2.6 per 10,000 for exposures surrounding 1970. Added to that risk was his exposure to Tarawa Terrace WTP from June 1, 1980, through December 1, 1987, which is mostly within the interval of time (1950s to mid-1980s) in which ATSDR estimated the lifetime cancer risk by age group over time based on 15 years of exposure to workers on base who lived off base to all chemical contaminants of Tarawa Terrace (i.e., PCE, TCE, and vinyl chloride) to be over 1 per 1 million and in the range

of 1 per 100,000.

Is that right?

A. That's what I wrote.

Q. What is your basis for assuming Mr. Carter had 15 years of exposure at Hadnot Point?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: So as I wrote in the previous page in the report by Dr. Reynolds, she noted that Mr. Carter was exposed to drinking water from the Hadnot Water Treatment Plant from February 1, 1968 through May 30, 1980. So that's 12 years, not quite 15 years, but it's close.

BY ATTORNEY ANTONUCCI:

Q. Okay. When writing your report, did you use the cumulative 15-year exposure period for your calculations and analyses?

A. Well, I mean, like I said, I -- for quantitative risk, I relied on the ATSDR extrapolation. So once I found that they had done an extrapolation that involved an individual very similar to Mr. Carter, I

found that compelling.

ATTORNEY ANTONUCCI: Okay. With that, I believe we need to take a break for the record -- excuse me, for the tape.

THE VIDEOGRAPHER: This is the end of media Unit Number 2. We are off the record at 12:11 p.m.

(Recess taken from 12:11 p.m. to 1:10 p.m.)

THE VIDEOGRAPHER: We are back on the record. This is the beginning of media Unit Number 3, and the time is 1:10 p.m.

BY ATTORNEY ANTONUCCI:

Q. Okay, Dr. Hu, we are back on the record, and you are still under oath.

Do you understand?

A. I do.

Q. Did you discuss the substance of your testimony with counsel during the break?

A. No.

Q. Dr. Hu, for your specific causation reports in this case, did you rely on

Dr. Reynolds' determination of the months each plaintiff spent on base?

A. Yes.

Q. Did you conduct separate exposure calculations or analysis based on the different jobs Mr. Carter worked at Marine Corps base Camp Lejeune?

A. I did not do that by myself, no.

Q. Did anyone else conduct separate exposure calculations or analysis based on Mr. Carter's different jobs at Camp Lejeune?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Not that I know.

BY ATTORNEY ANTONUCCI:

Q. Which parts of the base did Mr. Carter work on during his time at Camp Lejeune?

A. Let me refresh my memory by looking at my report. Carter. We're on Carter?

Q. Yes, sir.

Again, for the record, the question was: Which parts of the base did Mr. Carter work on during his time at Camp Lejeune?

ATTORNEY GREENWALD: And I still

object. I'm sorry. That was the last question.

THE WITNESS: Hadnot Point and -- or at least the part of the base that was supplied by Hadnot Point's water supply and then Tarawa Terrace.

BY ATTORNEY ANTONUCCI:

Q. Other than Hadnot Point and Tarawa Terrace, do you know if Mr. Carter ever worked anywhere else on Marine Corps base Camp Lejeune during his time there?

A. Not that I discussed in my own report, but I'd have to go back to Dr. Reynolds' report to see if there's anything else.

Q. Did you consider time spent on other parts of the base in reaching your opinion?

A. Not that I recall. Oh, sorry. I have a note here about time at Midway Park.

Q. Okay. Do you know where on Marine Corps base Camp Lejeune Midway Park is?

A. Geographically?

Q. Do you know whether or not Midway Park is serviced by Hadnot Point, Holcomb

Boulevard, Tarawa Terrace water treatment plants?

A. It has its own water treatment plant.

Q. Are you aware of whether or not the Midway Park -- excuse me, Midway Point Water Treatment Plant was ever contaminated?

A. Well, according to my notes here, Dr. Reynolds' calculated exposure to PCE and TCE that occurred from exposure to drinking water from the Midway Park Water Treatment Plant.

Q. Okay. Do you know whether or not Mr. Carter ever spent time at Holcomb Boulevard?

A. I don't recall that.

Q. And as we previously discussed, you compared the plaintiffs' exposure scenarios to those from ATSDR's 2017 public health assessment; is that correct?

A. Correct.

Q. And those resulted in excess lifetime cancer risks that you compared for each plaintiff; correct?

A. Yes.

Q. And then estimated excess lifetime cancer risk is the estimated number of increased cases of cancer in a population above background that might result from exposure to a particular contaminant; correct?

A. Correct.

Q. And ATSDR's 2017 PHA calculated excess lifetime cancer risk for multiple types of cancer; right?

A. I do recall that, yes.

Q. So ATSDR's 2017 PHA did not only calculate estimated excess lifetime cancer risk for NHL; correct?

A. Correct.

Q. If you'll turn to Exhibit 16, page 32, that's ATSDR's 2017 public health assessment.

A. Okay.

Q. About -- under the heading Calculation of Cancer Risk underneath the formulas there, there's a paragraph that starts with "To apply the best."

A. Yes. I see it.

Q. The second sentence of that

paragraph reads: TCE exposure is associated with kidney cancer, liver cancer, and lymphoma; however, the experimental evidence indicates that the mutagenic mode of action only applies to the kidney (USEPA 2011b).

Did I read that correctly?

A. You did.

Q. ATSDR's PCE and benzene cancer toxicity criteria were also based on liver and leukemia endpoints respectively; right?

A. Say that again.

Q. ATSDR's PCE cancer toxicity criteria was based on liver cancer endpoints; correct?

A. I don't specifically recall, but if you could point me to the right section of this report, I could check on that.

Q. Do you recall whether ATSDR's benzene cancer toxicity criteria were based on leukemia endpoints?

A. I believe that's true. And that would make sense, but I would have to check the relevant part of the report to confirm that.

Q. Do you know whether liver cancer

has a more sensitive cancer endpoint than NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I don't specifically recall, sitting here today, whether that's true.

BY ATTORNEY ANTONUCCI:

Q. Do you know whether leukemia has a more sensitive cancer endpoint than NHL?

ATTORNEY GREENWALD: Same objection.

THE WITNESS: I don't specifically recall that, as I sit here today.

BY ATTORNEY ANTONUCCI:

Q. ATSDR's 2017 public health assessment was a population-based study; correct?

A. I mean, it was not an epidemiology study, per se. It was an assessment of the overall situation at Camp Lejeune as it pertains to exposure to toxics in drinking water and risk for cancer.

Q. ATSDR's 2017 public health assessment used the 1 times 10 to the

negative 4 to 1 times 10 to the negative 6 target for cancer risk, didn't it?

A. Target for cancer risk. If you could define that, I could try to answer that. And if you could show me where in the document they discuss that, that would be helpful.

Q. If you look at page 35 of Exhibit 16, that's the page with Bates number ending in 67.

A. 30 --

Q. 35.

A. Okay.

Q. There's a figure -- this page of Exhibit 16, page 35, features Figure 9.

There's multiple estimated lifetime cancer risks by age group here; is that right?

A. Yes.

Q. And you can see in the key for this graphic that ATSDR shaded in gray the cancer risk range of 10 to the negative 4 for 1 in 10,000 to 10 to the negative 61 in 1 million?

A. Yes.

Q. I'd appreciate if you could please turn to page 20 of Exhibit 16.

A. Okay.

Q. So the first bullet point up from the bottom starting with "The Marines who trained."

Do you see where I'm reading from?

A. Where it says tour of duty data? First bullet up from the bottom.

Q. I'm sorry. So from there, it's two bullet points down, "The Marines who lived."

A. Oh, yes. Okay.

Q. So, again, page 20 of Exhibit 16 states: The "Marines who trained and lived on base" group includes those servicemen and women who regularly engaged in field exercises. If a person lived on base and either worked on base or was the spouse of an active duty marine but did not regularly engage in field exercises, then that person would be considered an adult who resided on base.

Did I read that correctly?

A. You did.

Q. Did you review the transcript from

the deposition of Mr. Kidd in forming the opinions in your report?

A. I might have skimmed it, but I don't even recall any details.

Q. Do you recall that Mr. Kidd testified under oath about his field exercises?

A. I don't recall that at all.

Q. So you don't know what Mr. Kidd said about his field exercise activity; is that right?

A. I do not.

ATTORNEY GREENWALD: Objection. Form.

BY ATTORNEY ANTONUCCI:

Q. According to the PHA that Mr. Kidd had testified that he didn't regularly engage in field exercises, he might more accurately be described as an adult who resided on base; is that right?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: According to this statement, that's how ATSDR would classify him.

BY ATTORNEY ANTONUCCI:
Q. And do you know what threshold ATSDR established to determine whether a Marine regularly engaged in field exercises?
A. I do not.
Q. How would you delineate whether a Marine regularly engaged in field exercises?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: I have no opinion on that.
BY ATTORNEY ANTONUCCI:
Q. I'd like you to turn your attention now to Exhibit 5, please. Again, Exhibit 5 is the rebuttal to the report of Dr. Richard F. Ambinder for Robert Arnold Kidd that was disclosed on May 16, 2025.
A. Okay.
Q. If you could, please turn to page 2 of Exhibit 5.
A. Okay.
Q. The first paragraph on page 2 reads in part: In short, I agree with Dr. Ambinder that it is an oversimplification that all lymphomas or all

NHLs share the same causation for all possible carcinogens given the possibility that some carcinogens only exert their effects at post-differentiation lymphocytic line level; however, given that the evidence for benzene specifically supports that it produces multiple chromosomal changes at the pluripotential level, genotoxic effects in circulating lymphocytes, and in a substantial body of rigorously conducted epidemiological studies is associated with NHL, in my opinion, it is reasonable to conclude that benzene can cause each of the NHL subtypes, all of which stem from the same pluripotential subtype.

Did I read that correctly?

A. You did.

Q. So is it your opinion that benzene exposure is associated with every subtype of NHL?

A. It's my opinion that it's capable of causing any of the subtypes of NHL.

Q. Is it your opinion that TCE exposure is capable of causing any of the subtypes of NHL?

A. I haven't specifically addressed that. Certainly there is evidence that TCE causes genotoxicity, and if I recall, some chromosomal damage as well, but I haven't actually given that much thought.

Q. Are you offering the opinion that TCE exposure is capable of causing every subtype of NHL?

ATTORNEY GREENWALD: Objection. Form, asked and answered.

THE WITNESS: I don't have an opinion on that.

BY ATTORNEY ANTONUCCI:

Q. Are you offering the opinion that PCE exposure is capable of causing every subtype of NHL?

A. I don't have an opinion on that.

Q. Is the underlying genetic mutation associated with all NHL subtypes the same?

A. I don't think that's known. And I doubt it.

Q. Why do you doubt that?

A. Because my understanding of carcinogenesis is that there's multiple avenues of genetic damage that can all

contribute towards the causation of the actual cancer.

Q. Based on your understanding, would different genetic mutations cause different NHL subtypes?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: It's possible but not necessarily required.

BY ATTORNEY ANTONUCCI:

Q. Do lymphomas always rise at the same stage of lymphocyte maturation?

A. Not that I'm aware of.

Q. You noted on page 9 of Exhibit 5 -- excuse me --

A. There is no page 9.

Q. That's correct. Page 9 of Exhibit 3. That's your specific causation report for Mr. Kidd.

A. Okay.

Q. On page 9 of your specific causation report for Mr. Kidd, you wrote that: With regards to subtype, Mr. Kidd's NHL was classified as a diffuse large B-cell lymphoma, which is the most common type of

NHL, accounting for about a third of all NHLs. Thus, I consider my opinions regarding TCE, PCE, and benzene in relation to NHL as being directly applicable to the diffuse large B-cell lymphoma experienced by Mr. Kidd.

Did I read that correctly?

A. Yes.

Q. And it's your opinion that because DLBCL is the most common subtype of NHL, your opinions about the causation of NHL generally are directly applicable to DLBCL in particular; is that right?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Yes.

BY ATTORNEY ANTONUCCI:

Q. Okay. I'm sorry to ask you to jump around, but I'd like you to look at Exhibit 4. That's your specific causation report for Mr. Carter. Page 10.

A. Did you say page 2?

Q. I'm sorry. Page 10 of Exhibit 4.

A. Okay. All right.

Q. On page 10 of Exhibit 4, you state

that: Mantle cell lymphoma is one of the subtypes of B-cell non-Hodgkin's lymphomas. As such, I consider the opinion I expressed regarding the causal relationships between TCE, PCE, benzene, and NHL to be relevant to mantle cell lymphoma.

Did I read that correctly?

A. You did.

Q. And so it's your opinion that the causes of NHL generally are also relevant to mantle cell lymphoma in particular; is that correct?

A. Yes.

Q. Is there a reason why you said that the opinions about the causes of NHL are directly applicable for DLBCL and only relevant for mantle cell lymphoma?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I see those as equivalent statements, frankly. I don't really -- I could have used one word or the other.

BY ATTORNEY ANTONUCCI:

Q. Okay. So you noted that benzene in

particular has a substantial body of rigorously conducted epidemiological studies showing an association with NHL; is that correct?

A. I did review that, yes.

Q. And because of that, in your opinion, it's reasonable to conclude that benzene can cause each of the NHL subtypes, which all stem from the same pluripotential subtype; right?

A. Yes.

Q. Do TCE -- does TCE have the same substantial body of rigorously conducted epidemiological studies that shows it's associated with NHL?

A. There is evidence, if you're asking me to sort of compare the body of evidence connecting benzene with NHL versus the body of evidence connecting TCE and NHL. I reviewed them independently. I did not compare them to see which one was better or greater or larger than the other; so I can't really answer your question today.

Q. Okay. Is it reasonable to conclude that TCE can cause each of the NHL subtypes

based on the body of literature connecting TCE exposure to NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I mean, the reason I hesitate is because there's so many different subtypes of NHL that when you say every subtype, I have to kind of look at what I know about the literature. I said that for benzene because clearly its impact on the pluripotential stage is clear. I'm not sure the evidence base for TCE and PCE is quite the same.

On the other hand, the epidemiology is there for NHL and it's substantial, which is why I talked about it at some length in my general causation report.

So just getting back to your question, I'm not sure I have an opinion on that, whether TCE can cause any subtype of NHL.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, are you aware that IARC

concluded there was limited evidence that benzene is carcinogenic based on a causative association between exposure to benzene and non-Hodgkin's lymphoma?

A. I am aware that's the conclusion they made based on the working group meeting and report that was issued. I forget what year, but it was some years ago.

Q. Are you aware of any evidence that supports the proposition that PCE produces multiple chromosomal changes at the pluripotential level?

A. Allow me to reacquaint myself with my own general causation report. Hold on.

Well, as I sit here today, I don't see evidence in my report that addresses the issue of whether PCA causes genetic damage at the pluripotential level, and I don't recall coming across scientific evidence of that. That doesn't mean it doesn't exist. I just don't recall if I came across it.

Q. Are you aware of any evidence that supports the proposition that PCE produces genotoxic effects in circulating lymphocytes?

A. PCE; right?
Q. Yes.
Are you aware of any evidence that supports the proposition that PCE produces genotoxic effects in circulating lymphocytes?
A. So on page 32 of my general causation report, I discussed how PCE was shown to be genotoxic to peripheral blood lymphocytes in vitro, causing chromosomal aberrations and the formation of micronuclei in peripheral blood lymphocytes in vitro, citing a study by Kocaman, et al., in 2021.
Q. Okay. Are you aware of any evidence that supports the proposition that TCE produces multiple chromosomal changes at the pluripotential level?
A. I did not comment on that topic in my report, and I don't recall studies documenting TCE's impact on the pluripotential cells of bone marrow. So I'm not aware of such evidence, as I sit here today, but there might be. I just didn't comment on it.
Q. As you sit here today, are you

aware of any literature or study that has found a specific NHL subtype has a pluripotential stem cell mutation associated with benzene?

A. A specific subtype that has a pluripotential cell -- no, I don't think the subject has been studied. You would have to have some kind of bone marrow evidence of genotoxicity in an individual who then, many years later, developed that specific subtype, and I don't think -- I'm not aware of any molecular epi study or case series or case study that had the potential to even address that question.

Q. Dr. Hu, I believe you previously testified that some NHL subtypes are associated with mutations that arise after the pluripotential stem cells begin differentiating; is that right?

A. I think that's possible, and I don't remember enough of the biology of NHL and the science behind it to actually quote such studies or give you more specific information on that.

Q. If it's the case that some NHL

subjects are associated with mutations arising after the pluripotential stem cells begin differentiating, how is it possible that exposure to benzene can cause all NHL subtypes?

ATTORNEY GREENWALD: Objection to form.

THE WITNESS: Well, just because benzene might cause a mutation in the post-pluripotential stage doesn't mean it also -- it can't also cause mutations in the pluripotential stage. There's no reason to expect that the benzene's impact on hematopoesis is limited to one stage of the cell differentiation process, as far as I know.

BY ATTORNEY ANTONUCCI:

Q. Is there any evidence that benzene's impact on hematopoietic is relevant to more than just the pluripotential stage of the cell differentiation process?

A. I can't answer that because I haven't specifically looked into that

question. But my colleague Dr. Goldstein, as I mentioned, has written and opined on that subject of benzene's ability to cause damage at an early stage, and I believe at any stage.

I'd be happy to review the primary source articles of what I quoted in my specific causation report to go over that scientific -- those scientific reports.

Q. Do you know of any evidence that TCE is associated with changes in any genetic loci associated with a NHL subtype?

A. I think that's a level of specificity on TCE's effect that I don't recall taking note of when I reviewed the literature on TCE and cancer.

Q. Did you go into that level of specificity for PCE?

A. No.

Q. Did you go into that level of specificity for benzene?

A. I might have but not in relation to this litigation.

Q. Okay. So in this litigation, you're not offering the opinion that benzene

exposure is associated with changes in genetic loci associated with an NHL subtype?

ATTORNEY GREENWALD: Objection. Form.

BY ATTORNEY ANTONUCCI:

Q. Correct?

A. Well, benzene's clearly associate -- benzene is clearly associated with all sorts of genetic damage of types that are well known to result in hematopoietic cancers. Whether it's specific for NHL, I'd have to review my understanding of the literature to understand that well.

Dr. Goldstein, as I already opined, had specifically addressed benzene's ability to cause lymphoproliferative disorders because its -- its action at the very early pluripotential stage. And, of course, the ability of a pluripotential sought to differentiate both into -- to both arms of cancer whether it's lymphoproliferative or hematological.

Q. Which changes in genetic loci is exposure to benzene associated?

A. Let me refresh what I wrote in my report.

Well, I'm sorry, Counselor. I do recall that benzene is associated with a number of different genotoxic effects. Rather than try to recall what specifically they are, I would -- I don't see that. I've discussed them at length in many of my reports.

If you'd like me to essentially reference the evidence for that, I'd have to go see one of the primary sources that I quoted.

Q. Shifting gears slightly, in both of your specific causation reports for Mr. Carter and Mr. Kidd, you cited the Bove, et al., 2024 study titled Cancer Incidence Among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at U.S. Marine Corp Base Camp Lejeune, A Cohort Study; is that right?

A. Correct.

Q. And in the -- strike that.

If I refer to that study as Bove

2024, will you understand what I'm talking about?

A. Yes. Although there is also a Bove 2024 mortality study; so I just want to make sure that we're just going to concentrate on the incidence study.

Q. You're right. Excuse me.

If I refer to that as the Bove cancer incident study, will you understand what I mean?

A. Yes.

Q. Thank you for that. I appreciate it.

So in the Bove 2024 cancer incidence study, Dr. Bove evaluated the incidence of NHL in general; correct?

A. Correct.

Q. I'm going to hand you that study. I'm marking it for identification as Exhibit 17.

(Exhibit Number 17 was marked for identification.)

THE WITNESS: This is like the pile system I use on my desk at home.

BY ATTORNEY ANTONUCCI:

Q. Okay. Dr. Hu, I just handed you Exhibit 17. That's Bove 2024 cancer incidence study. I'd like you to turn to Table 3. Let me know when you get there.

A. Okay.

Q. So in Table 3 of Exhibit 17, the Bove 2024 cancer incidence study, Bove, et al., found that the Camp Lejeune Marines and Navy personnel had an elevated adjusted hazard ratio for NHL; is that right?

If you're having difficulty finding it, Dr. Hu, it's under L for lymphoid cancers.

A. I'm just trying to comport this with what I wrote.

ATTORNEY ANTONUCCI: For the record, the witness is reviewing Exhibit 2, his general causation supplement of January 31, 2025.

THE WITNESS: So to be specific, in my report, I focused in on the elevated adjusted hazard ratios for mantle cell and marginal zone B-cell lymphomas in the Camp Lejeune Marines and Navy personnel and also on NHL

among the civilian workers.
BY ATTORNEY ANTONUCCI:
Q. Okay. I'm not sure I'm following. My question was just whether Table 3 of this study found that there was an elevated adjusted hazard ratio for NHL.
A. Well, for NHL overall, the adjusted hazard ratio was 1.01. So I would not consider that elevated.
Q. Okay. The 95 percent confidence interval is between 0.9 and 1.14 for that adjusted hazard ratio; correct?
A. Correct.
Q. And there's a confidence interval ratio of 1.3; correct?
A. Correct.
Q. And as you just alluded to, Bove, et al., in the cancer incidence study evaluated non-Hodgkin's lymphoma as a group and also the subtypes of non-Hodgkin's lymphoma; correct?
A. Correct.
Q. So the value for NHL, in general, is the overall adjusted hazard ratio and confidence intervals for all NHLs treated as

a group; correct?
A. Correct.
Q. So, for example, Burkitt's lymphoma. I believe that's the highest adjusted hazard ratio for NHL subtype that's listed in Table 3 with the adjusted hazard ratio of 1.53 and a confidence level between .71 and 3.30; correct?
A. Correct.
Q. And the lowest adjusted hazard ratio was for diffuse large B-cell lymphoma with an adjusted hazard ratio of 0.89 and a confidence interval between 0.72 and 1.10; correct?
A. Correct.
Q. So between diffuse large B-cell and Burkitt's non-Hodgkin's lymphoma, one was found to have a positive association to drinking water at Camp Lejeune, and the other was found to have a negative association; correct?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: Not negative association but rather no association.

BY ATTORNEY ANTONUCCI:

Q. Are you referring to diffuse large B-cell lymphoma?

A. Yeah. Well, I mean, it's a little bit less than .9. I guess you could say negative, but okay. That's fine.

Q. I'm sorry. Do you agree that 0.89 -- an adjusted hazard ratio of 0.89 is a negative association?

A. Yeah, it's below 1 for sure.

Q. Okay. So do you have any opinion as to why the different subtypes of NHL had different adjusted hazard ratios?

A. Well, I mean, this is a cohort follow-up study of men who were exposed to relatively low levels; so the power of the study to even see a signal is relatively limited. Plus the length of the follow-up is relatively short since these are relatively -- still relatively young men.

I think I discussed this in my report, whereas most non-Hodgkin's lymphomas, including the various subtypes, don't arise until later ages. So, you know, I think this study was performed because it

was commissioned. It was important to take a first-look at this.

I would expect and hope that they would continue following these individuals, but I think this is an initial set of evidence in which to try to discern whether there's some elevated rates of cancer.

Q. So, Dr. Hu, in your opinion, is it inappropriate to use Bove 2024's cancer incidence study to evaluate whether or not a plaintiffs' NHL was caused by exposure to water at Camp Lejeune?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I don't think it's inappropriate. It just has to be interpreted in the context of all the other evidence that we've been discussing today.

BY ATTORNEY ANTONUCCI:

Q. I'd like you to turn to Table 4 of Exhibit 17. This table is for a comparison of cancer outcomes among Camp Lejeune and Camp Pendleton civilian workers employed on either base between October '72 and December

of '85; correct?
A. Correct.
Q. And for civilian workers for all NHLs as a group, Bove, et al., 2024 calculated an adjusted hazard ratio of 1.19; correct?
A. Correct.
Q. And that has a confidence interval ranging from 0.83 to 1.71; correct?
A. Correct.
Q. And for civilian workers with marginal zone B-cell lymphoma, this study, Exhibit 17, calculated an adjusted hazard ratio of 0.33 with a confidence interval of 0.06 to 1.72; correct?
A. Yes, based on two cases.
Q. And why did you elaborate that that's based on two cases?
A. Because the numbers are so small, both among the Lejeune and Pendleton workers, that, you know, these are pretty small numbers with which to take quantitative assessments of risk very seriously. They're just relatively unstable.

Q. Okay. For diffuse large B-cell here in Table 4, there were 27 participants at Lejeune, and 20 participants at Pendleton; right?

A. Correct.

Q. Do you think that there were sufficient participants for this metric to be taken seriously in determining causation of DLBCL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, it's certainly moving into the range where you would have more precision, and I think that the elevated hazard ratio is a signal. It's certainly not definitive, but it's certainly consistent with the notion that the Camp Lejeune exposures may be a risk factor for diffuse large cell -- I'm sorry, diffuse large B-cell cancer.

BY ATTORNEY ANTONUCCI:

Q. How many participants would a study like this need to have for you to say definitively that the association indicates

a causal relationship?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, it depends on a whole number of factors. If you're just focusing on diffuse large B-cell, then you would have to do a formal sample size calculation based on what is known about the baseline incidence rate, what the expected incidence rate would be in order to see an elevated cancer risk of whatever quantitative risk you want to pose and then do the sample size calculation with an alpha of .05 and a beta of 80 percent power to figure out just how many people would be necessary.

BY ATTORNEY ANTONUCCI:

Q. Understood.

So I'd appreciate it if you could turn to Table 5.

A. Okay.

Q. And this is Table 5 in Exhibit 17, Bove 2024 cancer incidence study. And here Bove, et al., calculated cancer outcomes by

duration for Marines and Navy personnel stationed at Camp Lejeune compared with Camp Pendleton; correct?

A. Correct.

Q. Looking first at non-Hodgkin's lymphoma treated as a group, we see that for the low duration at Camp Lejeune, the adjusted hazard ratio is 1.02 with confidence interval ranging between 0.89 and 1.17; is that correct?

A. Yes.

Q. For the immediate duration at Camp Lejeune, the adjusted hazard ratio is 1.01 with a confidence level between 0.83 and 0.24; correct?

A. Correct.

Q. And for the high duration at Camp Lejeune, the adjusted hazard ratio is 1.00 with a confidence interval between 0.79 and 1.26; correct?

A. Correct.

Q. This study, therefore, does not support the conclusion that there's a dose response relationship between exposure to chemicals of concern at Camp Lejeune and NHL

as a group; correct?

A. I agree with that.

Q. If there is an association between exposure to water at Camp Lejeune and NHL, why did this study not show a dose response relationship?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Again, as I discussed beforehand, this study is laboring under the limitation of having not enough years of follow up to have a cohort in the age range where most of these cancers can be expected to arise even among people who have had no exposures at all.

So it's -- you know, that's a limitation of this particular study. And for that reason, I don't think one can make an outright conclusion that Camp Lejeune exposures pose no cancer risk at all. I think that time will tell with something like these counts of non-Hodgkin's lymphoma, but given all the other signs that we discussed

today, I feel quite comfortable with my general causation conclusion that these exposures at Camp Lejeune are a significant risk factor for NHL.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, your point about the limitations of the study preventing it to be used to definitively claim no association is well taken.

Do those same limitations prevent you from using this study to definitively prove an association?

A. I would never use this study to definitively prove anything. It's just another piece of evidence that contributes to the whole body of knowledge.

Q. Okay. And then for the sake of completeness, if you look at mantle cell lymphoma on Table 5, Exhibit 17, this also does not show a dose response relationship; correct?

A. Yeah, the number of cancers are really too small to say much. It looks like there's a bump up going from low to medium, and then it goes back down and high. High

only has five cases. So overall, I'd say there's just too few cases to draw any conclusions.

Q. And for diffuse large B-cell lymphoma, can you draw conclusions from Table 5 of Exhibit 17?

A. No.

Q. And why is that?

A. Well, for the reasons that we discussed earlier regarding the limitations to the study, in general.

Q. Keeping in mind the limitations of the study, Table 5 of Exhibit 17 also does not show a dose response relationship between the exposure to the chemicals of concern at Camp Lejeune and diffuse large B-cell lymphoma; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Yeah. I would agree with that. And the last thing I would add, since we're on the subject, is that Camp Lejeune -- these studies are based also on exposure assessments that are done that have what's known

as nondifferential misclassification. That is, they're not expected to have perfect classification in terms of low, medium, and high.

That tends to dilute relationships, true relationships as they may exist, and I'll just leave it at that.

Counsel, can I take a bathroom break? Thank you.

THE VIDEOGRAPHER: We are off the record at 2:08 p.m., and this concludes media Unit Number 3.

(Recess taken from 2:08 p.m. to 2:14 p.m.)

THE VIDEOGRAPHER: We are now back on the record. This is the beginning of media Unit Number 4, and the time is 2:14 p.m.

BY ATTORNEY ANTONUCCI:

Q. Okay. Back on the record.

Dr. Hu, you understand you're still under oath; correct?

A. Yes.

Q. Did you discuss the substance of

your testimony with counsel during the break?

A. No.

Q. I would like you to turn to Exhibit 4, page 12, please. That's your initial report for Mr. Carter.

A. Okay. Let me first put this away. I'm sorry. What page?

Q. Page 12, please.

A. Okay.

Q. So in paragraph 3 on page 12 of Exhibit 4, you state that: Mr. Carter's exposure timing and profile is closely aligned with an exposure scenario that ATSDR estimated to be associated with a lifetime cancer risk of over 1 per 10,000 per -- for exposures between the mid-1960s to around 1982 with a peak of 2.6 per 10,000 for exposures surrounding 1970.

Is that right?

A. Correct.

Q. You go on to say: These magnitudes of risk greatly exceed the risk communication category of negligible risk promulgated by the World Health

Organization, less than 1 in 1 million, and it exceeds the de minimis risk level typically promulgated by the U.S. Environmental Protection Agency for carcinogens of 1 in 1 million.

Did I read that correctly?

A. Yes.

Q. So for the -- I believe footnote 23 on page 12, you cited the World Health Organization, Communicating Radiation Risk in Pediatric Imaging?

A. Correct.

Q. And you cited that for the proposition that the exposure, timing, and profile of Mr. Carter greatly exceeds the risk communication category of negligible risk; correct?

A. Correct.

Q. And are you aware that the source you cited, Communicating Radiation Risks in Pediatric Imaging, states that an approximate level of additional risk of cancer incidence of 1 in 10,000 is very low?

A. You'd have to pull that source for me to look at again, but I have no reason to

disagree with that. I just wanted to make sure that, in this report, I made very clear what was considered a de minimis risk.

ATTORNEY ANTONUCCI: Okay. Well, I'm marking for identification Exhibit 18. This is the World Health Organization's publication Communicating Radiation Risks in Pediatric Imaging, Information to Support Healthcare Discussions About Benefit and Risk.

(Exhibit Number 18 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Please turn to page 25, Exhibit 18. If you look at Table 6, Table 6 is titled Examples of a Qualitative Approach to Communicate Different Levels of Risk of Cancer Incidence Compared with the Lifetime Baseline Risk of Cancer Incidence.

Do you see where I'm reading from?

A. Yes.

Q. And for the approximate level of additional risk of cancer incidence between 1 in 50,000 and 1 in 5,000, the risk

qualification is described as, quote, very low.

Did I read that correctly?

A. Yes.

Q. Okay. And for the discussion of the Environmental Protection Agency's de minimis risk level, you cited in footnotes 24 and 25, page 12 of Exhibit 4 -- excuse me, just footnote 25, you cited Castorina R, Woodruff TJ, Assessment of Potential Risk Levels Associated With U.S. Environmental Protection Agency Reference Values; is that correct?

A. Yes.

Q. And you wrote that: Mr. Carter's magnitude of risk exceeds the de minimis risk level promulgated by the U.S. Environmental Protection Agency for carcinogens of 1 in 1 million; right?

A. Correct.

Q. Dr. Hu, you're already aware that EPA's de minimis risk level is used for policy development regarding the management of cancer risks; right?

ATTORNEY GREENWALD: Objection.

Form.

THE WITNESS: I understand that.

BY ATTORNEY ANTONUCCI:

Q. And you're aware that EPA defines acceptable exposure levels as generally concentration levels that represent an excess upper bound in lifetime cancer risk to an individual of between 1 in 10,000 and 1 in 1 million; right?

A. That -- as far as I'm aware, that's changing goalposts. But certainly at this time when this article was published, that was considered the de minimis risk level in relation to the pollutants and the environmental health issues being discussed by my colleagues Castorina and Woodruff.

Q. Dr. Hu, do you know what percentage of the United States population will develop cancer during their lifetime?

A. Oh, it's up there. It's the second most leading cause of death in the United States after cardiovascular disease. I can't give you the precise figure, but it's high.

Q. I'll represent to you that it's

roughly 40 percent.

How does exceeding a lifetime risk of all cancers increase of between 1 in 10,000 and 1 in 1 million lead to the conclusion of specific causation?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, in this case, it's not even an issue. He developed cancer. So the question really is as it was posed to me: Could his exposures have contributed to that risk of developing cancer? That's the question, not whether he's at risk of getting cancer. He got it.

BY ATTORNEY ANTONUCCI:

Q. Is it your testimony that you did not evaluate the increased risk of contracting non-Hodgkin's lymphoma for the plaintiffs in this case?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No. The question I was asked to evaluate is different. The question was: What's the

relationship between his exposure and the cancer he did develop. That's a different question than what was his quantitative risk of developing cancer from the exposures.

BY ATTORNEY ANTONUCCI:

Q. Did you consider his quantitative risk of developing cancer from the exposures?

ATTORNEY GREENWALD: Objection. Form, asked and answered.

THE WITNESS: Sure, I mean, that's why it's point number 3 in my specific causation summary.

BY ATTORNEY ANTONUCCI:

Q. So in point number 3 in your specific causation summary, how does that contribute to your conclusion that it is a -- that Mr. Carter's exposure to contamination at Camp Lejeune was more likely than not a substantial contributing factor to his NHL?

A. Well, he developed cancer. He's been exposed to all of these different solvents that, as I went through my general

causation argument, are significant risks for the development of the cancer at issue.

He has an estimated lifetime cancer risk that's well over the so-called de minimis risk level, and from my point of view, it falls into this concept of the multiple processes and risks that go into somebody developing cancer. So particularly given that he has almost no other risk factor -- known risk factors for the cancer at issue, I feel it reasonable to consider this based on everything I just discussed as a significant contribution towards his risk for getting this cancer which he then got.

Q. You used the pronoun "he."

Were you referring to Mr. Carter?

A. Yes.

Q. Would you say that your opinion that Mr. Carter's NHL was caused by his exposures was informed by his increased risk of contracting NHL based on his exposures?

A. Yes.

Q. How so?

A. I think I just went through the chain of reasoning that goes with it. I

think, you know, if you're not wearing your seat belt and you get into a car accident that causes injury, is it known that not wearing a seat belt every time you drive is going to cause an injury? No. But it certainly increases the risk; right? So would drinking alcohol. So would having dementia. So would other things.

But, you know, when you're in the realm of risk and you see that there's a risk that's elevated below a substantially above so-called background risk, then there's no reason to suspect that it did not contribute towards the elevated risk or the incident that occurred.

So I think, you know, in the face of the evidence of exposure, that his exposure is many, many times -- a couple of orders of magnitude greater than so-called de minimis risk, the absence of other known risk factors, the fact that he got cancer, I think it's reasonable to conclude that, well, it is a substantial contributing factor.

Q. How do you square that with the

World Health Organization's classification of Mr. Carter's increase in risk as being very low?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I mean, you know, it's the same thing. You get into a car. What's the risk you're going to get into a car accident and have an injury? It's low. But when it happens, you have to understand what the potential factors that might have occurred that would have increased the risk. And those are qualitative, you know, designations that are meant for the purpose of communication. That's what -- that's how the WHO viewed that terminology.

BY ATTORNEY ANTONUCCI:

Q. So would it be inappropriate to use those determinations as part of an evaluation of causation?

A. No. It's just a way of trying to weigh, you know, what's the -- how to think of the risk on a, I guess you could say,

semi-quantitative basis.

Q. And earlier I believe you used the analogy of driving a car and the potential risk factors for injury such as not wearing a seat belt, drinking alcohol, or having dementia; is that right?

A. Yes.

Q. Are there other risk factors for getting injured while driving a car?

A. Oh, sure. A zillion.

Q. Do we know all the risk factors for getting injured when driving a car?

A. We know a lot of them.

Q. Is there ever a possibility that someone could get injured driving a car based on a risk factor we previously didn't know about?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Good question. I don't know. Certainly there could be a risk factor that's out of your control. Road conditions, the other driver, et cetera, et cetera, et cetera. Is there a risk factor

that's completely unknown? I don't know.

I would say that, you know, when an accident investigation is done, usually some sort of combination of risks is identified. Restraining it to a territory, of course, that is a bit far from this particular case.

BY ATTORNEY ANTONUCCI:

Q. Yeah, I'd like to rein us back in. I'm sorry about that. That was my fault.

If you look at Exhibit 4, I believe that's the Carter specific causation initial report, which you have in front of you, page 12.

A. Okay.

Q. Paragraph 2. The second sentence there starting with the word "Finally."

Do you see that?

A. Yes.

Q. It says: Finally, the ability of the environmental exposures Mr. Carter sustained to increase the risk of cancer is also consistent with two of the few epidemiological studies that have been

conducted on communities exposed to PCE or TCE in drinking water.

Did I read that correctly?

A. You did.

Q. And I believe you made a similar comparison in Mr. Kidd's report; is that right?

A. I think so.

Q. Okay. So the two studies you're referring to here are Aschengrau, et al., 1993 and Cohn, et al., 1994; correct?

A. Correct.

Q. The title of Aschengrau is Cancer Risk and Tetrachloroethylene Contaminated Drinking Water in Water in Massachusetts, and the title of Cohn, et al., is Drinking Water Contamination and the Incidence of Leukemia and Non-Hodgkin's Lymphoma; correct?

A. Yes.

Q. Okay. Dr. Hu, you previously testified that the 1993 Aschengrau study was a population-based case control study; correct?

A. Yes.

Q. And that used -- and the Aschengrau 1993 used ecological data to evaluate the relationship between PCE, bladder cancer, kidney cancer, and leukemia; correct?

A. Correct.

Q. And the Aschengrau 1993 study did not evaluate the relationship between PCE and NHL; correct?

A. I don't recall, but if you give me the study, I'll try to refresh my memory if they also looked at that.

Q. Sure.

This is Exhibit 19. Cancer Risk and Tetrachloroethylene Contaminated Drinking Water in Massachusetts by Aschengrau, et al.

(Exhibit Number 19 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. And my question for you, Dr. Hu, based on Exhibit 19 is: Does the Aschengrau 1993 study evaluate a relationship between PCE and NHL?

A. It doesn't look like they studied the matter.

Q. I'm sorry. Can you please repeat that? I missed it.

A. It doesn't look like they studied the matter. That is, it doesn't look like they focused on NHL as part of their research.

Q. Understood.

Did the Aschengrau 1993 study evaluate a relationship between PCE and any NHL subtype?

A. No.

Q. Okay. So you also cited to the Cohn, et al., 1994 study.

That was also an ecological study; right?

A. Yes.

Q. And that study did not have individual data on the study participants' water consumption; correct?

A. Correct.

Q. They did not, in the Cohn 1994 study, have individual data on the study participants' volatile organic compound inhalation data; correct?

A. Correct.

Q. The Cohn, et al., 1994 study did not specify the study participants' exposure levels above 5 parts per billion; correct?

A. Can you repeat the question, please?

Q. The Cohn, et al., 1994 study does not contain information on whether individuals who developed NHL in the above 5 parts per billion group were exposed to, for example, 5.1 parts per billion or 50,000 parts per billion; right?

A. Correct.

Q. The Cohn, et al., 1994 authors noted that their conclusions are limited by a potential exposure misclassification; correct?

A. I mean, that's a limitation of most epidemiology studies, and it's a limitation of theirs as well.

Q. And Cohn, et al., 1994 did not find an association between NHL incidence and exposure to benzene; correct?

A. I am not sure they considered benzene, but if you give me the study, I'll try to refresh my memory.

Q. Sure. This is Exhibit 20.

(Exhibit Number 20 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. And this is Drinking Water Contamination and the Incidence of Leukemia and Non-Hodgkin's Lymphoma by Perry Cohn, et al.

My question, based on Exhibit 20, is whether or not the study authors found an association between non-Hodgkin's lymphoma incidence and an exposure to benzene.

It might help to direct your attention to page 559 of Exhibit 20 under the subheading Other Contaminants.

A. It's a little mysterious because on the one hand, they say, as you pointed out: No association was detected between leukemia or NHL incidence and trihalogenated methyl compounds or with other non-THM VOCs such as benzene, et cetera, et cetera.

But then earlier on, under Exposure Assessment, they say in their '84, '85 first-round mandatory testing, the most commonly occurring non-THM VOCs were TCE,

PCE, and 1,1,1-trichloroethane. They don't even mention benzene.

So all I can say is that they didn't -- they certainly didn't comment on any relationship with benzene except saying that they didn't find a relationship, but it's not even clear whether they had enough data to even examine the subject.

So that's why I'm saying it's a little mysterious. There's just not enough detail that's provided here to see how many cases or controls might have actually been exposed to benzene.

Q. Okay. So on page 10 of Exhibit 3, that's your specific causation initial report for Mr. Kidd.

A. Hold on. I'm bouncing back and forth between Carter and Kidd. All right. Page 10, did you say?

Q. Yes, sir.

A. Okay. All right.

Q. At the, I believe, last sentence of paragraph number 2 beginning with "In another population case control study," and that's the citation to Cohn, et al.

Do you see that?

A. Yes.

Q. You wrote: In another population case control study of cancer incidence rates in New Jersey in relation to drinking water contamination, Cohn, et al., 1994 found that in comparison to women living in unexposed towns, women exposed to TCE at levels exceeding 5.0 parts per billion had a relative risk for NHL of 1.36, 95 percent confidence interval 1.08 to 1.70, and men exposed to TCE at levels exceeding 5 parts per billion had a relative risk for intermediate-grade NHL, diffuse large cell/reticulosarcoma, 1.59, 95 percent confidence interval 1.04 to 2.43; right?

A. Correct.

Q. So first, diffuse large cell and reticulosarcoma are different subtypes of NHL; correct?

A. They certainly were when this report was written. I'd have to know, given the classification that NHL has changed over time, how they're considered now. I would suspect they're also still considered as

distinct.

Q. Okay. However, those two NHLs, diffuse large cell and reticulosarcoma, were analyzed together for the purposes of this study; right?

A. I believe so.

Q. And the Cohn, et al., 1994 study did not find a dose response relationship between TCE and total NHL in men; correct?

A. Let me look at this study, please.

Q. Sure. That is Exhibit 20.

A. Oh, right here.

Q. It may help to look at page 559.

A. Thank you.

Well, there was a suggestion of a dose response relationship among the females with the -- with the rate ratio going from 1.0 to 1.02 to 1.36, but I wouldn't consider that as substantial evidence of a dose response relationship and that you don't see evidence of that in the males. So I'd agree with you.

Q. Okay. And then I believe Table 2 of page 559 is where you were looking for that information?

A. Yes.
Q. So if you look at the next row down below total NHL, there's low-grade NHL total; right?
A. Yes.
Q. And for males, there's no dose response relationship detected in this study for low-grade NHL total; correct?
A. Correct. There's a suggestion of it among the females again but not in the males.
Q. And for PCE and total NHL in men, the Cohn, et al., study also did not find a dose response relationship; correct?
A. Well, before we leave Table 2, I noted that for high-grade NHL, there seems to be a dose response relationship in men and women, but again, the numbers are low.
So what was your question?
Q. Actually, I'd like to look at high-grade NHL. You noted that there's a dose response relationship, but the numbers are low.
What does that mean?
A. The number of cases are low; so the

risk estimates are somewhat unstable. So I wouldn't put too much meaning in it, but I just note that there does seem to be a monotonic increase in the rate -- ratios.

Q. What's the difference between a high- and low-grade NHL?

A. That refers to the pathology and whether it seems to be, you know, highly abnormal in terms of the cytology and grade of the tumor.

Q. Is mantle cell lymphoma a high-, intermediate-, or low-grade NHL?

A. I don't think it's classified as either. I think within mantle cell, there might be high-grade and low-grade, but I don't think it itself falls into one of those baskets, as far as I know.

Q. Do you know why then they grouped diffuse larger cell as an intermediate grade of NHL for this study?

A. I don't.

Q. Is there anything about diffuse large cell lymphoma that indicates it would have an intermediate-grade cytology?

A. I don't. And, you know, again, I

point out that this study was done some time ago. Let's see, 1993 it was submitted, and I just don't know how the classification system might have evolved since then.

Q. If you turn to page 560, Table 4 is titled Number of Recorded Cases, Age-Adjusted Rate Ratios, and 95 Percent Confidence Interval For Non-Hodgkin's Lymphoma, NHL, in the Northern New Jersey Study Area, 1979 to '87 by Perchloroethylene, PCE, Exposure Category and Sex, All Races; correct?

A. Yes.

Q. So the first row of this study on page -- of this table on page 560, Table 4 says TCE exposure; right?

A. You mean the first column?

Q. Excuse me, yes. The first column.

A. You said the first row. I think you meant the first column. First column says it's about TCE exposure.

Q. But the title of the table says it's about PCE exposure, doesn't it?

A. Yes.

Q. Do you happen to know -- I mean,

given that the Table 2 is TCE exposure, would it make sense that Table 4 is actually discussing PCE exposure?

A. It looks like it's a typo. I would guess it's a typo.

Q. Okay. So for PCE exposure in Table 4, the Cohn, et al., study did not find a dose response relationship between exposure to PCE and total NHL in them; correct?

A. Well, before I conclude that it's a typo, I'd better look at the text to see how they refer to Table 4. Just a minute. This is weird.

Do you happen to see where in the text it's referring to Table 4?

Q. So I'll tell you, I couldn't find it personally.

A. Oh, really? This editor was asleep at the job. This is unfortunate. Oh, here we go. Yeah, perchloroethylene, Tables 3 and 4. So that's a typo in Table 4. That should be PCE exposure in the first column. All right.

So what was your question again?

Q. So the Cohn, et al., 1994 study did not find a dose response relationship between exposure to PCE and total NHL in men; correct?

A. I would agree with that.

Q. It also did not find a dose response relationship between intermediate-grade NHL diffuse large cell reticulosarcoma in men; correct?

A. I agree with that.

Q. The Cohn, et al., 1994 study also did not find a dose response relationship between high-grade NHL total in men exposed to PCE; correct?

A. I agree with that.

Q. And the Cohn, et al., 1994 study did not find a dose response relationship between exposure to PCE and total -- excuse me, high-grade NHL non-Burkitt's in men; correct?

A. I'd agree with that.

Q. And Cohn, et al., on page 557 note that an earlier New Jersey Department of Health study reported an association between leukemia and drinking water contaminants

among females only, and so the cases were grouped by sex for separate analysis; is that right?

A. That's a pretty busy page, but if you give me a little help, I can try to find that sentence.

Q. Second-to-last paragraph on page 557. It's the last sentence.

A. Okay.

Q. It says: Since the earlier NJ DOH study (3) reported an association between leukemia and drinking water contaminants among females only, cases were grouped by sex for separate analysis.

Did I read that correctly?

A. You did.

Q. And then 3 in that sentence is a citation; right?

A. Yes.

Q. Would you expect to see an association between NHL and drinking water contaminants in women only?

A. I mean, the more we do environmental epidemiology, the more that we see there are sex-specific effects, and

there's very good biological reasons for that. In terms of hormones, physiology, anatomy, habits, et cetera.

So would I expect to see differences in terms of the environmental carcinogen between the sexes for NHL specifically? Not necessarily.

I'm not aware of that, but if they were found, I wouldn't be surprised. It could be as a result of some of the factors I just mentioned that there are biological reasons for that behavior, but also, you know, these ecological studies are pretty crude tools.

You might see a relationship here or there, but for the most part, it's subject to, again, non-differential misclassification which tends to dilute the truer relationships between exposures and diseases. We've seen in one sex versus the other that doesn't necessarily mean that there's always going to be sex-specific differences in that exposure disease relationship.

Q. Dr. Hu, is it your opinion that

non-differential misclassification always biases towards the null?

A. That is true for the great majority of cases, and I am aware there have been some exceptions that have been hypothesized and discussed. But in these types of studies, I would expect the non-differential misclassification to bias the effect estimates towards the null.

Q. And why is that?

A. Because without directly identifying the exposure of individuals, you will, in the most extreme situation, assume that someone that was highly exposed -- actually, lowly exposed. Someone who was lowly exposed actually is highly exposed. And if there was a true exposure disease relationship, I think common sense would dictate, gee, you're not going to see it that way because you've misclassified whether they're exposed or not.

That's the most extreme situation. But in terms of a continuous exposure measure, it's very similar. You know, you just start to dilute the contrast between

exposure categories.

Q. Is there a way to test whether the misclassification bias is biasing towards or away from the null hypothesis in a human epidemiology study?

A. Well, the most important way to try to determine whether that might be the case is to look at the methods used to classify exposure and then do a validation study to see whether the true exposure is different from the estimated exposure in a non-differential way. That would be the one way to do it. Non-differential in terms of the relationship to the outcome.

But, you know, we don't have the luxury of that for most of these kinds of investigations.

Q. Can a quantitative bias analysis be used to assess whether misclassification bias is biasing towards or away from the null hypothesis?

A. No. I think that tool, which Bove, et al., used, was used to determine whether there was uncontrolled confounding, not bias.

Q. So on page 10 of Exhibit 3, the specific causation initial report for Mr. Kidd.

A. Yeah.

Q. Paragraph 2 starts with "That Mr. Kidd's."

Do you see that?

A. Yes.

Q. Here you wrote: That Mr. Kidd's exposures to TCE, PCE, and benzene were at least as likely as not causative of his NHL is also consistent with the findings of the most recently published epidemiological study of the cancer incidence experience of personnel stationed or employed at Camp Lejeune versus Camp Pendleton that I discussed earlier.

Did I read that correctly?

A. You did.

Q. And on pages -- starting on page 8 of the specific causation report for Mr. Kidd, again, Exhibit 3, under subheading A. TCE, PCE, Benzene, and NHL, do you see the sentence starting "Furthermore"?

A. Yes.

Q. Here you wrote: Furthermore, in my January 31, 2025, supplement to my general causation report, I provided the opinion that the results of the Bove, et al., cancer incidence study allow me to specifically conclude that the combination of TCE, PCE, and benzene at the levels of community exposure experienced at Camp Lejeune are more likely than not a risk factor for NHL.

As noted in my supplemental report, this was a cancer incidence study in which elevated hazard ratios, AHRs, with confidence interval ratios (the ratio of the upper to lower limits of the 95 percent confidence interval) less than or equal to 3 (an indicator of precision) were reported for a number of cancers.

Among them were the mantle cell subtype of NHL and the marginal zone B-cell subtype of NHL, which among the Camp Lejeune (vs. Camp Pendleton) Marines/Navy personnel had adjusted hazard ratios of 1.26 (95 percent confidence interval: 0.73 to 2.19; confidence interval ratio: 3.0) and 1.45 (95 percent confidence interval: 0.92 to

2.28; confidence interval ratio: 2.5.)
Did I read that correctly?
A. You did.
Q. Mr. Kidd had -- first of all, Mr. Kidd had diffuse large B-cell lymphoma; correct?
A. Right.
Q. Not marginal zone B-cell or mantle cell lymphoma?
A. Correct.
Q. So the cancer incidence study, which is Exhibit 17, did not include any statistical significance testing; correct?
A. Well, it had confidence intervals which are just as good, if not better.
Q. Are you aware that Dr. Bove testified he did not perform statistical significance testing for his 2024 cancer incidence study?
A. I'm aware of that.
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: You know, that's just a matter of entering P values, but if you have confidence intervals,

like I said, it's just as good.
BY ATTORNEY ANTONUCCI:
Q. Okay. In the 2024 cancer incidence study, there was no individualized exposure assessment; correct?
A. I would agree with that.
Q. And the 2024 cancer incidence study by Dr. Bove did not control for or consider family history of NHL; correct?
A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for family history of cancer, in general; correct?
A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for inherited immunodeficiency syndrome; correct?
A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for a history of organ transplants; correct?
A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for or consider organ transplants; correct?

A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for or consider Sjogren's syndrome; correct?
A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for or consider systemic lupus erythematosus; correct?
A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for or consider HIV; correct?
A. I agree with that.
Q. The 2024 Bove cancer incidence study did not control for or consider other potential occupational exposures among Marines and Navy service members; correct?
A. I agree with that.
Q. And if you look at Table 3 of the cancer incidence study and, again, that's Exhibit 17.
A. I'm sorry. Which table?
Q. Table 3 of Exhibit 17.
A. Okay.
Q. As we discussed, Table 3 shows the

overall NHL adjusted hazard ratio of 1.01; correct?

A. Correct.

Q. The diffuse large B-cell lymphoma adjusted hazard ratio is 0.89; correct?

A. Correct.

Q. And what that is showing is that the risk of diffuse large B-cell lymphoma was 0.89 times as high in Marines from Camp Lejeune compared to Marines from Camp Pendleton between 1975 and 1985; correct?

A. Correct.

Q. The Bove 2024 cancer incidence study also gave results by duration of residency at Camp Lejeune for the Marine and Navy cohort; correct?

A. Correct.

Q. Are you aware of any other studies with PCE exposure levels similar to those at Camp Lejeune?

ATTORNEY ANTONUCCI: For the record, the witness is currently reviewing Exhibit 10.

THE WITNESS: Yeah, I just wanted to see whether the U.K. Biobank study

might have included PCE. I don't think so. Not as I sit here today.

BY ATTORNEY ANTONUCCI:

Q. Are you aware of any other studies with TCE exposure levels similar to those at Camp Lejeune?

A. Well, we just talked about ecological studies -- right? -- which involved TCE, I thought?

Q. Are you referring to Cohn or Aschengrau?

A. Yeah.

Q. Other than Cohn or Aschengrau, are you aware of any other studies with TCE exposure levels similar to those at Camp Lejeune?

A. Not that I'm aware of -- not that I recall, sitting here today.

Q. And other than Cohn, Aschengrau, or ATSDR's work, are you aware of any other studies with benzene exposure levels similar to those at Camp Lejeune?

A. Well, have we talked about the U.K. Biobank study?

Q. Not yet.

A. That has exposures, I think, within order of magnitude of what was experienced at Camp Pendleton -- Camp Lejeune. Sorry.

Q. Are you aware of any other studies with benzene exposure levels similar to those at Camp Lejeune?

A. Not as I sit here today.

ATTORNEY ANTONUCCI: I would like to take a quick five-minute break if that's okay.

THE VIDEOGRAPHER: We are now going off the record. This is the end of media Unit Number 4, and the time is 3:04 p.m.

(Recess taken from 3:04 p.m. to 3:10 p.m.)

THE VIDEOGRAPHER: We are now back on the record. This is the beginning of media Unit Number 5, and the time is 3:10 p.m.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, we are back on the record, and you understand you're still under oath; correct?

A. Yes.

Q. Did you discuss the substance of your testimony with your attorney during the break?

A. I did not.

Q. Please turn to Exhibit 3. That's the specific causation initial report for Mr. Kidd.

A. Okay. All right.

Q. Specifically page 10.

A. Okay.

Q. Excuse me. I meant page 11. My apologies.

A. Okay.

Q. Paragraph 5 on page 11 lists risk factors, and I'm just going to read from your report here.

It says: Other than TCE, PCE, and benzene as risk factors (that I had discussed in my general causation report), other risk factors for which there is evidence of an association with NHL include being white (versus African-American or Asian American), having family history of a first-degree relative with NHL, previous treatment with cancer chemotherapy drugs,

radiation exposure, weakened immune systems (e.g., those weakened by immunosuppressive drugs or HIV/AIDS), certain inherited syndromes associated with immunodeficiency (e.g., ataxia-telangiectasia, Wiskott-Aldrich syndrome), autoimmune diseases (such as rheumatoid arthritis, systemic lupus erythematosus), previous lymphoma, chronic infections that cause continuous immune system activity (e.g., Helicobacter pylori; Chlamydophila psittaci, Campylobacter jejuni, Hepatitis C), and breast implants.

Did I read that correctly?

A. Yes. I am impressed.

Q. Thanks.

So these are -- what I just read were the risk factors for NHL; correct?

A. These are the risk factors that appear on the American Cancer Society website.

Q. Okay. And are these risk factors for NHL, in general, as a group of cancers?

A. Yes.

Q. As opposed to any given subtype;

correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Correct.

BY ATTORNEY ANTONUCCI:

Q. So, for example, would you consider H. pylori infection to be associated with diffuse large B-cell lymphoma?

A. Specifically? I think I would consider that as a risk factor by default, but if -- you know, I haven't looked intensely into the literature myself.

Q. Do you know whether H. pylori infection is a risk factor for diffuse large B-cell lymphoma?

A. I don't know if that particular specific question has ever been examined.

Q. Do you know whether H. pylori is associated with a different subtype of non-Hodgkin's lymphoma?

A. As I sit here today, I don't recall looking at the literature to look at that topic.

Q. Okay. Did you consider risk factors for diffuse large B-cell lymphoma in

particular?

ATTORNEY GREENWALD: Objection. Form, asked and answered.

THE WITNESS: Well, these are general risk factors for NHL as they appear on the American Cancer Society website. If Mr. Kidd had evidence of any of these other risk factors, I would have plowed more deeply into the literature, but since he didn't, I did not.

BY ATTORNEY ANTONUCCI:

Q. Are the causes of diffuse large B-cell lymphoma generally known?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, there's some that are probably listed if you want to get specifically into diffuse large B-cell lymphoma. I don't remember exactly what those risk factors are specifically just for DLBCL, but I know that some of that has been done.

BY ATTORNEY ANTONUCCI:

Q. In what percent of DLBCL cases is

the cause of cancer known?
A. I don't think -- I don't recall anybody ever trying to estimate that.
Q. Are the causes of MACL known?
A. You mean mantle cell lymphoma?
Q. Yes, sir.
Are the causes of mantle cell lymphoma known?
A. Well, some have been -- some risk factors have been identified.
Q. Dr. Hu, you agree that the different subtypes of non-Hodgkin's lymphoma have different risk factors; correct?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: I agree that different studies have been done looking at different subtypes. All of these are somewhat rare; so it's -- the literature on the epidemiology of each of the subtypes is -- I guess I would say it's sparse.
So I wouldn't say that they're known, but there have been various risk factors have been identified in

various studies.
BY ATTORNEY ANTONUCCI:
Q. So my question was that the different subtypes have different risk factors.
Do you agree with that?
ATTORNEY GREENWALD: Objection. Form, asked and answered, mischaracterizes his testimony.
THE WITNESS: I'll say it again. Different risk factors have been identified in studies. That doesn't mean that it is the absolute truth that each subtype of NHL definitely has different risk factors. We don't know that for certain. I can say that, in general.
So yes, maybe some study found these risk factors. Some other study found other risk factors for other tumors. But given the rarity of these tumors, I think it's very hard to conclude that the subtypes of NHL either have all the same risk factors or that they're all different.

BY ATTORNEY ANTONUCCI:

Q. Okay. Earlier you mentioned citing to the American Cancer Society for the list of non-Hodgkin's lymphoma risk factors; correct?

A. Correct.

ATTORNEY ANTONUCCI: All right. I'm marking for identification Exhibit 21. This is a printout of the American Cancer Society's website titled Non-Hodgkin's Lymphoma Causes, Risk Factors, and Prevention.

(Exhibit Number 21 was marked for identification.)

THE WITNESS: And what date was that done?

BY ATTORNEY ANTONUCCI:

Q. It should say at the bottom of the printout.

Dr. Hu, I will ask that you review Exhibit 21 and let me know if this is the website that you reviewed when drafting your report.

A. It is.

Q. So if you look at page 2, there's,

again, a list of risk factors; correct?
A. Correct.
Q. Above that list of risk factors, the American Cancer Society states: Researchers have found several factors that can affect a person's chances of getting non-Hodgkin's lymphoma, NHL. There are many types of lymphoma, and some of these factors have been linked only to certain types.
Did I read that correctly?
A. You did.
Q. Okay. Other than the risk factors listed here, did you consider any other risk factors for NHL?
A. No.
Q. And to clarify, my last question was referring to page 2 of Exhibit 21.
You did not review -- or consider any other risk factors other than those; correct?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: Before I respond, let me also just refer to my general causation report to be certain. Hold

on.

Okay. Can you repeat your question, please?

BY ATTORNEY ANTONUCCI:

Q. Other than the risk factors listed on page 2 of Exhibit 21, did you consider any other risk factors in offering your opinions?

A. In terms of the differential etiology, no.

Q. One of the risk factors on page 2 of Exhibit 21 is the history of breast implants; correct?

A. Yes.

Q. Do you know whether a history of breast implants is associated with all NHL, in general, or just specific types?

A. I don't recall. I don't know.

Q. Is a history of breast implants a risk for mantle cell lymphoma?

A. I don't -- I don't know. I don't recall.

Q. Is a history of breast implants a risk factor for any non-Hodgkin's lymphoma that's not situated near the breast?

A. Non-Hodgkin's lymphoma can crop up anywhere; so this is not a listing of risk factors for breast cancer. This is a listing of risk factors for non-Hodgkin's lymphoma.

So it's not a matter of cancer cropping up in the breast. It's the fact that the exposure here is in the breast. It's the breast implants and their associated chemicals and the reaction that's caused by those chemicals.

Q. So I apologize if my question was unclear.

I'd like to know if a history of breast implants is a risk factor for a non-Hodgkin's lymphoma that is not near the breast.

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I mean, the fact that it's on this list is a reflection of the fact that breast implants are associated with non-Hodgkin's lymphoma of some type. Non-Hodgkin's lymphoma of any type could have cropped up all

over the body, maybe near the breast but typically in lymph nodes or other places where there are collections of lymphocytes.

I think I've answered your question, hopefully.

BY ATTORNEY ANTONUCCI:

Q. Do you know what caused these risk factors to be placed on the American Cancer Society's website?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: The American Cancer Society has a scientific advisory board. They go over the science. They go over research objectives. They actually sponsor some of their own research. They sponsor some research fellowships that some of my trainees have obtained.

So I don't know exactly what review body in the American Cancer Society would be responsible for identifying those factors that have a sufficient amount of evidence to show

up on their website, but I am pretty certain that they have done it in a systematic way that there's a process for doing that.

BY ATTORNEY ANTONUCCI:

Q. Okay. And it's true that the American Cancer Society's website says that some risk factors have been linked only to certain types of non-Hodgkin's lymphomas; correct?

A. Correct.

Q. Dr. Hu, you rolled in exposure to TCE, PCE, and benzene as risk factors in your differential etiology for Mr. Kidd based on Dr. Reynolds' exposure calculations; is that correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I wouldn't quite put it that way. I mean, I considered it a significant risk factor based on my general causation analysis, based on the exposure assessment and the estimates of likely exposure that the Camp Lejeune residents endured, and

also the specific exposure assessments done with these plaintiffs.

BY ATTORNEY ANTONUCCI:

Q. Okay. Did you rule out exposure to TCE, PCE, or benzene as risk factors in your differential etiology for Mr. Kidd?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Rule it out? Clearly not. I mean, I accepted the exposure assessment that was done on Mr. Kidd.

BY ATTORNEY ANTONUCCI:

Q. Okay. And because exposure to TCE, PCE, and benzene are risk factors for NHL and because Mr. Carter ultimately got NHL, you opine that the exposure caused his disease; correct?

ATTORNEY GREENWALD: Object to form.

THE WITNESS: Well, there's a whole number of other factors I looked at. I looked at exposure duration. I looked at latency. I considered these other alternative risk factors. It's

a process that needs to be done systematically and in a case where specific causation is proposed.

BY ATTORNEY ANTONUCCI:

Q. I'd like you to turn to page 7 of Exhibit 21, the American Cancer Society's non-Hodgkin's lymphoma risk factors. Underneath what causes non-Hodgkin's lymphoma, the first sentence states: Researchers have found that non-Hodgkin's lymphoma, NHL, is linked with a number of risk factors, but the cause of most lymphomas is not known. This is complicated by the fact that there are many types of lymphomas are actually a diverse group of cancers which might have different causes.

Did I read that correctly?

A. You did, and there's a typo there. I think they forgot the word "that."

Q. So it's true that the cause of most cases of DLBCL is unknown; correct?

ATTORNEY ANTONUCCI: Objection. Form.

THE WITNESS: Well, when there's a very -- a person who presents with

DLBCL, diffuse B-cell lymphoma, I think it's true that, in many of those cases, there will not be any of the causes listed here identified, and they may not have been exposed to TCE, benzene, or PCE in any meaningful way as well.

So that would be someone whose, you know, cancer it's unclear what caused it. Of course, you know, we have limited knowledge about all the genetics, poly genetics. And that's all the subject of current research.

BY ATTORNEY ANTONUCCI:

Q. Are you familiar with the InterLymph Non-Hodgkin's Lymphoma Subtypes Project?

A. I am familiar with the -- one of the non-Hodgkin's lymphomas consortia. It might have the same name. What was the name you mentioned again?

Q. InterLymph non-Hodgkin's lymphoma subtypes.

A. What is that exactly?

Q. I believe what you just described

as a consortia.

A. Consortia?

Q. And you cited to several studies as part of that project in your causation report.

A. Right. So I have some familiarity with it.

Q. Are you aware that the InterLymph Non-Hodgkin's Lymphoma Subtypes Project concluded that although risk factors for diffuse large B-cell lymphoma have been suggested, that their independent effects modification by sex and association with anatomical sites are largely unknown?

A. I'm not aware that they stated that, but I have no reason to disagree with it.

Q. Did you consider the fact that most cases of DLBCL is unknown in performing your differential etiology for Mr. Kidd?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I think what your question poses for me is the concept that since you can't definitively say

that one thing is the cause, and there is no way of identifying an exact cause, along with other scientific opinions expressed in the literature, like isn't it true that the cause is just random.

And in my view, in the field of cancer epidemiology, what has been debated is the extent to which cancer is random, and I've already discussed that actually in one of my rebuttal reports because, in fact, that view which was expounded by two scientists, I think they were both at Stanford, based on some in vitro studies in math modeling was vigorously debated and disagreed with by the International Agency For Research on Cancer and other scientists based on very reasonable considerations in my view.

So back to your question, did I consider whether the cause of Mr. Kidd's cancer was simply unknown? That's a theoretical, but in his particular situation, we know he got

cancer. We know that there's some risk factors that are identified. He has a significant risk factor that was identified. He was exposed to that risk factor at a pretty significant level for many years, and many of the other attributes of his clinical course and his personal history are consistent with this being a substantial contributing factor towards his cancer, and that's why I concluded what I concluded.

BY ATTORNEY ANTONUCCI:

Q. So there was a lot there.

A. Yeah.

Q. Maybe we -- we'll try to take this one at a time. You mentioned the two scientists at the Stanford that had the random cancer theory.

Was that Tomasetti --

A. Tomasetti & Vogelstein.

Q. -- and Vogelstein?

A. Yes.

Q. We'll come back to discuss that.

So I think really what I was

getting at was sort of the last part of the answer you just gave, the fact that Mr. Kidd was exposed, he didn't have other risk factors, the exposure was substantial, and he ultimately got the disease; right?

A. Yes.

Q. Is that -- is that an accurate characterization of --

A. That's an accurate general characterization, although this -- other things that I discussed in the -- in my reasoning.

Q. And the fact that the cause of most cases of NHL is unknown is sort of theoretical in your view and not relevant to this analysis; is that right?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I mean, it's not theoretical. I mean, it's kind of true. We can't explain every case of cancer in a definitive way. There's no epidemiology studies where you put in all the risk factors and you explain 100 percent of the variance of

the cancer rate. It just doesn't exist. But that doesn't mean that risk factors that are identified are insignificant or not contributory or irrelevant.

BY ATTORNEY ANTONUCCI:

Q. I want to take a step back and clarify.

When we talk about unknown causes of cancer, is that different from random causes?

A. Well, random causes really is a reference to complete randomness. It's not that there's unidentified causes. There's just randomness. And randomness -- and this is part of the -- part of the critique that scientists have made of Tomasetti & Vogelstein is that randomness is not something, for instance, that you would expect to be different in one country versus another or over time with different rates of cancer. And what's really notable about cancer rates is that they do differ between populations, between time periods, between geographic areas, and those are not -- you

know, those are not things that one would expect if cancer was just random or if a significant portion of cancer was just random.

Q. So ultimately, Dr. Hu, you concluded that the causes of Mr. Carter's and Mr. Kidd's non-Hodgkin's lymphomas were not unknown; correct?

A. Well, I concluded -- I'm not sure it's the equivalent that the exposures that were identified at Camp Lejeune were more likely than not a significant contributing factor towards the cancers.

Q. Why is that not equivalent to being a known cause?

A. I think the way it's phrased, these are a known cause. It makes it ambiguous as to whether it's known because the risk factors that they had and they had proper exposure, et cetera, et cetera, or whether, oh, yeah, we did a test, and it's known that these are the causes; right?

So that ambiguity makes me uncomfortable with characterizing their relationship that way; so I feel more

comfortable characterizing it the way I just did and in my reports.

Q. Dr. Hu, you would agree that even among epidemiological studies that find increased risk of non-Hodgkin's lymphoma, the risk ratios and hazard ratios are very rarely over 2; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, that's often the case with cohort studies. That's not necessarily the case with case-control studies since case-control studies are specifically designed to focus on cancers themselves and less on the population and it's longitudinal experience. Those are general statements and not always true.

BY ATTORNEY ANTONUCCI:

Q. Which case-control studies indicate an increased risk of non-Hodgkin's lymphoma over 2?

A. I'd have to go back and look at my general causation report. Shall I?

Are you talking about a specific exposure, TCE, PCE, benzene or just in general?

Q. Are you aware of any epidemiological studies that find an increased risk of non-Hodgkin's lymphoma for any of TCE, PCE, or benzene over the risk or hazard ratio of 2?

A. So just turning to my general causation report, if you look at the page 38 in my Bradford Hill factor analysis of benzene, I was looking at the strength of association criteria -- consideration 1. And among the studies that I discussed was a cohort study by Wong, et al., that found a relative risk of 4.12; a cohort study by Hayes, et al., that found a relative risk of 4.7; a case-control study by Fabbro-Peray that found a odds ratio of 5.07; a case-control study by Xu, et al., that found an odds ratio of 2.78.

So that's an example of epi studies that found risk ratios greater than 2 for one of the contaminants in non-Hodgkin's lymphoma.

Q. Are those all specific to benzene?
A. Yes. And then if you go to my other analyses, let's see. Then in the strength of association consideration for TCE, I discussed the case-control study by Hardell that found an odds ratio for TCE with NHL of 7.2, an occupational cohort study that found that workers exposed to TCE had an elevated standardized incidence ratio of 3.5. So those are some examples for TCE.
And as for PCE, let's see if I can find anything. And then for PCE under Strength of Association under page 32, I quoted Antilla, et al., which showed among Finnish workers a standardized incidence rate for PCE and NHL of 2.55; Selden and Ahlorg's cohort study in Swedish dry cleaning workers found a standardized incidence rate of 2.05.
And I quoted some other studies too. But those are examples, I guess.
Q. Okay. Did any of the studies you just listed evaluate diffuse large B-cell lymphoma or mantle cell lymphoma individually?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I don't recall, but if you give me the slew of studies I just quoted, I can take a look.

BY ATTORNEY ANTONUCCI:

Q. Are you, Dr. Hu, confident that the cause of Mr. Carter's non-Hodgkin's lymphoma must have been one of or a combination of the risk factors you listed?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, I mean, I stated my opinion in precisely the way my view exists, which is that -- let's go to the -- make sure I don't misquote myself.

It is my opinion to a reasonable degree of medical certainty that the combination of Mr. Carter's exposures to TCE, PCE, and benzene from Camp Lejeune more likely than not was a substantial contributing factor to the causation of mantle cell lymphoma.

That is precisely the way I think

about it.
BY ATTORNEY ANTONUCCI:
Q. In your opinion, is it at all possible that Mr. Carter or Mr. Kidd's NHL was caused by a random genetic mutation?
A. It is possible.
Q. In your opinion, is it at all possible that Mr. Carter or Mr. Kidd's non-Hodgkin's lymphoma was caused by a risk factor other than the ones you listed in your reports?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: Well, let me just back up for a moment. As I said earlier in this deposition, in my view, and I believe this is true of most of the people who are in environmental epidemiology or do research in carcinogenesis -- and carcinogenesis is not only a multistep but multifactorial process.
So when I said would I be surprised that, let's say, a genetic factor was the cause, I would say I

would not be surprised if it was a cause particularly since most chronic disease outcomes we now believe are a complex interplay between environment and genes.

So I would feel comfortable saying it wouldn't surprise me if it was a cause. But would I be surprised if it was the cause and nothing else was the cause? I'd be surprised. Of course, there's no way of knowing.

BY ATTORNEY ANTONUCCI:

Q. And to be clear, is it your opinion -- strike that.

It's not your opinion that everyone who is exposed to contaminated water at Camp Lejeune developed non-Hodgkin's lymphoma; correct?

A. Correct.

Q. How did you decide which risk factors to consider in performing your differential etiology? Was it based on the ACS list?

ATTORNEY GREENWALD: Objection. Asked and answered.

THE WITNESS: Yes, and that's because it's a fairly comprehensive list. It's been agreed upon by a sub-guidance body in one of the most prominent, you know, objective rigorous cancer advocacy and research organizations, and I feel that was a reasonable way of approaching it.

BY ATTORNEY ANTONUCCI:

Q. Okay. So for Mr. Kidd, you wrote -- you listed the risk factors on page 11 of your initial report for Mr. Kidd and continued: Of these risk factors, Mr. Kidd had only one, i.e., he's white.

Is that correct?

A. Yes.

Q. You go on to say: Overall, Mr. Kidd had no clear evidence of any known risk factor for NHL; correct?

A. I guess I could have said other than the fact that he was white.

Q. Okay. So it is your opinion that being white is a risk factor for NHL?

A. Well, that's, you know, what the ACS listed among risk factors. I do believe

seen some of that identified in epidemiology that I've read; so I have no reason to disagree with it.

Q. Sex and specifically being male is also a risk factor for NHL; correct?

A. That is true. He is a male.

Q. Did you consider the fact this Mr. Kidd is a male in your differential etiology?

A. Well, I mean, yes, but I think that's -- doesn't -- it doesn't undercut the rest of my opinions.

Q. How did you weigh Mr. Kidd's sex when performing your differential etiology?

A. Well, if it turns out that sex was an overwhelming risk factor, in other words, like testicular cancer, you're not going to get any women. That's only a risk factor for men. Then, of course, I would look at that very strongly, very different.

But in this case, yes, being male is associated with a somewhat elevated risk. Okay. But would that undercut the other carcinogenic risk factors that he had? No.

Q. Is that explained in your report?

A. No, but I'm explaining it to you now.

Q. You also noted that Mr. Kidd's exposure to radiation would not be considered an unusually high amount of radiation exposure in relation to the general public; correct?

A. Correct.

Q. That's because he had X-rays in 2009, five to six dental X-rays, and no CT scans; correct?

A. Correct.

Q. How did you determine Mr. Kidd did not have an unusually high amount of radiation exposure in relation to the general public?

A. Well, and one of the things I also do is I review radiation epidemiology and the radiation epidemiology studies that are done looking at radiation as a risk factor, and there has been some really good low-level radiation studies. The typical dose response in a general population is looking at categories of radiation exposure that the lowest exposure level is very

typical of the kinds of radiation experience that he had.

And the higher levels that are associated with, you know, these elevated risks are typically folks who have had CAT scans, or who have had radiation therapy, or who have had other sources of radiation exposure that are much more high dose than what he experienced.

Q. So is it fair to say that you compared Mr. Kidd's radiation doses to radiation doses in epidemiology studies?

A. In what I have seen in epi studies of low-level radiation and cancer, yes.

Q. Genetic susceptibility loci have also been associated with an increased risk for diffuse large B-cell lymphoma; correct?

A. Yes.

Q. Did you consider genetics as a risk factor for Mr. Kidd's diffuse large B-cell lymphoma?

A. Well, we have no information on that. The genetic loci that you mention come from GWAS studies, so-called genome-wide association studies. And, you

know, they have not identified a particular gene that he was either tested for that is commonly tested for that would allow clinicians to understand whether a particular genetic mutation was, in fact, a major factor in his condition.

So I considered it, but there was no evidence of that in his case.

Q. Is there any evidence that genetics was not involved in the causation of Mr. Kidd's non-Hodgkin's lymphoma?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No.

BY ATTORNEY ANTONUCCI:

Q. In Mr. Kidd's initial report, and, again, that's Exhibit 3, page 6, just above subheading 2.

A. I think I just mixed up my own file. One second.

Q. I think you might have it backwards if you flip it over.

A. Yeah. Oh, there it is. Thank you. I just tucked it underneath by mistake. Sorry. Yeah, here we go.

What page again?

Q. Page 6, Exhibit 3.

A. Thank you.

Q. For Other environmental history of potential relevance you wrote: Mr. Kidd related that he was occasionally exposed to paint thinners and ammonia bleach. He also occasionally used insecticides to get rid of flies. He also used Roundup, about ten times total.

Did I read that correctly?

A. Yes.

Q. Is exposure to paint thinner a risk factor for NHL?

A. Not that I recall.

Q. Why did you list it under environmental history of potential relevance?

A. Well, when I take a history from a patient, you know, I certainly pay attention to the historical attributes that are relevant to the disease, but I also tend to take a general history just to see if there are other general exposures or general considerations that should be recorded. I

try to be thorough.

Q. Is exposure to ammonia bleach a risk factor for NHL?

A. Not that I recall.

Q. Is exposure to insecticide a risk factor for NHL?

A. I believe that -- and I have to recheck on this -- that exposure to herbicides, particularly among, for instance, the veterans who are exposed to Agent Orange, is a risk factor for NHL in some epidemiology studies, but that's a recollection I have. But he certainly had no exposures like that.

Q. And by "exposures like that," you mean that Mr. Carter was not exposed to Agent Orange?

A. Correct.

Q. Is exposure to Roundup a risk factor for NHL?

A. I haven't looked at the latest epidemiology on that. I haven't looked at the latest epidemiology on that.

Q. Do you recall citing Wang 2023 Epidemiology and Etiology of Diffuse Large

B-Cell Lymphoma in your report for Mr. Kidd?

A. Refresh my memory.

Q. That is page 9 of your report for Mr. Kidd, footnote 21. That's Exhibit 3.

A. Okay.

Q. I'm going to hand you Exhibit 22. This is Epidemiology and Etiology of Diffuse Large B-Cell Lymphoma by Sophia S. Wang published in Seminars in Hematology 2023.

(Exhibit Number 22 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. Page 262 of Exhibit 22, so on the top of the second column on this page, the second sentence of that paragraph, "In a pooled analysis."

Do you see where I am?

A. Yes.

Q. In a pooled analysis of three large agricultural worker cohorts including 2,430 NHL cases across the United States, France, and Norway, a positive association was reported between glyphosate and DLBCL, hazard ratio of 1.48, which was further supported in a meta-analysis of seven

studies 1.3 fold DLBCL risk for the highest category of glyphosate exposure.

Did I read that correctly?

A. You did.

Q. Did you consider Mr. Kidd's exposure to Roundup in performing your differential etiology?

A. Well, I took note of that, that he had used Roundup. I don't consider his usage of Roundup as being particularly high. I think most adults of his age, just from common sense, have used Roundup to get rid of weeds, unfortunately. I did.

So I didn't consider that as a, you know, major high-risk factor if he used it ten times in his life, which is what he reported on history.

Q. Okay. So because Mr. Kidd did not use the Roundup product frequently enough, you did not consider it a risk factor in his NHL; is that correct?

A. Well, I didn't -- right. I didn't make note of that as a risk factor. But even if I had and said, well, gee, he's been exposed to some Roundup, it's a very low

level. Maybe it played a role. It certainly wouldn't undercut, again, the risk factor that were posed by the TCE, PCE, and benzene, in my view.

Q. Is it your opinion that Roundup is a risk factor for NHL?

ATTORNEY GREENWALD: Objection to form.

THE WITNESS: I don't have an opinion.

ATTORNEY ANTONUCCI: I'm going to ask again because there was some cross talk, and you can object again.

ATTORNEY GREENWALD: I will.

BY ATTORNEY ANTONUCCI:

Q. Is it your opinion that Roundup is a risk factor for NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I haven't considered it formally, and I don't have an opinion.

BY ATTORNEY ANTONUCCI:

Q. Okay. Please turn to Exhibit 4. That's your specific causation report for

Mr. Carter. I'd appreciate it if you could look at page 13.

So on page 13 on Exhibit 4, paragraph 6, you wrote: Given that MACL is a relatively rare form of NHL, roughly 5 percent, research specific to environmental risk factors for MACL is challenging and few such studies exist. 2008 Swedish case-control study of NHL found infection by Borrelia, the agent that causes lyme disease, was associated with a significantly increased risk of MACL; however, such an association has reportedly not been found in studies of Borrelia infection in the USA, and a review of Mr. Carter's medical records has not uncovered evidence of Borrelia infection.

Did I read that correctly?

A. Yes.

Q. You go on to discuss NHL risk factors, which we previously discussed in the context of Mr. Kidd; correct?

A. Yes.

Q. So the risk factors that you considered for Mr. Carter were his race and

low-level and low-duration radiation exposure; correct?

ATTORNEY GREENWALD: Objection to form.

THE WITNESS: Yes, that's what I wrote later in that paragraph.

BY ATTORNEY ANTONUCCI:

Q. In addition to his exposure to contamination at Camp Lejeune; correct?

A. Correct.

Q. And you ruled out Borrelia -- or did not afford weight to Borrelia infection because there's no evidence of it; is that accurate?

A. That is accurate.

Q. Did you consider family history of a first-degree relative with non-Hodgkin's lymphoma in your differential etiology for Mr. Carter?

A. Well, as I wrote on page 6 of that report, I took a family history, and there was no evidence of someone who had lymphoma, although there was someone who died at the age of 76 of what the family described as some type of blood cancer, but without

having any other specifics, it's hard to know what that really was.

Q. And so you also wrote on page 13 of this report that: Having a family history of a first-degree relative with NHL is a risk factor for which there is evidence of an association with non-Hodgkin's lymphoma; correct?

A. Yes.

Q. Is there evidence of an association between having a family member with any kind of cancer and non-Hodgkin's lymphoma?

A. Not that I'm aware of.

Q. Is there evidence of an association between having a family member with blood cancers and non-Hodgkin's lymphoma?

A. Assuming that these are hematologic cancers versus lymphoma, I'm not aware of it.

Q. So on page 13 -- excuse me, page 14, you cited in footnote 33 the study by Smedby, et al., Epidemiology and Etiology of Mantle Cell Lymphoma; is that correct?

A. Yes.

ATTORNEY ANTONUCCI: I'm going to

fmark that for identification. So this is Exhibit 23, Smedby, et al.

(Exhibit Number 23 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. I'd like you to look at page 296 under Subsection 5, Family History and Genetic Susceptibility, Smedby, et al., state: Family history of hematopoietic malignancies has been linked to a twofold increase of MCL -- excuse me.

It says: Family history of hematopoietic.

Did I read that correctly?

A. You did.

Q. And hematopoietic malignancies means blood cancers; right?

A. Presumably.

Q. Did you consider the fact that Mr. Carter's brother also died of a hematopoietic malignancy in your differential etiology for Mr. Carter?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Without any

confirmatory evidence, death certificate, or anything else, it was hard to know what to make of that.

BY ATTORNEY ANTONUCCI:

Q. Do you normally request a death certificate from patients when you take a family history from them?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Sometimes. But in this case, it's somewhat vague.

BY ATTORNEY ANTONUCCI:

Q. If Mr. Carter's brother had died of a hematopoietic malignancy, that would mean Mr. Carter had a first-degree relative with a hematopoietic malignancy; correct?

A. Correct.

Q. Did you consider the death of Mr. Carter's first-degree relative from a blood cancer as a risk factor?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: It may have been, but if anything, that would have meant he had genetic predisposition that

could have interacted with the chemical exposure to increase his risk even more.

So let me just come back again to my conclusion that none of the risk factors that we talked about undercut the conclusion that TCE, PCE, and benzene, given all the evidence that we just discussed, was also a substantial contributing factor.

BY ATTORNEY ANTONUCCI:

Q. It's true that the causes of mantle cell lymphoma are not known; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I mean, again, it depends on what you mean by causes, but there are risk factors that have been identified, like in this particular review, 14 years ago.

BY ATTORNEY ANTONUCCI:

Q. Are you referring to the Smedby case you cited?

A. Yes.

Q. If you look at page 295 of the

Smedby case, on the first paragraph, the sentence beginning "MCL."

Do you see that?

A. I'm sorry, where on page 295?

Q. Yes. The paragraph above Subsection 4, about halfway through the paragraph there's a sentence beginning with the abbreviation MCL?

A. Oh, yes. I see it.

Q. That sentence says: MCL belongs to the group of NHL subtypes for which the etiology is yet to be explained.

Did I read that correctly?

A. You did.

Q. How did you account for the fact that the etiology of MCL has yet to be explained in determining the cause of Mr. Carter's MCL?

ATTORNEY GREENWALD: Objection to form.

THE WITNESS: This is a general statement, and I don't consider that as necessarily relevant to looking at some of the known risk factors and looking at -- opining on a

relationship between the known risk factor and the causation -- the occurrence of cancer in a particular individual. I just don't see how that's relevant to the exercise we're doing today.

BY ATTORNEY ANTONUCCI:

Q. So on page 7 of your report for Mr. Carter, and that's Exhibit 4.

A. What page?

Q. I might have the wrong page. On page 7, excuse me, of Exhibit 4.

A. Okay.

Q. The last sentence of the first paragraph says: In this job, he, Mr. Carter, was known to have had some occupational exposure to asbestos and to have conducted lead paint testing; is that right?

A. Yes.

Q. Is exposure to asbestos a risk factor for non-Hodgkin's lymphoma?

A. I know that's been looked at, and if it is, the evidence is pretty -- what I would say is weak. I don't recall that.

Certainly isn't one of the major cancers that has been associated with asbestos.

Q. You also noted there that Mr. Carter was known to have conducted lead paint testing.

Is exposure to lead a risk factor for non-Hodgkin's lymphoma?

A. Not that I know of.

Q. And you also noted on page 7 that Mr. Carter maintained a large vegetable garden and used pesticides containing Bifenthrin.

Is exposure to Bifenthrin a risk factor for NHL?

A. Specifically, not that I'm aware of.

Q. You noted on page 14 of Exhibit 4 that Mr. Carter smoked cigarettes; correct?

A. Yes.

Q. How much did Mr. Carter smoke?

A. A pack a day between 1974 to 1992 when he quit.

Q. Do you know how old he was when he started smoking?

A. 26.

Q. Did Mr. Carter ever quit smoking for any period of time?

A. Well, it says here in my history that he quit in 1992 at age 44.

Q. Do you know whether Mr. Carter quit at any period before 1992?

A. It's possible, but I certainly didn't record it in my report.

Q. You noted in your report that epidemiological studies of both mantle cell lymphoma and non-Hodgkin's lymphoma have not identified cigarette smoking as a risk factor for either cancer; correct?

A. That is correct.

Q. And in support of that, you cited to Smedby, et al.

That's Exhibit 23; correct?

A. Yes.

Q. Okay. Please look at page 296 of Exhibit 23.

A. Wait a second.

Q. If you'll look at the second column, the second sentence starting with the words "Body mass index."

Do you see that?

A. Yes.
Q. So here Smedby, et al., wrote: Body mass index, cigarette smoking, and alcohol intake were not implicated as risk factors for MCL, but the statistical power to detect any association was limited due to the relatively small sample size of MCL, in spite of the fact that several studies were pooled for these analyses.
Did I read that correctly?
A. You did.
Q. Does that change your conclusion that cigarette smoke is not a risk factor for MACL?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: No.
BY ATTORNEY ANTONUCCI:
Q. Okay. So I'd like you to turn to Exhibit 5. That's your rebuttal report to Dr. Ambinder for plaintiff Robert Kidd.
A. Okay.
Q. And on page 1 of this report and also for the page 1 of the rebuttal report to Dr. Ambinder for Ronald Carter, you

wrote, quote: My rebuttal report does not contain a response to all of the points that Dr. Ambinder makes in his report with which I disagree, nor should my rebuttal to some portions of his report and not others be viewed as agreement with the portions of his report that I do not rebut below.

A. Right.

Q. Did I read that correctly?

A. You did.

Q. And, Dr. Hu, you're aware that you're required to disclose a complete statement of all the opinions you will express in this case and the basis and reasons for them in your reports; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Yes.

BY ATTORNEY ANTONUCCI:

Q. Okay. On page 1 of this same exhibit, this is, again, Exhibit 5, you quoted Dr. Ambinder where he states he relied on the expert report prepared by Dr. Goodman.

Do you see that?

A. Yes.
Q. And your comment and response was: I refer to my own general causation report on benzene, TCE, and PCE.
Did I read that correctly?
A. You did.
Q. Have you read Dr. Goodman's report titled Trichloroethylene, Perchloroethylene, Benzene, Vinyl Chloride and Trans-1,2-DCE Exposure and NHL risk, dated February 27, 2025?
A. Honestly, I don't recall. If you have it, I'll take a look at it.
Q. I'll note that this is not on any of your materials considered lists.
A. Then I probably did not.
Q. Do you know if Dr. Goodman reviewed any studies that you did not?
A. In her report?
Q. Do you know if Dr. Goodman reviewed any studies in her report that you did not?
A. I wouldn't know.
Q. Do you know if you reviewed any studies in your reports that Dr. Goodman did not review in hers?

A. I don't know.
Q. Do you know what methodology Dr. Goodman employed in reaching her conclusions?
A. Having not read the report, I do not know.
Q. Okay. Is it fair to say that you did not consider Dr. Goodman's report at all in reaching your conclusions on the specific causation of Mr. Kidd and Mr. Carter's diseases?
A. Correct.
Q. So page 2 of Exhibit 5 here, paragraph 3, you note that Dr. Ambinder quoted Wang 2023 for a list of established risk factors for DLBCL; correct?
A. Correct. But I just want to elaborate a little more on the last question, which is, I think, in one of my reports, I discussed Goodman's so-called systemic review of a subject that's relevant to general causation.
So I did not see her report in this litigation, but I have seen and critiqued her report on a general causation topic

that's relevant to this litigation, a Polish report.

Okay. So what's your question this time around?

Q. So page 2 of Exhibit 5, paragraph 3, you note that Dr. Ambinder quoted Wang 2023 for a list of established risk factors for DLBCL; correct?

A. Yes.

Q. And you noted that the abstract of that study states, quote: There is strong evidence for multiple environmental exposures in DLBCL etiology, including exposure to trichloroethylene, benzene, and pesticides and herbicides, with recent associations noted with glyphosate; correct?

A. Yes.

Q. Okay. I would like you to please turn to Exhibit 22. That's the Wang 2023 Epidemiology and Etiology of DLBCL study.

A. This is not the same study as I quoted in the text you just read. The text you just read, I quoted Wang study of Epidemiology and Etiology of Diffuse Large B-Cell Lymphoma.

Exhibit 23 is epidemiology and etiology of mantle cell lymphoma and other NHLs.

Q. I'm sorry. I meant to say Exhibit 22.

A. Oh, okay.

Q. Thanks for the correction.

A. Yeah.

Q. I appreciate that.

A. Okay.

Q. So if you could please look at Exhibit 22, page 261, at the last paragraph under the heading Environmental and Occupational Exposures.

Do you see where I am?

A. Yes.

Q. This reads: Although there is relatively robust evidence linking occupations and environmental exposures to increased risk of hematopoietic malignancies, the evidence that links these exposures specifically to DLBCL remains limited due to the nature of the studies. Much of the epidemiologic data is derived from occupational cohorts that follow

high-exposed populations for years but accrue relatively small numbers of NHLs, of which evaluating NHL subtypes is challenging. Nested case-control studies are also used to directly measure environmental exposures in biospecimens, but due to the expense in cost and biospecimen of measuring exposures, these studies are also typically limited in sample size.

Did I read that correctly?

A. You did.

Q. Wang goes on to state on page 262: Trichloroethylene (TCE) is classified as a probable carcinogen (group 2A) by IARC. A meta-analysis of 14 occupational cohort and four case-control studies linked TCE to elevated NHL risk but lacked NHL subtype data; however, a pooled analysis of 3,788 NHL cases and 4,279 controls within four participating InterLymph Consortium studies evaluated the association between TCE based on occupation categories and NHL risk and found DLBCL risk to be elevated in the highest category of exposure intensity; correct?

A. Correct.
Q. How does that highest category of occupational exposure intensity compare to Mr. Kidd's exposures at Camp Lejeune?
A. Well, let's pull out the study, and let's talk about it.
Q. Are you aware of what the level is sitting here today?
A. Well, I read the study, and I think I quoted it in my general causation report; so I'm aware of it. But I -- you know, sitting here, I can't specifically recall exactly how they defined highest category exposure intensity.
Q. Did you remember at the time you wrote your specific causation report?
A. I mean, I was looking at the study; so sure. You know, this is another example of where, you know, you need to look at these high dose studies in order to address the issue of general causation because if you did a power calculation of the number of people you need to study in order to get a statistically significant increased risk in a study that's properly designed, that would

be -- typically involve many more individuals than is typically possible in a epidemiology study.

So we're often relying on occupational studies to give us a sense of what the general risk is whether it's cancer or other chronic disease outcomes. We used those studies to understand general causation and oftentimes project what the risks are at lower levels of exposure.

THE VIDEOGRAPHER: Can we go off?

We are now going off the record. This is the end of media Unit Number 5, and the time is 4:23 p.m.

(Recess taken from 4:23 p.m. to 4:34 p.m.)

THE VIDEOGRAPHER: We are now back on the record. This is the beginning of media Unit Number 6, and the time is 4:34 p.m.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, we're back on the record.

You understand you're still under oath; correct?

A. Yes.

Q. Did you discuss the substance of your testimony with counsel during the break?

A. No.

Q. Okay. It's your opinion that Mr. Kidd's exposure to TCE, PCE, and benzene from Camp Lejeune increased the risk of and constituted the cause or a substantial contributing cause to his non-Hodgkin's lymphoma; correct?

A. Yes.

Q. Did Mr. Kidd's exposure to contaminated water at Camp Lejeune increase the risk of his NHL, or did it cause his NHL?

A. I would simply say it increased the risk.

Q. Okay. So increasing the risk of something and causing it are two different things; correct?

A. Well, the fact that he got NHL, in my view, ends up being a reason for concluding that the exposures at Camp Lejeune were a substantial contributing factor that caused his NHL.

Q. Increasing the likelihood of disease is different from actually causing such a disease; correct?

A. Of course.

Q. And it's your opinion that Mr. Kidd's exposure to contaminated water at Camp Lejeune did both; is that right?

A. Yes.

Q. How much does the risk of NHL have to be increased by exposure to contamination to say that the exposure was a substantial contributing cause to the NHL?

ATTORNEY GREENWALD: Objection. Form, asked and answered.

THE WITNESS: I mean, I think that that's a specific causation question, and that always requires a in-depth analysis of the specific circumstances of the individual, the cancer, the exposure, timing, et cetera, other potential causes which, as I've said many times in this deposition, is a process that has to be undertaken rigorously and relatively thoroughly, and that's what I believe I performed.

BY ATTORNEY ANTONUCCI:

Q. In your rebuttal to Dr. Ambinder for Mr. Kidd, that is Exhibit 5, you wrote on pages 2 and 3 that Dr. Ambinder ignores the fact that cancer causation is a multi-step and multi-factorial phenomenon; correct?

A. Yes. On page 3, I wrote that: Ignores the fact that as research on the process of carcinogenesis continues to make progress, it has become clear that cancer causation is a multi-step and multi-factorial phenomenon that is heavily influenced by extrinsic (i.e., environmental) factors.

That's my opinion, and it remains so today.

Q. And you wrote that Dr. Ambinder's critique of your discussion of differential etiology essentially implies that position; correct?

ATTORNEY GREENWALD: Objection. Form.

BY ATTORNEY ANTONUCCI:

Q. If you look at page 3 under the

subheading Comment, second paragraph: The second comment by Dr. Ambinder essentially implies two positions.

A. Yeah, no, I understand that. So I'm sorry. Can you repeat your question?

Q. I'd like you to look at Exhibit 9, please. That is your rebuttal report to Dr. Ambinder for Mr. Carter.

A. Okay.

Q. On page 2 of Exhibit 9 in the -- at the very top of the page, you write: I would agree with Dr. Ambinder that Mr. Carter likely had a higher risk of stroke given his family history of myocardial infarctions and strokes; correct?

A. Yes.

Q. Do you know what percentage of non-Hodgkin's lymphoma patients die from strokes?

A. Not as I sit here today.

Q. Do you agree that the vast majority of non-Hodgkin's lymphoma patients do not die from strokes?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: That sounds plausible, but without actually reviewing the evidence, I couldn't say for sure.

BY ATTORNEY ANTONUCCI:

Q. You noted in your report for Mr. Carter that he had a history of coronary artery disease, angina, a myocardial infarction in 2013, and glucose intolerance; correct?

A. I'm sorry. Where are you?

Q. That's going to be in Exhibit 4 on page 6. Excuse me. That's specifically on pages 4 and 5.

A. Okay. Can you repeat that question? Sorry.

Q. You're aware that Mr. Carter had a history of coronary artery disease, angina, myocardial infarction in 2013, and glucose intolerance; right?

A. Yes, I am.

Q. And you're aware that Mr. Carter's father died at the age of 69 of a heart attack; correct?

A. That's what was reported by the

family.

Q. Were you aware the family also reported Mr. Carter's son had two heart attacks and a stroke?

A. I don't recall that and did not record that in my report; so no.

Q. Okay. And it's your opinion that having non-Hodgkin's lymphoma increased Mr. Carter's risk of stroke; correct?

A. Yes.

Q. And that having non-Hodgkin's lymphoma was a substantial contributing factor to Mr. Carter's stroke?

A. I actually did not render a formal opinion on that in either my specific causation expert report or the rebuttal to Dr. Ambinder. I was simply responding to his critique of my report that was -- you know, essentially tried to take issue with my history.

Q. Okay. So it is not an opinion that you're offering in this litigation that having non-Hodgkin's lymphoma was a substantial contributing factor to Mr. Carter's stroke?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, his exposures clearly were a substantial contributing factor to his cancer, and the immediate cause of death on his death certificate was a stroke, and I have discussed how non-Hodgkin's lymphoma is known to be associated with an increased risk of stroke.

So it would be logical to conclude that if that's true, that the exposures at Camp Lejeune indirectly were a cause of his death through the causation of his lymphoma. I didn't explicitly say that in my report, but one could draw that conclusion based on what I've written in my rebuttal to Dr. Ambinder.

BY ATTORNEY ANTONUCCI:

Q. Okay. So yes or no: Are you offering the opinion that Mr. Carter's NHL was a substantial contributing factor to his stroke?

ATTORNEY GREENWALD: Objection.

Form, asked and answered.

THE WITNESS: You know, I'd have to look at my primary sources and review the literature again before I furnish a formal opinion on that.

BY ATTORNEY ANTONUCCI:

Q. Okay. I'm going to reserve the right to hold the deposition open to question you on the causation of Mr. Carter's stroke based on your review of the literature.

I'd like to move on to your rebuttals to Dr. Bailey. That's Exhibits 10 and 11. On page 1 of both Exhibits 10 and 11, I'll give you a moment to find those.

A. Okay. I've got it. 10 and 11. Okay.

Q. Dr. Hu, you agree that the linear no-threshold mechanism of action is applied in regulatory cancer risk evaluations where there is no known threshold dose below which exposure to a carcinogen is not expected to lead to some level of risk even if it is very low; correct?

A. Yes.

Q. And you agree that a threshold model for deriving toxicity criteria is based on the concept that there is some dose below which no adverse effects are expected; right?

A. For some toxicity relationships, that's true.

Q. And the linear no-threshold model likely overestimates exposures at very low doses; correct?

A. What do you mean "overestimates exposures"?

Q. Do you believe it's biologically possible that exposure to one molecule of TCE can increase an individual's risk for developing NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: It's theoretically impossible -- it's theoretically possible but, obviously, unlikely given the multi-hit hypothesis, the multi-cause hypothesis, and the existence of defense mechanisms in the body.

BY ATTORNEY ANTONUCCI:
Q. Do you believe it's biologically plausible that exposure to one molecule of PCE can increase an individual's risk for developing NHL?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: I'm going to go back to my general causation report for a moment. Excuse me.
Okay. Can you repeat the question?
BY ATTORNEY ANTONUCCI:
Q. Do you believe it's biologically plausible that exposure to one molecule of PCE can increase an individual's risk for developing NHL?
A. It's theoretically possible but, obviously, unlikely for the reasons I mentioned for the other scenario you posed.
Q. Do you believe it is biologically plausible that exposure to one molecule of benzene can increase an individual's risk for developing NHL?
ATTORNEY GREENWALD: Objection.

Form.
THE WITNESS: Same response.
BY ATTORNEY ANTONUCCI:
Q. So, in general, you agree that the human body is, at least in some circumstances, able to repair DNA damage; correct?
A. Yes.
Q. And you agree that the body's normal defense mechanisms can eliminate low concentrations of a mutagenic or carcinogenic substance and repair DNA damage that exposure to that substance may have caused; right?
A. In certainly the majority of cases but not necessarily all.
Q. Is DNA damage a cause of non-Hodgkin's lymphoma?
ATTORNEY GREENWALD: Objection.
Form.
THE WITNESS: I believe it is part of the intrinsic mechanism by which NHL is caused in many cases.
BY ATTORNEY ANTONUCCI:
Q. And, Dr. Hu, you conclude that PCE

causes -- exposure to PCE causes NHL through genotoxicity based on evidence of PCE's genotoxicity and peripheral blood lymphocytes; right?

A. I think that's one of the mechanisms that may be at play, yes.

Q. Peripheral blood lymphocytes are not involved in the development of NHL; correct?

A. Probably not. But they are, I guess you could say, a reflection of what may be happening in the bone marrow or in lymph nodes.

Q. You also conclude that PCE causes NHL through genotoxicity based on evidence of PCE's genotoxicity in leukocytes; right?

A. Right.

Q. Leukocytes are not involved in the development of NHL; correct?

A. No. Those are from the adaptive immune response, but they are also a reflection of what might be happening in cells of that origin.

Q. So what is your -- what is your methodology for concluding that -- that PCE

causes NHL based on evidence of genotoxicity in cells unrelated to the development of NHL like peripheral lymphocytes and leukocytes?

A. To the extent that it's a reflection of what's happening in the bone marrow itself and in lymph nodes and other lymph-containing organs.

Q. Is there any evidence that PCE causes genotoxicity on the cells related to the development of NHL?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: There may be some in vitro studies that have been done, but I'd have to go back and look at the scientific basis -- scientific evidence basis to answer that question accurately.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, you're aware that there's a debate in the scientific community regarding the linear no-threshold model; correct?

A. Yes.

Q. Is it your belief that there is a consensus in the scientific community around

the linear no-threshold model?

A. Well, it depends how you define consensus, but I think a majority of, I guess I would say, mainstream environmental epidemiologists and my colleagues would consider the linear no-threshold model as still operative.

Q. All right. On Exhibit 10, that's your rebuttal to Dr. Bailey for Mr. Kidd. At the bottom of page 3, last paragraph starting with the word "Finally."

Are you with me?

A. Yes.

Q. You write: Finally, despite these limitations, I point out there have been several key epidemiological studies that have leveraged extraordinarily large sample sizes to provide evidence of the cancer risk at very low levels of exposure to chemical carcinogens; correct?

A. Yes.

Q. And going on to page 4, you cite to Wang 2024, Long-Term Exposure to Low Concentrations of Ambient Benzene and Mortality in a National English Cohort;

correct?

A. Correct.

Q. And to Yu 2025, Long-Term Exposure to Low-Level Ambient BTEX and Site-Specific Cancer Risk: A National Cohort Study in the UK Biobank as examples of such studies; correct?

A. Yes.

Q. Are there any other studies you're aware of that have leveraged large sample sizes to provide evidence of the risk of cancer at very low levels of exposure to chemical carcinogens that you're aware of?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Not this large, no.

BY ATTORNEY ANTONUCCI:

Q. Okay. On page 4 of your rebuttal to Dr. Bailey for Mr. Kidd -- and, again, that is Exhibit 10 -- you wrote in the second paragraph about halfway through the paragraph starting with "Of note."

You wrote: Of note is that, according to EPA toxicokinetic models, inhalation of air contaminated with benzene

at a level of 0.18 parts per billion benzene would give rise to the same internal dose (i.e., level of benzene in blood) as ingesting drinking water with benzene at a level of 4.5 parts per billion. As noted in my report on Mr. Kidd, the exposure assessment by Dr. Reynolds resulted in an estimated time-weighted average exposure for Mr. Kidd of 9.6 parts per billion, which is over twice the level of benzene at which point the Yu, et al., study found direct epidemiological evidence of the risk of cancer increasing.

Did I read that correctly?

A. You did.

Q. Dr. Hu, how did you determine that the inhalation of air contaminated with benzene at a level of 0.18 parts per billion would give rise to the same internal dose for a level of benzene in blood as ingesting drinking water with benzene at a level of 4.5 parts per billion?

A. I consulted an EPA toxicokinetic model exercise.

Q. You ran an EPA toxicokinetic model

to determine the 4.5 parts per billion value that you cite here on page 4 of Exhibit 10?

A. Yes.

Q. Which one?

A. I didn't cite it here, but I'd have to go back to my computer to figure it out.

Q. Do you remember what it's called?

A. Oh, God. No.

Q. How did you decide which toxicokinetic model to run?

A. This is a very simple model. It was a conversion model that went from air modeling to what the likely level would be in a person's blood given some basic parameters of -- I think it was a 75-kilogram male breathing this concentration of benzene for a certain amount of time, and there was some other conditions as well. That's what I recall.

Q. Did you review EPA's risk summary for the derivation of toxicity values?

A. Which publication are you talking about?

Q. Did you perform -- or did you review an EPA risk summary that included

simple route-to-route extrapolation?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I think that's exactly what I consulted.

BY ATTORNEY ANTONUCCI:

Q. Okay.

A. Right.

Q. So is that actually a toxicokinetic model, or is it --

A. Well, it's based on a toxicokinetic model.

Q. Okay. And you mentioned that some of the inputs to your model were the 75-kilogram male --

A. That's not my model. Their model.

Q. Sure.

Some of the inputs to route-to-route extrapolation were the individual 75-kilogram male; correct?

A. That's what I recall, but don't quote me on that. I don't remember the exact conditions that they leveraged.

Q. You input the amount of benzene inhaled in the air, the concentration of

benzene inhaled; correct?

A. That's correct.

Q. Did you input the amount of time which the benzene is inhaled over?

A. I don't think the model -- at least the model that I consulted asked for that. This is a steady-stated model that they had where you could simply put in the air and get out what the blood was. They have assumptions on how long the inhalation was for.

Q. I see.

A. It's not like that was a variable that you had to put into the model.

Q. Do you know what that assumption was for how long the inhalation was?

A. It's like all the things we're talking about here. I don't specifically recall what those assumptions are.

Q. And you mentioned there were other conditions which bear on the outputs of the model; correct?

A. It's a vague memory. I don't remember exactly what their assumptions were. I'm sorry.

Q. And you don't remember the other conditions that --

A. No.

Q. Okay. Did you check any literature sources to decide which model to select?

A. Well, since it was the EPA, and this is their job, I relied on the EPA for that derivation.

Q. Okay. And did you document the process of running the model anywhere?

A. I may have it on my computer, and I'm sorry I didn't put a footnote in here like I usually do for that.

Q. If you did have that file on your computer, would you be able to produce it to counsel?

A. I can certainly do my best.

ATTORNEY ANTONUCCI: We're going to request, after this deposition in a formal request, that we receive those files.

THE WITNESS: That's fine.

ATTORNEY GREENWALD: We'll get a letter. Don't worry. If you want to make your own note, go ahead.

THE WITNESS: I'm going to make my own note and stick it in my pocket.

BY ATTORNEY ANTONUCCI:

Q. Did the toxicokinetic model that you consulted provide you with a confidence interval for the 4.5 parts per billion value generated?

A. I don't think so. It just churned out a number.

Q. Did it provide you with a P value for the 4.5 parts per billion value it generated?

A. That's a research metric, not a model metric; so no, it did not.

Q. What's your level of confidence in the results of this toxicokinetic model you ran?

A. Well, I mean, it's the best estimate, I think, that's available from one of the best potential sources for generating those kinds of estimates; so my degree of confidence is high.

Q. Okay. And the inhalation value of 0.18 parts per billion benzene was taken from the Wang and Yu studies; correct?

A. Yes.
Q. And so here you're comparing the inhalation of 0.18 parts per billion benzene in a long-term study with Mr. Kidd's exposure; correct?
A. That's correct.
Q. Mr. Kidd was on base for about 17 months; correct?
A. Yes.
Q. Do you know how long the exposure duration of the Wang and Yu studies was?
A. Wait. Did you say 17 months?
Q. Yes.
A. I just want to make sure that was right. Okay. Yes, that was correct.
So what was your question again? I'm sorry.
Q. Dr. Reynolds stated that Mr. Kidd was on base for about 17 months; correct?
A. Yes.
Q. Okay. And so with this 4.5 parts per billion value that you created, you're comparing the results of the long-term Wang and Yu studies with 17 months of exposure for Mr. Kidd; correct?

A. That's correct.
Q. You're also comparing a population study with exposure for one individual; correct?
A. Yes.
Q. Wang in 2024 and Yu in 2025 evaluated the same cohort; correct?
A. Yes.
Q. And that was using the U.K. Biobank?
A. Yes.
Q. Okay. I'm going to provide you with the Wang study. This is Exhibit 24.
(Exhibit Number 24 was marked for identification.)
BY ATTORNEY ANTONUCCI:
Q. I'd appreciate if you could please turn to page 993. If you look in the center column, it's the third paragraph starting with "However."
Do you see where I am?
A. Yes.
Q. Wang 2024 write: However, the study also has certain limitations that must be acknowledged. First, this observational

study could not fully control for all unknown or unmeasured confounding factors and was unable to demonstrate a causal relationship between benzene and mortality.

Did I read that correctly?

A. You did.

Q. Now I'm going to provide you with Yu 2025, and that will be Exhibit 25.

(Exhibit Number 25 was marked for identification.)

BY ATTORNEY ANTONUCCI:

Q. The Yu 2025 study titled Long-Term Exposure to Low-Level Ambient BTEX and Site-Specific Cancer Risk: A National Cohort Study in the UK Biobank focuses on low-level ambient exposure to BTEX; right?

A. Yes.

Q. Meaning it studied lower exposure doses than an occupational study would; correct?

A. I would say that's -- yes.

Q. It also evaluates long-term exposures; correct?

A. Yes.

Q. Do you know how long?

A. Well, the study's methodology section notes that their exposure data come from measurements taken from 2006 to 2021; however, in their paper, they do not actually go over the results of that testing for benzene, toluene, and xylene during that time. So it's impossible to know whether the exposures were meaningful in 2006 or whether they were low or high or what the trend was, and I think you'd have to go to some of the other information they've published to answer your question accurately.

Q. What other information would you have to look at? Is that the Wang study?

A. Well, they said in their first sentence the annual mean levels of benzene, toluene, xylenes from 2006 to 2021 were derived from a three-dimensional chemistry-climate model, and then they cited reference 16 through 18. And it's kind of hard to tell whether those are references that might have gone over the data on exposure.

So it just -- I can't answer your

question based on the information provided in this paper.

Q. Okay. Do you know if the Yu 2025 study controls for co-exposures?

A. I mean, they have -- because it's a cohort study and it's UK Biobank, they do have data, as they mentioned, in term of covariates, demographic, socioeconomic, lifestyle, health-related information. Passive smoking, smoking, exposure to PM2.5, nitrogen dioxides.

So they have quite a bit of additional individual information among the covariates that they have to control for, which they did.

Q. Do you know if the Yu 2025 study controls for confounding factors?

A. Well, everything that I mentioned could potentially be a confounder.

Q. It's true that Yu 2025 could not rule out residual confounding by unmeasured factors that could affect exposure and cancer incidents; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: That is an acknowledgment they made, and that's an acknowledgment every epidemiologist has to make in just about every epidemiology study. It's impossible to control for absolutely everything.

BY ATTORNEY ANTONUCCI:

Q. Okay. The Yu 2025 study does not present data on NHL subtypes; correct?

I would direct you to page 4 for the Figure 1 Associations of Long-Term Exposures to BTEX with Risk of Overall and Site-Specific Cancer.

A. Do you have a magnifying glass? Okay. I can make out some of it. Hold on a moment.

Yeah, they just list non-Hodgkin's lymphoma but not the subtypes.

Q. Okay. And Yu 2025 is an ecological study; correct?

A. It's a what?

Q. Ecological study; correct?

A. No, it's not.

Q. Can you explain why Yu 2025 is not an ecological study?

A. Because they have individual level data on the subjects. Yes, the exposure data is extrapolated based on the claimant model that they used, but that's a very common technique for doing any air pollution type of epidemiology. But the fact that this is a cohort that's very well characterized and followed over years and they have individual level data on all these other potential confounders, in my book, that is a perspective cohort study.

Q. The Yu 2025 study estimates population-based exposure information; correct?

A. That's correct.

Q. Okay. And that's, again, based on a climate model; right?

A. Yes.

Q. Population-based exposure information was not measured in the Yu 2025 study; correct?

A. I think you just contradicted yourself. Say that again.

Q. If the information was estimated by the model, it wasn't measured by air

sampling; correct?

A. Oh, they did plenty of air sampling, but then they used the air sampling using a geospatial model to estimate what the individual level concentrations were. They didn't just pluck these, you know, estimated air pollution measures out of the air and just try to model what they were based on nothing. They actually had measurements.

Q. Okay. On page 5, the paragraph above Subsection 5, Conclusion, the sentence starting with "The exposure assessment."

This reads: The exposure assessment based on residential addresses 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 incidence. 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. You did.

Q. The study authors are saying here that there's potential for exposure misclassification based on their exposure assessment; correct?

A. Correct.

Q. And Yu used a climate -- Yu 2025 used a climate model that predicted atmospheric benzene concentrations in boxes with horizontal dimensions of about 100 kilometers by 80 kilometers; right?

A. Yes.

Q. They then assigned average benzene concentrations to study participants with residential addresses at a 1-kilometer-by-1-kilometer resolution; right?

A. Yes.

Q. The 1-kilometer-by-1-kilometer resolution was superimposed on the

10-kilometer-by-80-kilometer boxes that their model created; right?

A. Correct.

Q. And the study authors compared modeled BTEX concentrations with on-site measurements from European countries, and the comparisons showed a normalized mean bias of plus or minus 25 percent; right?

A. Yes.

Q. What is normalized mean bias?

A. Read that sentence again.

Q. So I'm looking at 2.2, Exposure Assessment, on page 2: We compared modeled BTEX concentrations with on-site measurements from European countries, and the comparison showed a normalized mean bias of plus or minus 25 percent.

A. Yeah, so they -- okay. So what they did here is that they used their modeling methodology to model BTEX concentrations in European countries where there are actual on-site measurements in the locations where they used the BTEX modeling to model it.

Then in order to compare them and

not compare apples to oranges, they normalized, in other words, they standardized the measurements between the two geographic locations so that they're in the same metric, and then when they're on the same metric, now they can calculate the coefficient variation, which is essentially this calculation of plus or minus 25 percent which is that -- you know, the precision of their estimates compared to actual measurements was about plus or minus 25 percent. That's the meaning of that sentence.

Q. And that's an average; correct?

A. Yes.

Q. And so they tested their bias in European countries rather than in the UK; correct?

A. Well, I mean, it's -- you know, it's testing a methodology. It's not so much the dependent on the actual measurements of pollutants in each of these countries. They're testing the measurement methodology approach. Let's put it that way.

Q. Isn't the normalized mean bias a measure of its actual ability to predict BTEX values?

A. Yeah, it's, like I said, a measurement of the coefficient of variation. It's like you want to take a measurement of something, and the true measurement is 10. Sometimes you get 9 or 8 or 11 or 12. But on average, it comes out to be about a 10; so there's no bias, but there is what is known as noise, and that's also known as non-differential misclassification.

Q. So the normalized mean bias has a value of plus or minus 25 percent.

What does it mean for the mean bias to be plus or minus 25 percent?

A. It means the true value might be a little bit higher, might be a little bit lower.

Q. So plus or minus means approximately?

A. Yeah.

Q. Okay. And I believe you just testified that the Yu study has the potential for differential bias; correct?

A. I don't think I --

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I don't think I testified that. Differential bias?

BY ATTORNEY ANTONUCCI:

Q. I'm sorry. Did you say non-differential bias?

A. Non-differential bias.

Q. Pardon me.

So you testified that the Yu study has the potential for non-differential bias; correct?

A. Non-differential misclassification in terms of exposure, yes.

Q. Okay. And is that based on classifying exposures based on residences and not occupations?

A. I mean, it's based on these using modeling and air pollution measurements to estimate what the exposures were for a population where they -- as the authors themselves noted, they didn't have indoor measurements. They didn't know how much people actually worked over here but resided

over there for how many hours of the day.

Those are all the kinds of things that happen in exposure measurement limitations that can end up, as I mentioned before, diluting the apparent effect of the exposure you're trying to study.

Q. So if the methodology dilutes the apparent effect of the exposure we're studying, that would mean that Yu is probably reporting artificially low associations at high exposures; correct?

A. Well, these are all low exposures. None of them -- these are environmental exposures that are residential community, et cetera. So the fact that there's non-differential misclassification, as I mentioned earlier in this deposition, typically means that the effect estimate is an underestimate of the true magnitude of the association between the exposure of interest, in this case benzene, and the outcome of interest, risk of cancer.

Q. So conversely accepting as true that benzene causes NHL, people who got NHL because they were exposed to high doses of

benzene will probably be shown in Yu as having been exposed to lower doses than they were really exposed to because of the dilution effect; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Whoa. That made no sense to me. Can you repeat the question.

BY ATTORNEY ANTONUCCI:

Q. Sure. We're discussing the effect of dilution on the exposure classifications --

A. That's an epidemiology concept. That's pertinent to interpreting the results of an epidemiology study, not a, you know, specific causation analysis.

Q. Dr. Hu, I know you probably didn't mean to, but please allow me to finish my question.

A. Okay. Sorry.

Q. So the people who were exposed to high doses of benzene may be modeled as having been exposed to low doses because of dilution; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Correct. And the other way around. Some people who had low exposures may have actually had higher exposures.

BY ATTORNEY ANTONUCCI:

Q. That means that the Yu study had potential for differential bias too; correct?

A. No. No. What we just discussed is non-differential bias. That is, high may go to low; low may go to high. But there's no reason to suspect that oh, in this study, the ones who were -- had, for instance, higher cancer rates were much more likely to have been estimated to have higher exposures. You know, that -- on a differential basis, that's not true.

These are -- whatever misclassification there is, is kind of random because you don't have data on who moved or, you know, whether air pollution is a little bit higher in one place than the other, et cetera, et cetera.

So non-differential misclassification is what they're talking about here. I don't think anybody is talking about differential misclassification.

Q. The Yu study found a positive association between every cancer and every contaminant that it evaluated; correct?

And for that, you can look to page 4, Figure 1.

A. Yes. For each of the contaminants and for each of these cancer outcomes, except for a few, the point estimates of risk seem to be above 1, and the 95 percent confidence, the lower bound of the 95 percent confidence limit seem to be above 1 for the majority of the cancers.

Q. Are there any cancers that the Yu study evaluated that did not find a hazard ratio of above 1?

A. So they selected a number of cancer sites which they called major cancer sites for this analysis. They didn't look at all cancers.

Q. However, for all of the cancers

they evaluated, they found an increased hazard ratio; correct?

A. That's what I'm trying to figure out, whether this actually is a listing of all the cancers they looked at, or whether it's just the listing of the cancers for which the risks exceeded 1. I'm not sure. I'm just trying to discern that from the paper.

Well, they didn't say explicitly so, but my assumption from reading the paper is that they selected a bunch of cancers that they identified as major cancers, and all of the results, it seems, were presented in Figure 1. And in Figure 1, it does appear that all of the risks exceeded 1, and many of them exceeded 1 with the lower bound confidence interval exceeding 1 as well.

Q. Okay. On page 3 of Exhibit 10, which is your rebuttal to Dr. Bailey for Mr. Kidd.

A. Yes.

Q. Here you cite the Bove, et al., 2024 cancer incident study as evidence that there's direct epidemiological evidence of

risk at very low levels of carcinogen exposure; correct?

A. Correct.

Q. Do you believe that individuals stationed at Camp Lejeune between 1953 and 1987 were exposed to very low levels of carcinogen exposure?

A. Well, I mean, low is a qualitative term, but they certainly weren't exposed to the kind of, what I would say, high levels of these chemicals as would happen in people who were occupationally exposed to those particular solvents, trichloroethylene, perchloroethylene, and benzene.

Q. You specifically reference, on pages 4 and 5, the Bove, et al., cancer incident studies adjust hazard ratios for myeloid cancers, acute myeloid leukemia, myelodysplastic and myeloproliferative syndromes; correct?

A. Yes. Those are the ones I discussed in that paragraph where they found elevated adjusted hazard ratios.

Q. If you will please turn to Exhibit 11, that's your specific causation

rebuttal report for Dr. Bailey for Mr. Carter.

A. Okay. What page?

Q. Page 5 of Exhibit 11.

A. Okay.

Q. You commented on Dr. Bailey's statement that there's a high likelihood of exposure misclassification in ATSDR's Camp Lejeune studies; right?

A. Yes.

Q. And you wrote that misclassification is common in epidemiological studies; right?

A. Yes.

Q. And you wrote that: However, unless there's reason to suspect a differential exposure misclassification that would introduce a specific bias that increases the likelihood of finding an association (where none truly exists), non-differential (i.e., random) exposure misclassification typically results in a bias to the null, i.e., a dilution of any apparent effect.

Did I read that correctly?

A. You did.
Q. Do you have any reason to believe the exposure misclassification in ATSDR's Camp Lejeune studies was non-differential?
A. Well, it's the other way around. There's no evidence that the exposure misclassification was differential. In the absence of that, any evidence to show that there's differential exposure misclassification, then the typical exposure misclassification that occurs in a setting like this is non-differential.
Q. If Dr. Bove's 2024 cancer incident study used exposure doses that were uniformly 10 percent higher than reality across all exposure classifications, that would constitute a non-differential misclassification bias; correct?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: No. No. Because non-differential misclassification has to -- that affects the results of a study. In terms of general causation has to be differential between those

who develop cancer and these who didn't, and that's how you can get a false impression of a elevated risk of cancer. But if the overall estimates of exposure are just 10 percent higher, 10 percent lower, it's 10 percent higher or lower for everybody, you know, whether they develop cancer or not, and that would not, theoretically, lead to a type of error that would create a false impression of an association between exposure and disease.

BY ATTORNEY ANTONUCCI:

Q. If Dr. Bove's study used exposure doses where some were higher than in reality and some were lower in random proportions, that would constitute a differential misclassification bias; correct?

A. No.

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I'm sorry.

ATTORNEY GREENWALD: That's okay.

THE WITNESS: No, because, like

you just said, it's random. So it's random; it's not differential.

BY ATTORNEY ANTONUCCI:

Q. If they were not random or differentiated in that some doses were higher than in reality and some were lower than in reality, that would constitute differential misclassification; right?

A. Only if there was a systematic bias where people who had cancer had a different exposure measurement based on the methodology used than the ones who didn't develop cancer.

Q. Do you have an opinion on whether ATSDR's water models calculated accurate historical concentrations of contaminants in drinking water?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: All I know is that I'm not aware of any better model.

BY ATTORNEY ANTONUCCI:

Q. Can you accurately assess the misclassification bias of Dr. Bove's studies without understanding the accuracy of

ATSDR's water models?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I can't estimate that, but like I said, what's most important from a general causation point of view is whether there's not -- where there's differential classification and that -- I've not seen any evidence for that.

ATTORNEY ANTONUCCI: Okay. I'd like to take one final break.

THE VIDEOGRAPHER: We are now going off the record. The time is 5:38 p.m.

(Recess taken from 5:38 p.m. to 5:50 p.m.)

THE VIDEOGRAPHER: We are now back on the record. The time is 5:50 p.m.

BY ATTORNEY ANTONUCCI:

Q. Dr. Hu, we're back on the record. You understand you're still under oath; correct?

A. Yes.

Q. Did you discuss the substance of

your testimony during the break?
A. No.
Q. Dr. Hu, has the hypothesis that benzene causes all NHLs because it's associated with pluripotential stem and cell mutation being tested?
ATTORNEY GREENWALD: Objection. Form.
THE WITNESS: Tested. I think you were referring to research that would either take the form of epidemiology, which would be very difficult to do unless you did bone marrow biopsies on people and followed them prospectively over time, or some type of experimental toxicology study, and I'm not aware of any such research that exists.
BY ATTORNEY ANTONUCCI:
Q. Dr. Hu, have your opinions as an expert witness ever been excluded by a judge?
A. No.
Q. Have portions of your opinions as an expert witness ever been excluded by a

judge?
A. Not that I'm aware of.
Q. Dr. Hu, are you aware that you were once excluded from offering testimony that you had diagnosed a plaintiff with multiple chemical sensitivity?
A. What year was that?
Q. 2000. Do you recall Anello versus Shaw Industries, Inc.?
A. Nope. That's 25 years ago.
Q. Your opinions have been excluded in more recent cases too, though, haven't they?
A. You'd have to refresh my memory because I'm not aware of that.
Q. Do you recall testifying in the case Butler versus Mallinckrodt, LLC, in 2022 Eastern District of Missouri?
A. Yes.
Q. Your opinions were excluded in that case, were they not?
A. I believe that the opinion of the exposure assessment expert I relied on was excluded, but my opinion was not excluded based on my science but rather on the fact that I relied on his testimony.

Q. Okay. And also McMunn versus Babock and Wilcox Power Generation Group, Inc., in 2013.

Do you recall that case?

A. I think it was a very similar case with the same exposure assessment expert.

Q. Okay. Are there any answers you've given to my questions during this deposition that you wish to change before we end it?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: No.

BY ATTORNEY ANTONUCCI:

Q. Is there any information I asked about that you didn't recall at the time but now remember?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I'm sorry. What was that question again.

BY ATTORNEY ANTONUCCI:

Q. Is there any information I asked about that you didn't recall at the time but now remember?

A. Not that I recall.

Q. Do you have any other opinions that you've not testified to today?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Not that are relevant to this case, as far as I know.

BY ATTORNEY ANTONUCCI:

Q. Once it's ready, you'll be provided with the transcript of this deposition. I ask that you carefully read, correct, and sign the transcript.

Do you understand?

A. I do.

Q. Thank you for your patience in answering my questions today, Dr. Hu.

ATTORNEY ANTONUCCI: I pass the witness.

ATTORNEY GREENWALD: We're going to take a little break.

THE VIDEOGRAPHER: We're now going off the record. The time is 5:54 p.m.

(Recess taken from 5:54 p.m. to 6:11 p.m.)

THE VIDEOGRAPHER: We are now back

on the record. The time is 6:11 p.m.

EXAMINATION

BY ATTORNEY GREENWALD:

Q. Dr. Hu, I just have a couple of questions to ask you, and it relates to the questions that counsel was asking you about Mr. Carter's stroke. Okay?

A. Yes.

Q. Do you have an opinion whether the diagnosis of non-Hodgkin's lymphoma increased Mr. Carter's risk of having a stroke?

A. Yes.

Q. And what is that opinion?

A. It does increase the risk of stroke.

Q. Okay.

A. Including Mr. Carter's stroke.

Q. Okay. Can you go to your expert report, your original report, your specific causation, not the rebuttal, which is Exhibit 4, I believe.

A. Yes.

Q. And go to page 14, please.

Are you there?

A. Yes.

Q. And you say in the sentence above the stars: Thus, given my general causation assessment and the factors reviewed above, it is my opinion to a reasonable degree of medical certainty that the combination of Mr. Carter's exposure to TCE, PCE, and benzene from Camp Lejeune more likely than not was a substantial contributing factor to the causation of his mantle cell lymphoma.

And that is still your opinion today; correct?

A. Correct.

Q. You go on under those lines to say: This concludes my evaluation of Mr. Ronald Carter with respect to the relationship between exposures of TCE, PCE, and benzene that he experienced from Camp Lejeune and the mantle cell lymphoma that he developed (and that was the proximate cause of his death).

Do you see that?

A. Yes.

Q. Do you still hold the opinion that

Mr. Carter's exposure to TCE, PCE, and benzene at Camp Lejeune and his mantle cell lymphoma is the proximate cause of his death?

A. Yes.

ATTORNEY GREENWALD: I don't have any other questions.

FURTHER EXAMINATION

BY ATTORNEY ANTONUCCI:

Q. Really brief redirect.

First of all, Dr. Hu, since we've come back on the record, you understand that you're still under oath; correct?

A. Yes.

Q. Did you discuss the substance of your testimony with counsel during the break?

A. I did.

Q. What you did you discuss?

ATTORNEY GREENWALD: I'm not letting him testify.

I'm instructing you not to answer. That's privileged communication.

We've gone over this with your

lawyers before. You can take it up with the judge if you like.

BY ATTORNEY ANTONUCCI:

Q. Are you going to listen to your attorney's instruction not to answer my question?

A. I will.

Q. Okay. I am going to hold the deposition open for the purpose of determining what you discussed during the last break of this deposition under 4th Circuit law.

Dr. Hu, you testified that it is now your opinion that Mr. Carter's stroke -- the risk of Mr. Carter's stroke was increased by his non-Hodgkin's lymphoma; correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I never testified that wasn't true.

BY ATTORNEY ANTONUCCI:

Q. When I asked earlier whether you were offering that opinion, you said you had to review the literature in order to offer

an opinion on that.

What's changed?

A. What's changed is the level of certainty. I mean, I quoted two scientific studies in my report -- or this is actually my rebuttal to Dr. Ambinder. And when I was reminded of those studies and their rigor, one was a national study looking at cancers and risk of stroke that included specifically NHL, I was persuaded that, in fact, that's a level of evidence that I could rely on.

Q. And you reviewed that study during the break?

A. I was reminded of it when I reread the report and et cetera.

Q. Okay. So what is proximate cause?

A. Proximate cause is essentially the disease process that likely was essentially the most impactful influence on whatever the outcome you're trying to look at.

Q. Are you aware that proximate cause is a legal term of art?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: Well, I'm just using my understanding of how we use it in medicine.

BY ATTORNEY ANTONUCCI:

Q. Okay. So on page 14 of your report for Mr. Carter, when you said that the relationship between exposures to TCE, PCE, and benzene that he experienced from Camp Lejeune and the mantle cell lymphoma that he developed, and that was the proximate cause of his death, you do not mean proximate cause in the legal sense; is that correct?

ATTORNEY GREENWALD: Objection. Form.

THE WITNESS: I don't even know what the proximate cause means in a legal sense. I just used it from my understanding of how we use it in medicine.

ATTORNEY ANTONUCCI: Okay. No further questions.

ATTORNEY GREENWALD: I don't either.

THE VIDEOGRAPHER: We are off the record at 6:16 p.m., and this

concludes today's testimony given by Dr. Howard Hu. The total number of media used was six and will be retained by Golkow, a Veritext division.

THE CERTIFIED STENOGRAPHER: Ms. Greenwald, do you need to order a rough draft?

ATTORNEY GREENWALD: No.

(Whereupon the deposition concluded at 6:16 p.m.)

REPORTER'S CERTIFICATE

The undersigned Certified Shorthand Reporter licensed in the states of California, Nevada, Illinois, and Washington does hereby certify:

That the foregoing deposition was taken before me at the time and place therein set forth, at which time the witness was duly sworn by me;

That the testimony of the witness and all objections made at the time of the examination were recorded stenographically by me and were thereafter transcribed, said transcript being a true copy of my shorthand notes thereof;

That if this is a Federal case, a request [ ] was [ ] was not made to read and correct said deposition.

I further declare that I have no interest in the outcome of the action.

In witness whereof, I have subscribed my name this 5th day of August, 2025.

_____

LISA MOSKOWITZ
California CSR 10816, RPR, CRR, CLR
Washington CCR 21001437, Nevada CCR 991,
Illinois CSR 084.004982

INSTRUCTIONS TO WITNESS

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

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

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

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

E R R A T A S H E E T

- - - - - -

PAGE LINE CHANGE

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

_____ _____ _____

REASON: _____

ACKNOWLEDGMENT OF DEPONENT

I, HOWARD HU, PH.D., do hereby certify that I have read the foregoing pages, 1-321, and that the same is a correct transcription of the answers given by me to the questions therein propounded, except for the corrections or changes in form or substance, if any, noted in the attached Errata Sheet.

_____ _____

HOWARD HU, PH.D. DATE

LAWYER'S NOTES

PAGE LINE

_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________
_____ _____ ________________________

b

l

o