

Exhibit 602

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

IN RE: CAMP LEJEUNE WATER)
LITIGATION,)

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)

This Document Relates To:) No. 7:23-CV-897
)

ALL CASES)
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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

1 APPEARANCES:

2
3 On behalf of Plaintiff:

4 WEITZ & LUXENBERG

BY: ROBIN L. GREENWALD

5 rgreenwald@weitzlux.com

1880 Century Park East

6 Suite 700

Los Angeles, California 90067

7 (310) 247-0921

8
9 On behalf of Defendant:

10 U.S. DEPARTMENT OF JUSTICE

BY: GIOVANNI ANTONUCCI

11 giovanni.antonucci@usdoj.gov

BY: AMY KLIMEK

12 amy.a.klimek@usdoj.gov

1100 L Street NW

13 Washington, DC 20005

(202) 514-2000

14
15 ALSO PRESENT VIA ZOOM:

16 ALLISON O'LEARY

17 MATTHEW ELLIOTT

MARCUS TUBIN

18 DIANA GJONAJ

19 VIDEOGRAPHER:

20 TORR PIZZILLO,

21 GOLKOW TECHNOLOGIES

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	19		Cancer Risk and Tetrachloroethylene Contaminated Drinking Water in Massachusetts by Aschengrau, et al.																					
	20		Drinking Water Contamination and the Incidence of Leukemia and Non-Hodgkin's Lymphoma by Perry Cohn, et al.																					
	21		American Cancer Society's website titled Non-Hodgkin's Lymphoma Causes, Risk Factors, and Prevention																					
	22		Epidemiology and Etiology of Diffuse Large B-Cell Lymphoma by Sophia S. Wang																					
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1 WEDNESDAY, JULY 23, 2025

2 LOS ANGELES, CALIFORNIA

3 9:10 A.M.

4
5 THE VIDEOGRAPHER: We are now on
6 the record. My name is Torr Pizzello.
7 I'm a videographer for Golkow, a
8 Veritext division. Today's date is
9 July 23, 2025, and the time is
10 9:10 a.m. This video deposition is
11 being held at 1880 Century Park East,
12 Los Angeles, California in re Camp
13 Lejeune Water Litigation versus United
14 States of America for the U.S.
15 District Court for the Eastern
16 District of North Carolina, Southern
17 Division. The deponent is Dr. Howard
18 Hu.

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

24 THE CERTIFIED STENOGRAPHER: My
25 00:-2625 name is Lisa Moskowitz. I'm a

1 1 California certified stenographic
2 2 reporter. My CSR license number is
3 3 10816.
4 4

5 00:-26 5 HOWARD HU, PH.D.,
6 6 called as a witness,
7 7 was examined and testified as follows:
8 8

9 9 EXAMINATION

10 10 BY ATTORNEY ANTONUCCI:

11 11 Q. Good morning.

12 12 A. Good morning.

13 13 Q. Could you please state your full
14 14 name for the record?

15 15 A. Howard Hu.

16 16 Q. Could you please state your current
17 17 business address?

18 18 A. 1845 North Soto Street, Los
19 19 Angeles, California 90089.

20 20 Q. Dr. Hu, my name is Giovanni
21 21 Antonucci. I'm an attorney for the
22 22 Department of Justice. I represent the
23 23 United States in the Camp Lejeune Water
24 24 Litigation that's currently pending in the
25 25 Eastern District of North Carolina.

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

3 A. Correct.

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

6 ATTORNEY GREENWALD: Objection to
7 form.

8 THE WITNESS: A bunch of times.

9 BY ATTORNEY ANTONUCCI:

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

13 A. Lifetime?

14 Q. Yes, sir.

15 A. I'd say maybe 30 or 40.

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

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

21 A. Correct.

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

24 A. Yes.

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

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

7 A. Yes.

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

12 Do you understand?

13 A. I do.

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

19 Is that fair?

20 A. Yes.

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

1 Do you understand?

2 A. I understand.

3 Q. Is there any reason why you're
4 unable to give your most truthful and
5 accurate testimony today?

6 A. No.

7 Q. Is there any reason why your memory
8 might be impaired?

9 A. No.

10 Q. Dr. Hu, you may ask for breaks at
11 any time. Please just answer any questions
12 I've asked before we go take a break.

13 Are you all right with that?

14 A. Yes.

15 Q. Am I correct, Dr. Hu, that you've
16 been retained by the plaintiffs' leadership
17 group as an expert witness in the in re Camp
18 Lejeune water litigation?

19 A. Yes.

20 Q. And you were retained as an expert
21 witness in general and specific causation;
22 is that correct?

23 A. Correct.

24 Q. Okay. Were you retained for both
25 at the same time?

1 A. I actually don't recall.

2 Q. Before being retained, had you
3 heard about Camp Lejeune?

4 A. Yes.

5 Q. What did you know about Camp
6 Lejeune prior to being retained?

7 ATTORNEY GREENWALD: Objection.
8 Form.

9 THE WITNESS: In the 2010s, I was
10 a member of the Board of Population
11 and Public Health Practice of the
12 National Research Council of the
13 United States, and part of our
14 oversight was to commission the study
15 of Camp Lejeune done by the Centers
16 for Disease Control.

17 BY ATTORNEY ANTONUCCI:

18 Q. Are you referring to the 2009 study
19 on the assessment of the evidence of health
20 effects at Camp Lejeune?

21 A. I think it was a study after that.
22 Or maybe -- actually, I think we were there
23 to review the study. It was commissioned by
24 the board before I became a member of the
25 board, but then our charge was to oversee

1 the conduct of the study and the progress
2 made.

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

7 A. I did not.

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

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

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

18 A. No.

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

21 A. Not that I recall.

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

25 A. I think I may have read it, but

1 other than that, I don't recall.

2 Q. And you read it after it was
3 released publicly; is that correct?

4 A. Correct.

5 Q. Do you have any opinions on the
6 quality of the 2009 NRC report on Camp
7 Lejeune?

8 ATTORNEY GREENWALD: Objection.
9 Form.

10 THE WITNESS: Not as I sit here
11 today.

12 ATTORNEY ANTONUCCI: I am marking
13 for identification Exhibit 6.

14 BY ATTORNEY ANTONUCCI:

15 Q. This is your amended deposition
16 notice.

17 (Exhibit Number 6 was marked for
18 identification.)

19 BY ATTORNEY ANTONUCCI:

20 Q. Dr. Hu, have you seen this document
21 before?

22 A. Yes.

23 Q. I will represent to you that this
24 is the amended deposition notice for the
25 deposition we're currently sitting at; is

1 that right?

2 A. Yes.

3 Q. And you can put that to the side
4 now. I appreciate that.

5 I'm now marking as Exhibit 7 the
6 initial deposition notice and subpoena.

7 (Exhibit Number 7 was marked for
8 identification.)

9 BY ATTORNEY ANTONUCCI:

10 Q. When you've had a chance to look
11 through that, please look back up at me.

12 Dr. Hu, I will represent that that
13 is the original deposition notice and
14 subpoena for your testimony prior to this
15 deposition being rescheduled.

16 Do you have any of the materials
17 mentioned in the subpoena in your custody or
18 control?

19 A. At home, not here.

20 Q. Which of the materials mentioned in
21 the subpoena are in your custody or control?

22 A. It's a long list, Counsel. You
23 really want me to go through it?

24 Q. I'll represent to you that your
25 counsel has objected to the subpoena and

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

3 Do you disagree with that?

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

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

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

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

21 Can you define what you mean by
22 that?

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

1 from your counsel.

2 A. Okay.

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

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

7 (Exhibit Number 8 was marked for
8 identification.)

9 BY ATTORNEY ANTONUCCI:

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

15 A. Yes.

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

18 A. Correct.

19 Q. Are these your current litigation
20 consulting fees?

21 A. Yes.

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

24 Dr. Hu, are you a hematologist?

25 A. No.

1 Q. And you're not an oncologist; is
2 that correct?

3 A. Correct.

4 Q. So you are not a medical or
5 radiation oncologist; correct?

6 A. Correct.

7 Q. Have you ever treated a patient for
8 non-Hodgkin's lymphoma?

9 A. Yes.

10 Q. In what capacity have you treated a
11 patient for non-Hodgkin's lymphoma?

12 A. As a resident in internal medicine
13 at Boston City Hospital.

14 Q. Did your residency include an
15 oncology rotation?

16 A. It did.

17 Q. And that's the capacity in which
18 you treated a non-Hodgkin's lymphoma
19 patient?

20 A. Yes.

21 Q. How long was your oncology rotation
22 during your residency?

23 A. Three months.

24 Q. Did you only treat the one
25 non-Hodgkin's lymphoma patient during that

1 time?

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

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

10 A. Consulted on but not treated.

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

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

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

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

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

1 lymphoma?

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

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

6 A. Correct.

7 Q. You are not an immunologist;
8 correct?

9 A. Correct.

10 Q. And you are not a toxicologist;
11 correct?

12 A. Correct.

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

15 ATTORNEY GREENWALD: Objection.
16 Form.

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

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

1 environmental epidemiologist; so I
2 would disagree with that
3 characterization.

4 BY ATTORNEY ANTONUCCI:

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

7 A. No.

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

11 A. Yes.

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

15 A. Maybe 10 or 12.

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

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

20 Q. Are you an expert in risk
21 assessment?

22 A. I would not say that's true.

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

25 A. Not per se.

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

5 A. Correct.

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

10 A. Correct.

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

15 A. Correct.

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

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

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

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

4 A. No.

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

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

14 Q. Thanks. I'm sorry about that.

15 A. It's all right.

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

20 A. Yes.

21 Q. Have you ever examined Mr. Kidd?

22 A. I have not.

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

25 A. No.

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

3 ATTORNEY GREENWALD: Objection.
4 Form.

5 THE WITNESS: No.

6 BY ATTORNEY ANTONUCCI:

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

10 A. Yes.

11 Q. Have you ever examined Mr. Carter?

12 A. I have not.

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

15 A. No.

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

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

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

22 A. I believe so.

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

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

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

8 (Exhibit Number 1 was marked for
9 identification.)

10 BY ATTORNEY ANTONUCCI:

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

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

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

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

25 A. Okay.

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

4 A. Yes.

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

8 (Exhibit Number 2 was marked for
9 identification.)

10 BY ATTORNEY ANTONUCCI:

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

14 A. Confirmed.

15 Q. Thank you.

16 Next is Exhibit 3. I will
17 represent to you that this is the Phase 3
18 specific causation initial report that you
19 disclosed for Mr. Robert Kidd on June 12,
20 2025.

21 (Exhibit Number 3 was marked for
22 identification.)

23 BY ATTORNEY ANTONUCCI:

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

1 for Mr. Robert Kidd?

2 A. Confirmed.

3 Q. Thank you.

4 Exhibit 4 is the specific causation
5 report that you disclosed for Frances Carter
6 for the estate of Ronald Lee Carter in the
7 Camp Lejeune water litigation on June 12,
8 2025.

9 (Exhibit Number 4 was marked for
10 identification.)

11 BY ATTORNEY ANTONUCCI:

12 Q. I ask that you please review this
13 and confirm that it is your initial specific
14 causation report for Mr. Carter.

15 A. Confirmed.

16 Q. And now Exhibit 5 is your rebuttal
17 report to Dr. Richard F. Ambinder for Robert
18 Arnold Kidd, dated May 16, 2025.

19 (Exhibit Number 5 was marked for
20 identification.)

21 BY ATTORNEY ANTONUCCI:

22 Q. Please review this and confirm that
23 it is your rebuttal report to Dr. Ambinder
24 for Mr. Kidd.

25 A. Confirmed.

1 Q. Great.

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

6 (Exhibit Number 9 was marked for
7 identification.)

8 BY ATTORNEY ANTONUCCI:

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

11 A. Confirmed.

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

17 (Exhibit Number 10 was marked for
18 identification.)

19 BY ATTORNEY ANTONUCCI:

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

22 A. Confirmed.

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

25 ///

1 (Exhibit Number 11 was marked for
2 identification.)

3 BY ATTORNEY ANTONUCCI:

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

9 A. Confirmed.

10 Q. Thanks, Dr. Hu.

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

14 A. Which one?

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

18 A. Oh. I think at some point I
19 chatted with Dr. Felsner in the presence of
20 counsel.

21 Q. And you reviewed the expert report
22 of Dr. Kelly Reynolds disclosed on February
23 7, 2025; is that correct?

24 A. Yes.

25 Q. And you also reviewed the expert

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

3 A. Yes.

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

6 A. I skimmed it and then reviewed
7 specific portions.

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

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

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

15 A. Yes.

16 Q. Are you also an expert in civil
17 engineering?

18 A. I am not.

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

24 ATTORNEY GREENWALD: Objection.
25 Form.

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

3 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

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

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

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

1 ago.

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

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

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

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

16 A. Yes.

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

22 A. Correct.

23 Q. What does that mean?

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

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

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

7 A. Okay.

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

11 A. Yes.

12 Q. Okay. And in this section of
13 page 7 of Exhibit 1, you quote portions of
14 the text of the Camp Lejeune Justice Act of
15 2022; correct?

16 A. Correct.

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

21 ATTORNEY GREENWALD: Objection.

22 Form.

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

1 BY ATTORNEY ANTONUCCI:

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

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

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

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

1 conditions sufficient to conclude that a
2 causal relationship exists.

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

8 Condition B, or -- I'm sorry.

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

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

19 A. Correct.

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

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

1 likely than not but at least as likely or
2 not is similar. Could be thought of as a
3 50 percent, you know, probability. Some
4 call it equipoise, but it's, I guess you
5 could say, one notch below more likely than
6 not.

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

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

15 Q. Understood. My apologies for the
16 confusion there.

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

23 A. I think so. Yes.

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

1 A. It was furnished to me.

2 Q. Was this furnished to you in your
3 capacity as a litigation consultant or as
4 part of your professional work?

5 A. That's a good question. I really
6 don't recall. I think it was probably
7 because of my membership on one of the
8 National Research Council expert committees.
9 And now that I think about it, I think
10 earlier in the deposition, I said I was part
11 of the board of Population and Public Health
12 Practice. I think it was part of this other
13 board I served on called the board of
14 Environmental Studies and Toxicology.

15 Q. Did you serve as a peer reviewer on
16 the ATSDR's 2017 assessment of the evidence?

17 A. Not formally. I was just a member
18 of the board trying to provide oversight on
19 the progress of the study.

20 Q. Did you provide criticism or
21 feedback on the study to ATSDR?

22 A. I'm sure I made some comments, but
23 I don't recall the nature of those comments
24 currently.

25 Q. Do you recall whether you reviewed

1 those as a Microsoft Word document?

2 A. No.

3 Q. Do you have your comments on the
4 ATSDR's 2017 assessment of the evidence in
5 your possession?

6 A. No.

7 Q. Okay. Could you find your comments
8 on ATSDR's 2017 assessment of the evidence
9 if you were asked to?

10 A. You'd have to look -- consult the
11 National Research Council. I'm sure they
12 took minutes at the meetings, and they could
13 find me as a member and find out where they
14 have documented comments. I don't have
15 them.

16 Q. Do you recall whether you ever
17 provided written comments on ATSDR's 2017
18 assessment of the evidence?

19 A. Not that I recall. I think it was
20 verbal at meetings.

21 Q. Okay. So on page 8 of Exhibit 1,
22 your general causation report, you comment
23 on ATSDR's use of four categories to
24 classify the strength of the evidence for a
25 causal relationship between chemicals in the

1 water at Camp Lejeune and various harms.

2 Do you see where I'm reading from
3 there?

4 A. Yes.

5 Q. Do you know where ATSDR got their
6 equipoise and above standard from for their
7 2017 assessment of the evidence?

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

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

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

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

25 A. Yes.

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

4 ATTORNEY GREENWALD: Objection.

5 Asked and answered.

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

13 BY ATTORNEY ANTONUCCI:

14 Q. Okay. Also, in Exhibit 1 on page
15 8, you state that you expressed some of your
16 opinions in your report under a more likely
17 than not standard; is that correct?

18 A. Correct.

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

21 ATTORNEY GREENWALD: Objection.

22 Form.

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

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

11 BY ATTORNEY ANTONUCCI:

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

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

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

20 A. Correct.

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

25 A. It's based on essentially what I

1 wrote here.

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

5 A. Sure.

6 Q. I appreciate that.

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

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

15 ATTORNEY ANTONUCCI: Not at all.

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

19 BY ATTORNEY ANTONUCCI:

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

24 Do you see that?

25 A. Yes.

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

10 Did I read that correctly?

11 A. You did.

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

16 Are you looking at Exhibit 3,
17 Dr. Hu?

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

20 Q. Yes, sir.

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

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

8 Did I read that correctly?

9 A. Yes.

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

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

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

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

25 Q. So in your view, if something is a

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

4 ATTORNEY GREENWALD: Objection.
5 Form. That mischaracterizes his
6 testimony.

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

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

14 BY ATTORNEY ANTONUCCI:

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

17 ATTORNEY GREENWALD: Objection.
18 Form.

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

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

7 BY ATTORNEY ANTONUCCI:

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

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

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

19 A. Yes.

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

23 That's Exhibit 3; is that correct?

24 A. Yes.

25 Q. You wrote that: The combination of

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

5 Is that correct?

6 A. Correct.

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

11 ATTORNEY GREENWALD: Objection.

12 Form.

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

22 BY ATTORNEY ANTONUCCI:

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

1 "substantial contributing factor."

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

4 ATTORNEY GREENWALD: Objection.
5 Form, asked and answered.

6 THE WITNESS: No.

7 ATTORNEY ANTONUCCI: I am marking
8 for identification Exhibit 12.

9 (Exhibit Number 12 was marked for
10 identification.)

11 BY ATTORNEY ANTONUCCI:

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

16 A. Yes.

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

19 A. Okay.

20 Q. This is an invoice dated May 1,
21 2025; is that correct?

22 A. Yes.

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

1 A. Correct.

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

11 A. Yes.

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

15 ATTORNEY GREENWALD: Objection.
16 Form.

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

21 BY ATTORNEY ANTONUCCI:

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

1 A. I did not, if I recall correctly.

2 Q. Did you review the Wang, et al.,
3 2025 UK Biobank Study prior to issuing your
4 supplemental general causation report?

5 A. I did not, as I recall.

6 Q. Did you review the Yu, et al., 2025
7 UK Biobank Study prior to issuing your
8 general causation report?

9 A. 2025 did you say?

10 Q. Yes, sir.

11 Had you reviewed the Yu, et al.,
12 2025 UK Biobank Study prior to issuing your
13 general causation report?

14 A. No.

15 Q. Had you reviewed the Yu, et al.,
16 2025 UK Biobank Study pre-print prior to
17 issuing your general causation report?

18 A. No.

19 Q. Had you reviewed the Yu, et al.,
20 2025 UK Biobank Study prior to issuing your
21 supplemental general causation report?

22 A. Not that I recall.

23 Q. Dr. Hu, how did you become aware of
24 the Wang, et al., and Yu, et al., studies?

25 A. I believe counsel pointed out that

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

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

10 A. Correct.

11 Q. What is Nix versus Chemours and
12 DuPont?

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

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

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

25 Q. Okay. Do you recall why you

1 reviewed Nix versus Chemours and DuPont?

2 ATTORNEY GREENWALD: Objection to
3 form.

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

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

10 THE WITNESS: Okay.

11 ATTORNEY GREENWALD: That's
12 privileged.

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

15 BY ATTORNEY ANTONUCCI:

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

19 A. No.

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

23 ATTORNEY GREENWALD: Objection.
24 Form.

25 THE WITNESS: No.

1 BY ATTORNEY ANTONUCCI:

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

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

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

10 ATTORNEY GREENWALD: Objection.
11 Form.

12 THE WITNESS: No.

13 BY ATTORNEY ANTONUCCI:

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

17 ATTORNEY GREENWALD: Same
18 objection.

19 THE WITNESS: No.

20 BY ATTORNEY ANTONUCCI:

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

25 Is that because you did not

1 consider them in rendering your opinions in
2 this case?

3 A. Correct.

4 ATTORNEY GREENWALD: Objection.

5 Asked and answered.

6 THE WITNESS: Sorry. Correct.

7 BY ATTORNEY ANTONUCCI:

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

13 A. Correct.

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

17 A. Yes.

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

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

25 Q. Okay. If a mutation had occurred

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

4 A. Correct.

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

7 A. Yes.

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

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

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

15 A. Correct.

16 ATTORNEY GREENWALD: Objection.
17 Form.

18 BY ATTORNEY ANTONUCCI:

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

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

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

1 A. Correct.

2 Q. Diffuse large B-cell lymphoma is a
3 subtype of NHL; correct?

4 A. Correct.

5 Q. If I refer to diffuse large B-cell
6 lymphoma as DLBCL, will you understand what
7 I mean?

8 A. Yes.

9 Q. DLBCL is the most common subtype of
10 NHL; correct?

11 A. Correct.

12 Q. DLBCL accounts for about a third of
13 NHLs; correct?

14 A. I don't recall what the latest
15 proportion is under -- using the Seer data,
16 but that would be the source of data that I
17 would use to make that estimate.

18 Q. Mr. Carter was diagnosed with
19 mantle cell lymphoma; right?

20 A. Correct.

21 Q. Mantle cell lymphoma is a type of
22 B-cell lymphoma; isn't it?

23 A. Type of what?

24 Q. B-cell lymphoma.

25 A. Yes.

1 Q. And mantle cell lymphoma is a
2 subtype of NHL; correct?

3 A. Correct.

4 Q. And if I refer to mantle cell
5 lymphoma as MACL, will you understand what I
6 mean?

7 A. Yes.

8 Q. MACL is a rare subtype of NHL;
9 correct?

10 A. More or less.

11 Q. MACL accounts for about 5 percent
12 of all NHLs; correct?

13 A. More or less, correct.

14 Q. Are you familiar with the term
15 "hematopoietic malignancies"?

16 A. Yes.

17 Q. The term "hematopoietic
18 malignancies" encompasses blood cancers;
19 correct?

20 A. Correct.

21 Q. The term "hematopoietic
22 malignancies" encompasses NHL; correct?

23 A. I'd agree with that.

24 ATTORNEY ANTONUCCI: Okay. I'd
25 like to take a five-minute break.

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

6 (Recess taken from 10:15 a.m. to
7 10:37 a.m.)

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

12 BY ATTORNEY ANTONUCCI:

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

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

17 A. Correct.

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

21 A. Not at all.

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

25 A. Thank you.

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

3 A. Okay.

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

6 A. Yes.

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

11 Did I read that correctly?

12 A. You did.

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

15 A. Yes.

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

19 A. Yes.

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

22 A. I agree with that.

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

25 A. Correct.

1 Q. Dr. Hu, you never mention multiple
2 myeloma in any of your reports; correct?

3 A. I did not.

4 Q. And you're not offering opinions
5 about multiple myeloma in this case; is that
6 correct?

7 A. Correct.

8 Q. Dr. Hu, did I understand you
9 correctly that you said all NHL subtypes
10 arise from mutations in stem cells that will
11 become lymphocytes?

12 A. I don't think I meant to say all,
13 but I meant to say that they can start in
14 mutations in the pluripotential stem cells.

15 Q. What is the basis of that opinion?

16 A. It's based on my understanding of
17 current cancer biology as it relates to
18 hematopoietic cancers as well as some of the
19 writings that have appeared in peer-reviewed
20 journals by my colleague, Dr. Bernard
21 Goldstein, who's a hematologist and
22 environmental health expert and who has
23 studied in depth the subject of
24 environmental carcinogenesis and
25 hematopoietic cancers. I quoted some of his

1 work in one of my reports.

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

6 A. That's one of them, yes.

7 Q. Are there any others?

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

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

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

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

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

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

10 A. Correct.

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

14 A. Yes.

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

18 Do you agree?

19 A. Yes.

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

23 A. Yes.

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

1 A. I would say that's correct.

2 Q. What databases did you use to
3 perform your literature search?

4 A. Mostly PubMed.

5 Q. Other than PubMed, did you use any
6 others?

7 A. You know, I would use Google
8 Scholar particularly to see if there's any
9 so-called gray literature that's not
10 published in the peer-reviewed literature.
11 It includes things like monographs and
12 books.

13 Q. Other than PubMed and Google
14 Scholar, did you use any other research
15 database for your literature review?

16 A. No.

17 Q. For your specific causation
18 conclusions, what were your search terms?

19 A. Well, my specific causation reports
20 were based first on my general causation
21 reports in which I used the searches I just
22 explained to you. And then the rest of it
23 was mostly dependent on the specifics of the
24 plaintiff themselves, and sometimes I would
25 review the literature further if there are

1 nuances of their particular case that needed
2 further explanation.

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

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

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

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

1 as your search terms.

2 Did I understand that correctly?

3 A. Yes.

4 Q. What is a medical subject heading?

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

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

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

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

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

1 but I think I did.

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

4 A. I don't remember.

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

8 A. I don't remember.

9 Q. Are your search terms included in
10 your reports?

11 A. Not that I recall, no.

12 Q. Why not?

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

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

17 ATTORNEY GREENWALD: Objection.
18 Form.

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

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

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

11 BY ATTORNEY ANTONUCCI:

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

16 ATTORNEY GREENWALD: Objection.

17 Form.

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

1 between an exposure of interest and
2 outcome of interest.

3 BY ATTORNEY ANTONUCCI:

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

8 A. Yes.

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

11 A. No.

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

14 A. DCE?

15 Q. 1,2 trans trichloroethylene.

16 A. Yeah, no.

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

20 ATTORNEY GREENWALD: Objection.

21 Form, asked and answered.

22 THE WITNESS: I did not.

23 BY ATTORNEY ANTONUCCI:

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

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

3 ATTORNEY GREENWALD: Objection.
4 Form.

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

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

16 BY ATTORNEY ANTONUCCI:

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

21 ATTORNEY GREENWALD: Objection.
22 Form.

23 THE WITNESS: Not that I recall.

24 BY ATTORNEY ANTONUCCI:

25 Q. Are you aware of any consensus

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

4 ATTORNEY GREENWALD: Objection.

5 Form.

6 THE WITNESS: Not that I'm aware
7 of.

8 BY ATTORNEY ANTONUCCI:

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

13 ATTORNEY GREENWALD: Objection.

14 Form.

15 THE WITNESS: Not that I'm aware.

16 BY ATTORNEY ANTONUCCI:

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

21 ATTORNEY GREENWALD: Objection.

22 Form.

23 THE WITNESS: Not that I'm aware.

24 I'm not sure it's ever been
25 considered.

1 BY ATTORNEY ANTONUCCI:

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

8 A. Yes.

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

14 ATTORNEY GREENWALD: Objection.
15 Form.

16 THE WITNESS: Correct.

17 BY ATTORNEY ANTONUCCI:

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

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

25 Q. Sure. So it might help if you look

1 at page 13 of Exhibit 4.

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

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

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

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

13 (Exhibit Number 13 was marked for
14 identification.)

15 BY ATTORNEY ANTONUCCI:

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

18 A. Yes.

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

21 A. Yes.

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

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

5 Did I read that correctly?

6 A. Yes.

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

15 A. Correct.

16 Q. All seven specimens that Racke, et
17 al., studied showed evidence of mantle cell
18 lymphoma, and the oldest specimen was taken
19 15.5 years prior to the diagnosis of mantle
20 cell lymphoma; correct?

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

1 stage prior to actual manifestation of the
2 disease.

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

7 A. I see it.

8 Q. Pathological specimens range from
9 2.1 to 15.5 years prior to the diagnosis of
10 MCL. In all seven specimens, cyclin D1
11 positive collections of lymphocytes were
12 identified, five with distinct homing to
13 mantle zones, one with follicular
14 colonization, and one with a diffuse
15 distribution.

16 Did I read that correctly?

17 A. You did.

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

22 A. Correct.

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

1 A. Correct.

2 Q. Do you know if any of the study
3 participants in Racke, et al., were exposed
4 to any toxicants?

5 A. I don't think that was ever
6 considered.

7 Q. Do you know if the latency periods
8 discussed in Racke, et al., is at all
9 related to their exposure to toxicants?

10 A. There's no information about that.

11 Q. Do you know whether or not
12 Mr. Carter had evidence of in situ mantle
13 cell lymphoma before 2010?

14 A. He's never had a lymph node biopsy,
15 as far as I know, prior to the manifestation
16 of his disease.

17 Q. Okay. You can put that to the
18 side. Thanks very much.

19 So, Dr. Hu, earlier you mentioned
20 you also cited Olsson and Brandt; is that
21 right?

22 A. Correct.

23 ATTORNEY ANTONUCCI: I'm going to
24 mark Olsson and Brandt as Exhibit 14.

25 ///

1 (Exhibit Number 14 was marked for
2 identification.)

3 BY ATTORNEY ANTONUCCI:

4 Q. Dr. Hu, I just handed you Risk of
5 Non-Hodgkin's Lymphoma Among Men
6 Occupationally Exposed to Organic Solvents
7 by Olsson and Brandt published in 20 --
8 1988, excuse me.

9 Is that correct?

10 A. Yes.

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

13 A. Correct.

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

19 Do you see that?

20 A. Yes.

21 Q. Olsson and Brandt wrote: There
22 were large variations in the length of
23 periods from the start of exposure to the
24 diagnosis of non-Hodgkin's lymphoma 2 to
25 60 years with a median of 21 years.

1 Did I read that correctly?

2 A. You did.

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

6 A. Well, this sentence basically says
7 that the latency period, which is the length
8 of period from exposure to diagnosis of the
9 disease, in the cases varied from 2 to
10 60 years with a median that is the 50th
11 percentile of 21 years.

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

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

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

21 A. Correct.

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

1 A. Well, I considered them.

2 Q. Okay. And you also considered
3 known risk factors for NHL that were not
4 applicable to each plaintiff; correct?

5 A. That's correct.

6 Q. And you ruled those out; correct?

7 A. Well, I considered them.

8 Q. Okay. Is there a reason that you
9 didn't rule out risk factors that were not
10 applicable to each plaintiff?

11 A. Well, when I say I considered them,
12 that also includes whether I -- you say rule
13 them out. I say I discount them, and it's a
14 somewhat equivalent term. Rule out really
15 is a bit more absolute, I guess, and I
16 hesitate to use a term like that unless
17 there's absolute certainty that, you know,
18 the factor was not present.

19 So, for instance, if there's no
20 documentation that someone had been tested
21 for a particular thing, you know, I -- may
22 not necessarily be ruled out, but I
23 discounted it because there was no evidence
24 that they had something else.

25 Q. In conducting the differential

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

3 ATTORNEY GREENWALD: Objection.
4 Form.

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

7 BY ATTORNEY ANTONUCCI:

8 Q. Let's start with Mr. Kidd.

9 A. Okay.

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

13 ATTORNEY GREENWALD: Same
14 objection.

15 THE WITNESS: Let me go to that
16 report.

17 BY ATTORNEY ANTONUCCI:

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

21 A. I'm there.

22 Q. Okay.

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

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

9 However, he does not have family
10 history of a first-degree relative with NHL.
11 There's no previous treatment for cancer
12 chemotherapy drugs. He did not have
13 evidence of a weakened immune system. He
14 did not have evidence of inherited syndromes
15 associated with immunodeficiency. He did
16 not have evidence of autoimmune disease,
17 previous lymphoma, or chronic infections
18 with *Helicobacter pylori*, *Chlamydia*,
19 *Siddiqi*, *Campylobacter jejuni*, or hepatitis
20 C, and no history of breast implants.

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

25 So just to go back to your

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

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

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

15 ATTORNEY GREENWALD: Objection.
16 Form.

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

1 would be considered a risk factor for
2 NHL.

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

8 BY ATTORNEY ANTONUCCI:

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

18 Did I read that correctly?

19 A. You did.

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

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

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

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

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

23 ATTORNEY GREENWALD: Objection.

24 Form.

25 THE WITNESS: Well, it's just one

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

9 BY ATTORNEY ANTONUCCI:

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

12 A. Yes.

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

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

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

1 causation initial report for Mr. Kidd.

2 A. Okay.

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

6 Do you see where I am?

7 A. Yes.

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

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

5 Did I read that correctly?

6 A. You did.

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

11 A. Correct.

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

16 A. Correct.

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

21 A. Correct.

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

25 A. That's not true.

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

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

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

9 Q. Yes, sir.

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

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

15 A. Yeah.

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

21 Did I read that correctly?

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

25 Q. Okay. I'll have you turn to

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

3 A. Yes.

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

8 Did I read that correctly?

9 A. That's correct.

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

16 Did I read that correctly?

17 A. You did.

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

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

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

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

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

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

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

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

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

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

3 A. Can you repeat the original
4 question?

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

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

13 Q. Is that also true of NHL subtypes?

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

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

1 Q. Dr. Hu, are you familiar with the
2 NHL subtype MALT lymphoma?

3 A. Which one?

4 Q. MALT lymphoma?

5 A. Is that an acronym, or is that the
6 name?

7 Q. MALT lymphoma is an acronym for
8 mucosa-assisted lymphoid tissue lymphoma.

9 Are you familiar with it?

10 A. No.

11 Q. Is there any way to tell from the
12 clinical presentation of a person's NHL
13 whether it was caused by exposure to one of
14 PCE, TCE, or benzene?

15 A. No --

16 ATTORNEY GREENWALD: Object --

17 THE WITNESS: Not that I'm aware
18 of.

19 BY ATTORNEY ANTONUCCI:

20 Q. And so the only way to determine
21 whether an individual's NHL was caused by
22 exposure to TCE, PCE, or benzene is through
23 toxicological and epidemiological analysis
24 coupled to evidence of exposure?

25 ATTORNEY GREENWALD: Objection.

1 Form.

2 THE WITNESS: Well, in my view, it
3 can only be done by the process that I
4 went through, which is first consider
5 the general causation and then
6 consider all the specific information
7 about the particular patient in terms
8 of their patient history, their family
9 history, their dietary history, their
10 infectious disease history, the
11 clinical presentation of the disease,
12 the data on exposure to the various
13 things that allegedly may have caused
14 their cancer, the length of exposure,
15 the level of exposure, on and on and
16 on.

17 BY ATTORNEY ANTONUCCI:

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

21 A. Yes.

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

1 Dr. Reynolds' exposure calculations?

2 ATTORNEY GREENWALD: Objection.

3 Form.

4 THE WITNESS: I incorporated her
5 calculations in my analysis.

6 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

1 exposure?

2 ATTORNEY GREENWALD: Objection.

3 Form.

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

10 BY ATTORNEY ANTONUCCI:

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

18 A. Correct.

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

22 A. That's correct.

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

1 Dr. Reynolds' exposure calculation; correct?

2 A. Yes.

3 Q. Do you agree that your opinions on
4 Mr. Kidd's and Mr. Carter's exposures caused
5 by their NHL are, therefore, only as good as
6 Dr. Reynolds' numbers?

7 ATTORNEY GREENWALD: Objection.
8 Form.

9 THE WITNESS: Well, as I also
10 discussed in my report, I compared the
11 experience of Mr. Carter with some of
12 the scenarios that appeared in the
13 ATSDR report, and one of the scenarios
14 in particular which I reproduced in my
15 specific causation report, was very
16 similar to the exposure scenario for
17 Mr. Carter.

18 I also found one that was very
19 similar to the exposure scenario for
20 Mr. Kidd, and ATSDR went through the
21 process of taking these illustrative
22 exposure scenarios and then
23 calculating the lifetime cancer risk
24 by age group based on the years of
25 exposure, whether they worked on base

1 or lived off base, et cetera.

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

7 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

1 Camp Lejeune?

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

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

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

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

15 A. I have no knowledge of that.

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

19 A. I have no knowledge of that.

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

24 (Exhibit Number 15 was marked for
25 identification.)

1 BY ATTORNEY ANTONUCCI:

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

7 A. Great.

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

10 ATTORNEY ANTONUCCI: 15.

11 ATTORNEY GREENWALD: Thank you.

12 BY ATTORNEY ANTONUCCI:

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

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

19 Q. Appendix 19.

20 A. 19. Okay.

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

25 THE WITNESS: There it is.

1 ATTORNEY GREENWALD: It's right
2 here.

3 THE WITNESS: All right. Thanks.
4 BY ATTORNEY ANTONUCCI:

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

9 A. Yes.

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

13 A. Yes.

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

18 A. Yes.

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

22 A. Yes.

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

1 month the plaintiff was exposed; correct?

2 A. Yes.

3 Q. Have you ever seen an exposure
4 assessment presented in terms of cumulative
5 microgram per liter month before?

6 A. Microgram per liter month. I
7 probably have. I've seen a lot of exposure
8 assessments, but I can't remember which ones
9 or when.

10 Q. Okay. Now, if you look at the next
11 column over, it's labeled Chart 1: 1L, for
12 one liter.

13 Do you see that?

14 A. Yes.

15 Q. And this shows cumulative monthly
16 total contamination exposure for each of the
17 volatile organic compounds at issue in this
18 case; right?

19 A. Yes.

20 Q. And this was calculated by
21 multiplying the number of exposure days by 1
22 liter per day by the summed monthly average
23 concentrations; is that right?

24 A. Yes.

25 Q. Okay. And chart 2, which is

1 labeled ATSDR, shows the same data as chart
2 1, but instead of 1 liter, Dr. Reynolds used
3 ATSDR's ingestion estimates of 6 liters per
4 day three days per week and 3 liters per day
5 three days per week; correct?

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

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

13 A. Something that referred to the
14 deposition, yes.

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

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

25 Q. Okay. Of all of the five columns

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

4 A. That's correct.

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

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

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

13 ATTORNEY GREENWALD: Objection.

14 Form, asked and answered.

15 THE WITNESS: Yes.

16 BY ATTORNEY ANTONUCCI:

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

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

25 Q. And I apologize if I already asked

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

6 A. Correct.

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

10 A. Yes.

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

13 ATTORNEY GREENWALD: Objection.
14 Form.

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

18 BY ATTORNEY ANTONUCCI:

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

21 What are you referring to, Dr. Hu?

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

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

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

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

7 BY ATTORNEY ANTONUCCI:

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

11 A. The ones at the very bottom.

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

15 A. That's correct.

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

19 A. That's correct.

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

22 Am I correct?

23 ATTORNEY GREENWALD: Objection.
24 Form.

25 THE WITNESS: To concentrations

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

4 BY ATTORNEY ANTONUCCI:

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

10 A. That's correct. Yes.

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

14 Is that your understanding?

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

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

23 A. Yes.

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

1 calculations be impacted?

2 ATTORNEY GREENWALD: Objection.

3 Form.

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

11 BY ATTORNEY ANTONUCCI:

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

17 ATTORNEY GREENWALD: Objection.

18 Form.

19 THE WITNESS: 15 percent?

20 Probably not.

21 BY ATTORNEY ANTONUCCI:

22 Q. Okay. If Dr. Reynolds' cumulative
23 exposure numbers decreased by a factor of
24 15 percent, would that impact your opinions
25 on the causation of Mr. Carter's NHL?

1 A. Probably not.

2 Q. How much would Dr. Reynolds'
3 cumulative exposure numbers have to decrease
4 to impact your opinions on the causation of
5 the plaintiffs' NHL?

6 ATTORNEY GREENWALD: Objection.
7 Form.

8 THE WITNESS: I don't know. I
9 haven't considered that, and I'm not
10 able to give you an opinion on that
11 right now.

12 BY ATTORNEY ANTONUCCI:

13 Q. Would it be more than 50 percent?

14 A. I don't know.

15 ATTORNEY GREENWALD: Objection.
16 Form.

17 BY ATTORNEY ANTONUCCI:

18 Q. So to be clear, you haven't
19 considered whether or not -- if
20 Dr. Reynolds' numbers were too high or too
21 low, if that would impact your final
22 conclusions; is that correct?

23 ATTORNEY GREENWALD: Objection to
24 form.

25 THE WITNESS: That's not an

1 exercise I performed.

2 BY ATTORNEY ANTONUCCI:

3 Q. Okay. And you testified that
4 15 percent would be too low to change your
5 opinions but that you weren't aware whether
6 50 percent would be too high; is that right?

7 ATTORNEY GREENWALD: Objection to
8 form. Asked and answered.

9 THE WITNESS: That's what I said,
10 and that's, you know, again, off the
11 top of my head, but I hesitate to say
12 anything with any more certitude given
13 that I haven't really considered the
14 question.

15 BY ATTORNEY ANTONUCCI:

16 Q. Okay. Dr. Reynolds did not perform
17 a risk assessment for either plaintiff, did
18 she?

19 A. Not that I'm aware.

20 Q. She did not characterize the
21 plaintiffs' risk from exposure to
22 contamination; correct?

23 A. That's another way of saying the
24 same thing. I'm not aware of it.

25 Q. And she didn't calculate their

1 excess lifetime cancer risk based on their
2 exposure to contamination?

3 A. Not that I'm aware of.

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

7 A. Define margins of exposure.

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

14 A. Correct.

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

18 A. Correct.

19 Q. Rather she just quantified
20 exposure; correct?

21 ATTORNEY GREENWALD: Objection.
22 Form.

23 THE WITNESS: More or less, yes.

24 BY ATTORNEY ANTONUCCI:

25 Q. And you did not perform a risk

1 assessment either; correct?

2 A. Correct.

3 Q. And you didn't use Dr. Reynolds'
4 exposure calculations to calculate lifetime
5 cancer risk; correct?

6 A. I did not.

7 Q. You didn't calculate a margin of
8 exposure for Mr. Kidd or for Mr. Carter;
9 correct?

10 A. Correct.

11 Q. You relied instead on ATSDR's 2017
12 Public Health Assessment lifetime cancer
13 risk estimates; is that correct?

14 ATTORNEY GREENWALD: Objection.
15 Form.

16 THE WITNESS: I used that, yes.
17 BY ATTORNEY ANTONUCCI:

18 Q. Okay. I'd like you to turn to
19 Exhibit 4. That's the initial specific
20 causation report for Mr. Carter.

21 A. Okay.

22 Q. And if you could, please go to
23 page 9.

24 A. All right.

25 Q. Under subheading 3, Exposure

1 Assessment, Risk of Cancer, Ronald Carter,
2 the end of the first paragraph you wrote:
3 Accounting for Mr. Carter's workweek,
4 vacations, et cetera, Dr. Reynolds estimated
5 Mr. Carter had a total exposure time of
6 6,865 days, which is equivalent to
7 229 months. One can consequently calculate
8 Mr. Carter's time-weighted average exposure
9 levels of PCE, TCE, and benzene by totaling
10 his microgram per liter months of exposure
11 to each chemical from Hadnot Point, Tarawa
12 Terra, and Midway Park and dividing by 229
13 months. This yields time-weighted average
14 exposure levels of 34.5 micrograms per liter
15 or parts per billion of PCE, 113 micrograms
16 per liter or parts per billion of TCE, and
17 1.7 micrograms per liter or parts per
18 billion of benzene.

19 Did I read that correctly?

20 A. You did.

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

25 A. Say that one more time.

1 Q. I'll break that down a little bit.

2 Mr. Carter's total exposure was
3 calculated as being 229 months; correct?

4 A. Yes.

5 Q. And his cumulative micrograms per
6 liter month of exposure to TCE as calculated
7 by Dr. Reynolds was 25,877 micrograms per
8 liter; correct?

9 You may need to refer to the
10 appendices of Dr. Reynolds' report to find
11 that out.

12 A. Of TCE?

13 Q. Correct.

14 A. I have 25,603 in my paragraph.
15 That's a little bit different than the
16 figure you just quoted.

17 Q. That is for the Hadnot Point Water
18 Treatment Plant; correct?

19 A. Yeah.

20 Q. And for the Tarawa Terrace Water
21 Treatment Plant for TCE, it was 225
22 micrograms per liter month; correct?

23 A. Yes.

24 Q. For PCE, Dr. Reynolds calculated --
25 strike that. I'm going to move on.

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

4 A. Correct.

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

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

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

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

15 Q. Page 7 of Exhibit 4.

16 A. Okay.

17 Q. If you look at note 17 of
18 Exhibit 4, you wrote that: CREG cancer risk
19 evaluation guide concentrations represent
20 concentrations of cancer-causing substances
21 unlikely to result in an increase of cancer
22 risk in an exposed population above a target
23 risk level of one excess cancer per
24 1 million exposed people. They are derived
25 by ATSDR using USEPA cancer slope factors,

1 default exposure assumptions (regarding
2 ingestion and body weights).

3 Did I read that correctly?

4 A. Yes.

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

10 A. Right.

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

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

14 Q. Okay. So it's over an average of
15 70 years --

16 A. Yes.

17 Q. -- is that right?

18 CREG values are developed for
19 drinking water to identify a concentration
20 at the tab representing one in a million or
21 1 times 10 to the negative 6 risk; is that
22 correct?

23 A. Yes.

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

1 A. Yes.

2 Q. And that's when consumed by a
3 70-kilogram human drinking 2 liters of such
4 water per day for a 70-year lifetime;
5 correct?

6 A. Is it 70 or 75? I don't remember
7 the exact parameters, but I have no reason
8 to disagree with you, Counsel.

9 Q. And you're aware that an MCL, or
10 maximum contaminant level, is the highest
11 level of a contaminant that's allowed in
12 drinking water as determined by the EPA;
13 correct?

14 A. Yes.

15 Q. EPA takes health risks into
16 consideration when setting MCLs; is that
17 right?

18 A. Correct.

19 Q. They also take available water
20 treatment technology into account when
21 setting MCLs; correct?

22 A. They also take into account what?

23 Q. When setting MCLs, EPA also takes
24 into account available water treatment
25 technology.

1 A. I don't know the answer to that.

2 Q. When setting MCLs, EPA takes into
3 account costs; is that right?

4 A. I knew the answer to that at one
5 time, but I don't now. I'm not sure,
6 Counsel.

7 Q. And MCL is not the same thing as a
8 reference dose; correct?

9 A. Correct.

10 Q. And then MCL is not the same thing
11 as a threshold dose; correct?

12 A. Correct.

13 Q. And I believe you wrote in your
14 rebuttal reports that comparing the CREG and
15 the MCL to ATSDR's -- excuse me. Strike
16 that.

17 You wrote in your rebuttal reports
18 that comparisons to time-weighted averages
19 of exposures to micrograms per liter to MCLs
20 and CREGs was not a methodology for
21 quantitating risk; is that right?

22 A. Right.

23 Q. So why did you make those
24 comparisons?

25 A. It's simply to give a sense of the

1 degree of exposure of the individual.

2 Q. Do you agree that an exposure
3 exceeding the CREG provides basis only to
4 conclude that there's an increased risk of
5 more than one in a million for any type of
6 cancer?

7 ATTORNEY GREENWALD: Objection.

8 Form.

9 THE WITNESS: I would agree with
10 that.

11 BY ATTORNEY ANTONUCCI:

12 Q. And do you agree that exceeding the
13 MCL is not a basis for any conclusion about
14 the risk of an individual's cancer?

15 A. I would agree that it's not useful
16 for that purpose.

17 Q. Okay. When you calculated the time
18 weighted cumulative microgram per liter
19 calculations, did you take into account
20 Mr. Kidd or Mr. Carter's body weight?

21 A. No, because I think in terms of
22 risk, it doesn't matter whether he's
23 150 kilograms or only 50 kilograms if he's
24 drinking water at a certain concentration.
25 He's being exposed relatively to the same

1 amount. Presumably someone who's
2 150 kilograms is drinking a lot more water
3 than someone who's 50 kilograms.

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

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

13 ATTORNEY GREENWALD: Objection.
14 Form.

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

21 BY ATTORNEY ANTONUCCI:

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

24 ATTORNEY GREENWALD: Objection to
25 form.

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

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

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

24 BY ATTORNEY ANTONUCCI:

25 Q. Okay. So you don't need

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

4 A. No.

5 ATTORNEY GREENWALD: Objection.
6 Form.

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

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

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

1 50 kilograms drinking the same
2 concentration of the pollutant.

3 BY ATTORNEY ANTONUCCI:

4 Q. So please help me understand.

5 A. Sure.

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

8 ATTORNEY GREENWALD: Objection.
9 Form.

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

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

22 BY ATTORNEY ANTONUCCI:

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

25 A. Dr. Reynolds simply quantified the

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

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

8 ATTORNEY GREENWALD: Objection.
9 Form.

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

13 BY ATTORNEY ANTONUCCI:

14 Q. Sure.

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

20 ATTORNEY GREENWALD: Objection.
21 Form.

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

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

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

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

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

20 (Exhibit Number 16 was marked for
21 identification.)

22 BY ATTORNEY ANTONUCCI:

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

1 report; is that right?

2 A. I'm not sure, as I sit here, but I
3 have no reason to disagree with that.

4 Q. Okay. So on page 7 and 8 of your
5 initial report on Mr. Carter, you wrote
6 that: ATSDR calculated three-year rolling
7 average concentrations of contaminants in
8 drinking water.

9 Would you like me to find that and
10 point it out for you?

11 A. No. I see it at the top of page 8.

12 Q. Right.

13 So do you agree that's what they
14 did?

15 A. Yes.

16 Q. And this was based on ATSDR's
17 historical reconstruction and carried out by
18 Mr. Maslia; is that right?

19 A. That's correct.

20 Q. Do you know how ATSDR calculated
21 that three-year rolling average?

22 A. I don't know the specifics, no.

23 Q. How did ATSDR perform the exposure
24 assessment for the public health assessment?

25 A. I read at one point that the --

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

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

7 ATTORNEY GREENWALD: Objection.
8 Form.

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

12 BY ATTORNEY ANTONUCCI:

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

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

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

23 A. We're back to Kidd?

24 Q. Yes, sir.

25 A. Okay. Page 8.

1 Q. Correct.

2 A. All right. Hold on. Okay.

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

17 Is that right?

18 A. Correct.

19 Q. And then just to clarify, one of
20 the acronyms you wrote, the Hadnot Point
21 WTP.

22 What does that mean?

23 A. Water treatment plant.

24 Q. And then you wrote something very
25 similar in your report for Mr. Carter.

1 That's Exhibit 4 on page 10.

2 Here, you wrote: In addition, I
3 note that Mr. Carter's status as a civilian
4 worker at Camp Lejeune and the timing of his
5 exposure profile very closely aligns with
6 and, in fact, exceeds the exposure profile
7 (discussed in 2G above) of 15 years of
8 exposure to workers on base who lived off
9 base to all chemical contaminants from
10 Hadnot Point (i.e., PCE, TCE, and benzene)
11 that ATSDR estimated was associated with a
12 lifetime cancer risk of over 1 per 10,000
13 for exposures between the mid-1960s to
14 around 1982 with a peak of 2.6 per 10,000
15 for exposures surrounding 1970. Added to
16 that risk was his exposure to Tarawa Terrace
17 WTP from June 1, 1980, through December 1,
18 1987, which is mostly within the interval of
19 time (1950s to mid-1980s) in which ATSDR
20 estimated the lifetime cancer risk by age
21 group over time based on 15 years of
22 exposure to workers on base who lived off
23 base to all chemical contaminants of Tarawa
24 Terrace (i.e., PCE, TCE, and vinyl chloride)
25 to be over 1 per 1 million and in the range

1 of 1 per 100,000.

2 Is that right?

3 A. That's what I wrote.

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

7 ATTORNEY GREENWALD: Objection.
8 Form.

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

17 BY ATTORNEY ANTONUCCI:

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

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

1 found that compelling.

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

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

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

11 THE VIDEOGRAPHER: We are back on
12 the record. This is the beginning of
13 media Unit Number 3, and the time is
14 1:10 p.m.

15 BY ATTORNEY ANTONUCCI:

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

18 Do you understand?

19 A. I do.

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

23 A. No.

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

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

3 A. Yes.

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

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

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

12 ATTORNEY GREENWALD: Objection.
13 Form.

14 THE WITNESS: Not that I know.

15 BY ATTORNEY ANTONUCCI:

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

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

21 Q. Yes, sir.

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

25 ATTORNEY GREENWALD: And I still

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

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

7 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

23 A. Geographically?

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

1 Boulevard, Tarawa Terrace water treatment
2 plants?

3 A. It has its own water treatment
4 plant.

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

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

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

16 A. I don't recall that.

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

21 A. Correct.

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

25 A. Yes.

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

7 A. Correct.

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

11 A. I do recall that, yes.

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

15 A. Correct.

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

19 A. Okay.

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

24 A. Yes. I see it.

25 Q. The second sentence of that

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

6 Did I read that correctly?

7 A. You did.

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

11 A. Say that again.

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

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

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

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

25 Q. Do you know whether liver cancer

1 has a more sensitive cancer endpoint than
2 NHL?

3 ATTORNEY GREENWALD: Objection.
4 Form.

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

8 BY ATTORNEY ANTONUCCI:

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

11 ATTORNEY GREENWALD: Same
12 objection.

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

15 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

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

11 A. 30 --

12 Q. 35.

13 A. Okay.

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

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

19 A. Yes.

20 Q. And you can see in the key for this
21 graphic that ATSDR shaded in gray the cancer
22 risk range of 10 to the negative 4 for 1 in
23 10,000 to 10 to the negative 61 in 1
24 million?

25 A. Yes.

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

3 A. Okay.

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

7 Do you see where I'm reading from?

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

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

12 A. Oh, yes. Okay.

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

23 Did I read that correctly?

24 A. You did.

25 Q. Did you review the transcript from

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

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

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

8 A. I don't recall that at all.

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

12 A. I do not.

13 ATTORNEY GREENWALD: Objection.
14 Form.

15 BY ATTORNEY ANTONUCCI:

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

21 ATTORNEY GREENWALD: Objection.
22 Form.

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

1 BY ATTORNEY ANTONUCCI:

2 Q. And do you know what threshold
3 ATSDR established to determine whether a
4 Marine regularly engaged in field exercises?

5 A. I do not.

6 Q. How would you delineate whether a
7 Marine regularly engaged in field exercises?

8 ATTORNEY GREENWALD: Objection.

9 Form.

10 THE WITNESS: I have no opinion on
11 that.

12 BY ATTORNEY ANTONUCCI:

13 Q. I'd like you to turn your attention
14 now to Exhibit 5, please. Again, Exhibit 5
15 is the rebuttal to the report of Dr. Richard
16 F. Ambinder for Robert Arnold Kidd that was
17 disclosed on May 16, 2025.

18 A. Okay.

19 Q. If you could, please turn to page 2
20 of Exhibit 5.

21 A. Okay.

22 Q. The first paragraph on page 2 reads
23 in part: In short, I agree with
24 Dr. Ambinder that it is an
25 oversimplification that all lymphomas or all

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

16 Did I read that correctly?

17 A. You did.

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

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

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

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

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

9 ATTORNEY GREENWALD: Objection.
10 Form, asked and answered.

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

13 BY ATTORNEY ANTONUCCI:

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

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

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

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

22 Q. Why do you doubt that?

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

1 contribute towards the causation of the
2 actual cancer.

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

6 ATTORNEY GREENWALD: Objection.
7 Form.

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

10 BY ATTORNEY ANTONUCCI:

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

13 A. Not that I'm aware of.

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

16 A. There is no page 9.

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

20 A. Okay.

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

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

7 Did I read that correctly?

8 A. Yes.

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

14 ATTORNEY GREENWALD: Objection.
15 Form.

16 THE WITNESS: Yes.

17 BY ATTORNEY ANTONUCCI:

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

22 A. Did you say page 2?

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

24 A. Okay. All right.

25 Q. On page 10 of Exhibit 4, you state

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

7 Did I read that correctly?

8 A. You did.

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

13 A. Yes.

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

18 ATTORNEY GREENWALD: Objection.
19 Form.

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

24 BY ATTORNEY ANTONUCCI:

25 Q. Okay. So you noted that benzene in

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

5 A. I did review that, yes.

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

11 A. Yes.

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

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

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

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

3 ATTORNEY GREENWALD: Objection.
4 Form.

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

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

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

24 BY ATTORNEY ANTONUCCI:

25 Q. Dr. Hu, are you aware that IARC

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

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

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

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

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

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

1 A. PCE; right?

2 Q. Yes.

3 Are you aware of any evidence that
4 supports the proposition that PCE produces
5 genotoxic effects in circulating
6 lymphocytes?

7 A. So on page 32 of my general
8 causation report, I discussed how PCE was
9 shown to be genotoxic to peripheral blood
10 lymphocytes in vitro, causing chromosomal
11 aberrations and the formation of micronuclei
12 in peripheral blood lymphocytes in vitro,
13 citing a study by Kocaman, et al., in 2021.

14 Q. Okay. Are you aware of any
15 evidence that supports the proposition that
16 TCE produces multiple chromosomal changes at
17 the pluripotential level?

18 A. I did not comment on that topic in
19 my report, and I don't recall studies
20 documenting TCE's impact on the
21 pluripotential cells of bone marrow. So I'm
22 not aware of such evidence, as I sit here
23 today, but there might be. I just didn't
24 comment on it.

25 Q. As you sit here today, are you

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

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

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

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

25 Q. If it's the case that some NHL

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

6 ATTORNEY GREENWALD: Objection to
7 form.

8 THE WITNESS: Well, just because
9 benzene might cause a mutation in the
10 post-pluripotential stage doesn't mean
11 it also -- it can't also cause
12 mutations in the pluripotential stage.
13 There's no reason to expect that the
14 benzene's impact on hematopoiesis is
15 limited to one stage of the cell
16 differentiation process, as far as I
17 know.

18 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

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

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

19 A. No.

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

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

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

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

3 ATTORNEY GREENWALD: Objection.
4 Form.

5 BY ATTORNEY ANTONUCCI:

6 Q. Correct?

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

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

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

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

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

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

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

23 A. Correct.

24 Q. And in the -- strike that.

25 If I refer to that study as Bove

1 2024, will you understand what I'm talking
2 about?

3 A. Yes. Although there is also a Bove
4 2024 mortality study; so I just want to make
5 sure that we're just going to concentrate on
6 the incidence study.

7 Q. You're right. Excuse me.

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

11 A. Yes.

12 Q. Thank you for that. I appreciate
13 it.

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

17 A. Correct.

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

21 (Exhibit Number 17 was marked for
22 identification.)

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

25 BY ATTORNEY ANTONUCCI:

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

5 A. Okay.

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

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

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

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

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

1 among the civilian workers.

2 BY ATTORNEY ANTONUCCI:

3 Q. Okay. I'm not sure I'm following.
4 My question was just whether Table 3 of this
5 study found that there was an elevated
6 adjusted hazard ratio for NHL.

7 A. Well, for NHL overall, the adjusted
8 hazard ratio was 1.01. So I would not
9 consider that elevated.

10 Q. Okay. The 95 percent confidence
11 interval is between 0.9 and 1.14 for that
12 adjusted hazard ratio; correct?

13 A. Correct.

14 Q. And there's a confidence interval
15 ratio of 1.3; correct?

16 A. Correct.

17 Q. And as you just alluded to, Bove,
18 et al., in the cancer incidence study
19 evaluated non-Hodgkin's lymphoma as a group
20 and also the subtypes of non-Hodgkin's
21 lymphoma; correct?

22 A. Correct.

23 Q. So the value for NHL, in general,
24 is the overall adjusted hazard ratio and
25 confidence intervals for all NHLs treated as

1 a group; correct?

2 A. Correct.

3 Q. So, for example, Burkitt's
4 lymphoma. I believe that's the highest
5 adjusted hazard ratio for NHL subtype that's
6 listed in Table 3 with the adjusted hazard
7 ratio of 1.53 and a confidence level between
8 .71 and 3.30; correct?

9 A. Correct.

10 Q. And the lowest adjusted hazard
11 ratio was for diffuse large B-cell lymphoma
12 with an adjusted hazard ratio of 0.89 and a
13 confidence interval between 0.72 and 1.10;
14 correct?

15 A. Correct.

16 Q. So between diffuse large B-cell and
17 Burkitt's non-Hodgkin's lymphoma, one was
18 found to have a positive association to
19 drinking water at Camp Lejeune, and the
20 other was found to have a negative
21 association; correct?

22 ATTORNEY GREENWALD: Objection.
23 Form.

24 THE WITNESS: Not negative
25 association but rather no association.

1 BY ATTORNEY ANTONUCCI:

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

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

7 Q. I'm sorry. Do you agree that
8 0.89 -- an adjusted hazard ratio of 0.89 is
9 a negative association?

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

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

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

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

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

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

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

13 ATTORNEY GREENWALD: Objection.
14 Form.

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

20 BY ATTORNEY ANTONUCCI:

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

1 of '85; correct?

2 A. Correct.

3 Q. And for civilian workers for all
4 NHLs as a group, Bove, et al., 2024
5 calculated an adjusted hazard ratio of 1.19;
6 correct?

7 A. Correct.

8 Q. And that has a confidence interval
9 ranging from 0.83 to 1.71; correct?

10 A. Correct.

11 Q. And for civilian workers with
12 marginal zone B-cell lymphoma, this study,
13 Exhibit 17, calculated an adjusted hazard
14 ratio of 0.33 with a confidence interval of
15 0.06 to 1.72; correct?

16 A. Yes, based on two cases.

17 Q. And why did you elaborate that
18 that's based on two cases?

19 A. Because the numbers are so small,
20 both among the Lejeune and Pendleton
21 workers, that, you know, these are pretty
22 small numbers with which to take
23 quantitative assessments of risk very
24 seriously. They're just relatively
25 unstable.

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

5 A. Correct.

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

10 ATTORNEY GREENWALD: Objection.
11 Form.

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

22 BY ATTORNEY ANTONUCCI:

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

1 a causal relationship?

2 ATTORNEY GREENWALD: Objection.

3 Form.

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

18 BY ATTORNEY ANTONUCCI:

19 Q. Understood.

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

22 A. Okay.

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

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

4 A. Correct.

5 Q. Looking first at non-Hodgkin's
6 lymphoma treated as a group, we see that for
7 the low duration at Camp Lejeune, the
8 adjusted hazard ratio is 1.02 with
9 confidence interval ranging between 0.89 and
10 1.17; is that correct?

11 A. Yes.

12 Q. For the immediate duration at Camp
13 Lejeune, the adjusted hazard ratio is 1.01
14 with a confidence level between 0.83 and
15 0.24; correct?

16 A. Correct.

17 Q. And for the high duration at Camp
18 Lejeune, the adjusted hazard ratio is 1.00
19 with a confidence interval between 0.79 and
20 1.26; correct?

21 A. Correct.

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

1 as a group; correct?

2 A. I agree with that.

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

7 ATTORNEY GREENWALD: Objection.
8 Form.

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

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

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

5 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

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

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

7 A. No.

8 Q. And why is that?

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

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

18 ATTORNEY GREENWALD: Objection.

19 Form.

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

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

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

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

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

14 (Recess taken from 2:08 p.m. to
15 2:14 p.m.)

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

20 BY ATTORNEY ANTONUCCI:

21 Q. Okay. Back on the record.

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

24 A. Yes.

25 Q. Did you discuss the substance of

1 your testimony with counsel during the
2 break?

3 A. No.

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

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

9 Q. Page 12, please.

10 A. Okay.

11 Q. So in paragraph 3 on page 12 of
12 Exhibit 4, you state that: Mr. Carter's
13 exposure timing and profile is closely
14 aligned with an exposure scenario that ATSDR
15 estimated to be associated with a lifetime
16 cancer risk of over 1 per 10,000 per -- for
17 exposures between the mid-1960s to around
18 1982 with a peak of 2.6 per 10,000 for
19 exposures surrounding 1970.

20 Is that right?

21 A. Correct.

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

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

6 Did I read that correctly?

7 A. Yes.

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

12 A. Correct.

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

18 A. Correct.

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

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

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

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

12 (Exhibit Number 18 was marked for
13 identification.)

14 BY ATTORNEY ANTONUCCI:

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

21 Do you see where I'm reading from?

22 A. Yes.

23 Q. And for the approximate level of
24 additional risk of cancer incidence between
25 1 in 50,000 and 1 in 5,000, the risk

1 qualification is described as, quote, very
2 low.

3 Did I read that correctly?

4 A. Yes.

5 Q. Okay. And for the discussion of
6 the Environmental Protection Agency's de
7 minimis risk level, you cited in footnotes
8 24 and 25, page 12 of Exhibit 4 -- excuse
9 me, just footnote 25, you cited Castorina R,
10 Woodruff TJ, Assessment of Potential Risk
11 Levels Associated With U.S. Environmental
12 Protection Agency Reference Values; is that
13 correct?

14 A. Yes.

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

20 A. Correct.

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

25 ATTORNEY GREENWALD: Objection.

1 Form.

2 THE WITNESS: I understand that.

3 BY ATTORNEY ANTONUCCI:

4 Q. And you're aware that EPA defines
5 acceptable exposure levels as generally
6 concentration levels that represent an
7 excess upper bound in lifetime cancer risk
8 to an individual of between 1 in 10,000 and
9 1 in 1 million; right?

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

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

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

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

1 roughly 40 percent.

2 How does exceeding a lifetime risk
3 of all cancers increase of between 1 in
4 10,000 and 1 in 1 million lead to the
5 conclusion of specific causation?

6 ATTORNEY GREENWALD: Objection.
7 Form.

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

16 BY ATTORNEY ANTONUCCI:

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

21 ATTORNEY GREENWALD: Objection.
22 Form.

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

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

6 BY ATTORNEY ANTONUCCI:

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

10 ATTORNEY GREENWALD: Objection.
11 Form, asked and answered.

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

15 BY ATTORNEY ANTONUCCI:

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

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

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

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

15 Q. You used the pronoun "he."

16 Were you referring to Mr. Carter?

17 A. Yes.

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

22 A. Yes.

23 Q. How so?

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

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

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

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

25 Q. How do you square that with the

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

4 ATTORNEY GREENWALD: Objection.
5 Form.

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

19 BY ATTORNEY ANTONUCCI:

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

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

1 semi-quantitative basis.

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

7 A. Yes.

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

10 A. Oh, sure. A zillion.

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

13 A. We know a lot of them.

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

18 ATTORNEY GREENWALD: Objection.

19 Form.

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

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

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

9 BY ATTORNEY ANTONUCCI:

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

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

16 A. Okay.

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

19 Do you see that?

20 A. Yes.

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

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

3 Did I read that correctly?

4 A. You did.

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

8 A. I think so.

9 Q. Okay. So the two studies you're
10 referring to here are Aschengrau, et al.,
11 1993 and Cohn, et al., 1994; correct?

12 A. Correct.

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

20 A. Yes.

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

25 A. Yes.

1 Q. And that used -- and the Aschengrau
2 1993 used ecological data to evaluate the
3 relationship between PCE, bladder cancer,
4 kidney cancer, and leukemia; correct?

5 A. Correct.

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

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

12 Q. Sure.

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

17 (Exhibit Number 19 was marked for
18 identification.)

19 BY ATTORNEY ANTONUCCI:

20 Q. And my question for you, Dr. Hu,
21 based on Exhibit 19 is: Does the Aschengrau
22 1993 study evaluate a relationship between
23 PCE and NHL?

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

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

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

7 Q. Understood.

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

11 A. No.

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

14 That was also an ecological study;
15 right?

16 A. Yes.

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

20 A. Correct.

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

25 A. Correct.

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

4 A. Can you repeat the question,
5 please?

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

12 A. Correct.

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

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

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

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

1 Q. Sure. This is Exhibit 20.

2 (Exhibit Number 20 was marked for
3 identification.)

4 BY ATTORNEY ANTONUCCI:

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

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

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

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

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

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

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

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

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

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

20 Q. Yes, sir.

21 A. Okay. All right.

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

1 Do you see that?

2 A. Yes.

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

17 A. Correct.

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

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

1 distinct.

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

6 A. I believe so.

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

10 A. Let me look at this study, please.

11 Q. Sure. That is Exhibit 20.

12 A. Oh, right here.

13 Q. It may help to look at page 559.

14 A. Thank you.

15 Well, there was a suggestion of a
16 dose response relationship among the females
17 with the -- with the rate ratio going from
18 1.0 to 1.02 to 1.36, but I wouldn't consider
19 that as substantial evidence of a dose
20 response relationship and that you don't see
21 evidence of that in the males. So I'd agree
22 with you.

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

1 A. Yes.

2 Q. So if you look at the next row down
3 below total NHL, there's low-grade NHL
4 total; right?

5 A. Yes.

6 Q. And for males, there's no dose
7 response relationship detected in this study
8 for low-grade NHL total; correct?

9 A. Correct. There's a suggestion of
10 it among the females again but not in the
11 males.

12 Q. And for PCE and total NHL in men,
13 the Cohn, et al., study also did not find a
14 dose response relationship; correct?

15 A. Well, before we leave Table 2, I
16 noted that for high-grade NHL, there seems
17 to be a dose response relationship in men
18 and women, but again, the numbers are low.

19 So what was your question?

20 Q. Actually, I'd like to look at
21 high-grade NHL. You noted that there's a
22 dose response relationship, but the numbers
23 are low.

24 What does that mean?

25 A. The number of cases are low; so the

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

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

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

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

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

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

21 A. I don't.

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

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

1 point out that this study was done some time
2 ago. Let's see, 1993 it was submitted, and
3 I just don't know how the classification
4 system might have evolved since then.

5 Q. If you turn to page 560, Table 4 is
6 titled Number of Recorded Cases,
7 Age-Adjusted Rate Ratios, and 95 Percent
8 Confidence Interval For Non-Hodgkin's
9 Lymphoma, NHL, in the Northern New Jersey
10 Study Area, 1979 to '87 by
11 Perchloroethylene, PCE, Exposure Category
12 and Sex, All Races; correct?

13 A. Yes.

14 Q. So the first row of this study on
15 page -- of this table on page 560, Table 4
16 says TCE exposure; right?

17 A. You mean the first column?

18 Q. Excuse me, yes. The first column.

19 A. You said the first row. I think
20 you meant the first column. First
21 column says it's about TCE exposure.

22 Q. But the title of the table says
23 it's about PCE exposure, doesn't it?

24 A. Yes.

25 Q. Do you happen to know -- I mean,

1 given that the Table 2 is TCE exposure,
2 would it make sense that Table 4 is actually
3 discussing PCE exposure?

4 A. It looks like it's a typo. I would
5 guess it's a typo.

6 Q. Okay. So for PCE exposure in
7 Table 4, the Cohn, et al., study did not
8 find a dose response relationship between
9 exposure to PCE and total NHL in them;
10 correct?

11 A. Well, before I conclude that it's a
12 typo, I'd better look at the text to see how
13 they refer to Table 4. Just a minute. This
14 is weird.

15 Do you happen to see where in the
16 text it's referring to Table 4?

17 Q. So I'll tell you, I couldn't find
18 it personally.

19 A. Oh, really? This editor was asleep
20 at the job. This is unfortunate. Oh, here
21 we go. Yeah, perchloroethylene, Tables 3
22 and 4. So that's a typo in Table 4. That
23 should be PCE exposure in the first column.
24 All right.

25 So what was your question again?

1 Q. So the Cohn, et al., 1994 study did
2 not find a dose response relationship
3 between exposure to PCE and total NHL in
4 men; correct?

5 A. I would agree with that.

6 Q. It also did not find a dose
7 response relationship between
8 intermediate-grade NHL diffuse large cell
9 reticulosarcoma in men; correct?

10 A. I agree with that.

11 Q. The Cohn, et al., 1994 study also
12 did not find a dose response relationship
13 between high-grade NHL total in men exposed
14 to PCE; correct?

15 A. I agree with that.

16 Q. And the Cohn, et al., 1994 study
17 did not find a dose response relationship
18 between exposure to PCE and total -- excuse
19 me, high-grade NHL non-Burkitt's in men;
20 correct?

21 A. I'd agree with that.

22 Q. And Cohn, et al., on page 557 note
23 that an earlier New Jersey Department of
24 Health study reported an association between
25 leukemia and drinking water contaminants

1 among females only, and so the cases were
2 grouped by sex for separate analysis; is
3 that right?

4 A. That's a pretty busy page, but if
5 you give me a little help, I can try to find
6 that sentence.

7 Q. Second-to-last paragraph on
8 page 557. It's the last sentence.

9 A. Okay.

10 Q. It says: Since the earlier NJ DOH
11 study (3) reported an association between
12 leukemia and drinking water contaminants
13 among females only, cases were grouped by
14 sex for separate analysis.

15 Did I read that correctly?

16 A. You did.

17 Q. And then 3 in that sentence is a
18 citation; right?

19 A. Yes.

20 Q. Would you expect to see an
21 association between NHL and drinking water
22 contaminants in women only?

23 A. I mean, the more we do
24 environmental epidemiology, the more that we
25 see there are sex-specific effects, and

1 there's very good biological reasons for
2 that. In terms of hormones, physiology,
3 anatomy, habits, et cetera.

4 So would I expect to see
5 differences in terms of the environmental
6 carcinogen between the sexes for NHL
7 specifically? Not necessarily.

8 I'm not aware of that, but if they
9 were found, I wouldn't be surprised. It
10 could be as a result of some of the factors
11 I just mentioned that there are biological
12 reasons for that behavior, but also, you
13 know, these ecological studies are pretty
14 crude tools.

15 You might see a relationship here
16 or there, but for the most part, it's
17 subject to, again, non-differential
18 misclassification which tends to dilute the
19 truer relationships between exposures and
20 diseases. We've seen in one sex versus the
21 other that doesn't necessarily mean that
22 there's always going to be sex-specific
23 differences in that exposure disease
24 relationship.

25 Q. Dr. Hu, is it your opinion that

1 non-differential misclassification always
2 biases towards the null?

3 A. That is true for the great majority
4 of cases, and I am aware there have been
5 some exceptions that have been hypothesized
6 and discussed. But in these types of
7 studies, I would expect the non-differential
8 misclassification to bias the effect
9 estimates towards the null.

10 Q. And why is that?

11 A. Because without directly
12 identifying the exposure of individuals, you
13 will, in the most extreme situation, assume
14 that someone that was highly exposed --
15 actually, lowly exposed. Someone who was
16 lowly exposed actually is highly exposed.
17 And if there was a true exposure disease
18 relationship, I think common sense would
19 dictate, gee, you're not going to see it
20 that way because you've misclassified
21 whether they're exposed or not.

22 That's the most extreme situation.
23 But in terms of a continuous exposure
24 measure, it's very similar. You know, you
25 just start to dilute the contrast between

1 exposure categories.

2 Q. Is there a way to test whether the
3 misclassification bias is biasing towards or
4 away from the null hypothesis in a human
5 epidemiology study?

6 A. Well, the most important way to try
7 to determine whether that might be the case
8 is to look at the methods used to classify
9 exposure and then do a validation study to
10 see whether the true exposure is different
11 from the estimated exposure in a
12 non-differential way. That would be the one
13 way to do it. Non-differential in terms of
14 the relationship to the outcome.

15 But, you know, we don't have the
16 luxury of that for most of these kinds of
17 investigations.

18 Q. Can a quantitative bias analysis be
19 used to assess whether misclassification
20 bias is biasing towards or away from the
21 null hypothesis?

22 A. No. I think that tool, which Bove,
23 et al., used, was used to determine whether
24 there was uncontrolled confounding, not
25 bias.

1 Q. So on page 10 of Exhibit 3, the
2 specific causation initial report for
3 Mr. Kidd.

4 A. Yeah.

5 Q. Paragraph 2 starts with "That
6 Mr. Kidd's."

7 Do you see that?

8 A. Yes.

9 Q. Here you wrote: That Mr. Kidd's
10 exposures to TCE, PCE, and benzene were at
11 least as likely as not causative of his NHL
12 is also consistent with the findings of the
13 most recently published epidemiological
14 study of the cancer incidence experience of
15 personnel stationed or employed at Camp
16 Lejeune versus Camp Pendleton that I
17 discussed earlier.

18 Did I read that correctly?

19 A. You did.

20 Q. And on pages -- starting on page 8
21 of the specific causation report for
22 Mr. Kidd, again, Exhibit 3, under subheading
23 A. TCE, PCE, Benzene, and NHL, do you see
24 the sentence starting "Furthermore"?

25 A. Yes.

1 Q. Here you wrote: Furthermore, in my
2 January 31, 2025, supplement to my general
3 causation report, I provided the opinion
4 that the results of the Bove, et al., cancer
5 incidence study allow me to specifically
6 conclude that the combination of TCE, PCE,
7 and benzene at the levels of community
8 exposure experienced at Camp Lejeune are
9 more likely than not a risk factor for NHL.

10 As noted in my supplemental report,
11 this was a cancer incidence study in which
12 elevated hazard ratios, AHRs, with
13 confidence interval ratios (the ratio of the
14 upper to lower limits of the 95 percent
15 confidence interval) less than or equal to 3
16 (an indicator of precision) were reported
17 for a number of cancers.

18 Among them were the mantle cell
19 subtype of NHL and the marginal zone B-cell
20 subtype of NHL, which among the Camp Lejeune
21 (vs. Camp Pendleton) Marines/Navy personnel
22 had adjusted hazard ratios of 1.26 (95
23 percent confidence interval: 0.73 to 2.19;
24 confidence interval ratio: 3.0) and 1.45
25 (95 percent confidence interval: 0.92 to

1 2.28; confidence interval ratio: 2.5.)

2 Did I read that correctly?

3 A. You did.

4 Q. Mr. Kidd had -- first of all,
5 Mr. Kidd had diffuse large B-cell lymphoma;
6 correct?

7 A. Right.

8 Q. Not marginal zone B-cell or mantle
9 cell lymphoma?

10 A. Correct.

11 Q. So the cancer incidence study,
12 which is Exhibit 17, did not include any
13 statistical significance testing; correct?

14 A. Well, it had confidence intervals
15 which are just as good, if not better.

16 Q. Are you aware that Dr. Bove
17 testified he did not perform statistical
18 significance testing for his 2024 cancer
19 incidence study?

20 A. I'm aware of that.

21 ATTORNEY GREENWALD: Objection.
22 Form.

23 THE WITNESS: You know, that's
24 just a matter of entering P values,
25 but if you have confidence intervals,

1 like I said, it's just as good.

2 BY ATTORNEY ANTONUCCI:

3 Q. Okay. In the 2024 cancer incidence
4 study, there was no individualized exposure
5 assessment; correct?

6 A. I would agree with that.

7 Q. And the 2024 cancer incidence study
8 by Dr. Bove did not control for or consider
9 family history of NHL; correct?

10 A. I agree with that.

11 Q. The 2024 Bove cancer incidence
12 study did not control for family history of
13 cancer, in general; correct?

14 A. I agree with that.

15 Q. The 2024 Bove cancer incidence
16 study did not control for inherited
17 immunodeficiency syndrome; correct?

18 A. I agree with that.

19 Q. The 2024 Bove cancer incidence
20 study did not control for a history of organ
21 transplants; correct?

22 A. I agree with that.

23 Q. The 2024 Bove cancer incidence
24 study did not control for or consider organ
25 transplants; correct?

1 A. I agree with that.

2 Q. The 2024 Bove cancer incidence
3 study did not control for or consider
4 Sjogren's syndrome; correct?

5 A. I agree with that.

6 Q. The 2024 Bove cancer incidence
7 study did not control for or consider
8 systemic lupus erythematosus; correct?

9 A. I agree with that.

10 Q. The 2024 Bove cancer incidence
11 study did not control for or consider HIV;
12 correct?

13 A. I agree with that.

14 Q. The 2024 Bove cancer incidence
15 study did not control for or consider other
16 potential occupational exposures among
17 Marines and Navy service members; correct?

18 A. I agree with that.

19 Q. And if you look at Table 3 of the
20 cancer incidence study and, again, that's
21 Exhibit 17.

22 A. I'm sorry. Which table?

23 Q. Table 3 of Exhibit 17.

24 A. Okay.

25 Q. As we discussed, Table 3 shows the

1 overall NHL adjusted hazard ratio of 1.01;
2 correct?

3 A. Correct.

4 Q. The diffuse large B-cell lymphoma
5 adjusted hazard ratio is 0.89; correct?

6 A. Correct.

7 Q. And what that is showing is that
8 the risk of diffuse large B-cell lymphoma
9 was 0.89 times as high in Marines from Camp
10 Lejeune compared to Marines from Camp
11 Pendleton between 1975 and 1985; correct?

12 A. Correct.

13 Q. The Bove 2024 cancer incidence
14 study also gave results by duration of
15 residency at Camp Lejeune for the Marine and
16 Navy cohort; correct?

17 A. Correct.

18 Q. Are you aware of any other studies
19 with PCE exposure levels similar to those at
20 Camp Lejeune?

21 ATTORNEY ANTONUCCI: For the
22 record, the witness is currently
23 reviewing Exhibit 10.

24 THE WITNESS: Yeah, I just wanted
25 to see whether the U.K. Biobank study

1 might have included PCE. I don't
2 think so. Not as I sit here today.

3 BY ATTORNEY ANTONUCCI:

4 Q. Are you aware of any other studies
5 with TCE exposure levels similar to those at
6 Camp Lejeune?

7 A. Well, we just talked about
8 ecological studies -- right? -- which
9 involved TCE, I thought?

10 Q. Are you referring to Cohn or
11 Aschengrau?

12 A. Yeah.

13 Q. Other than Cohn or Aschengrau, are
14 you aware of any other studies with TCE
15 exposure levels similar to those at Camp
16 Lejeune?

17 A. Not that I'm aware of -- not that I
18 recall, sitting here today.

19 Q. And other than Cohn, Aschengrau, or
20 ATSDR's work, are you aware of any other
21 studies with benzene exposure levels similar
22 to those at Camp Lejeune?

23 A. Well, have we talked about the U.K.
24 Biobank study?

25 Q. Not yet.

1 A. That has exposures, I think, within
2 order of magnitude of what was experienced
3 at Camp Pendleton -- Camp Lejeune. Sorry.

4 Q. Are you aware of any other studies
5 with benzene exposure levels similar to
6 those at Camp Lejeune?

7 A. Not as I sit here today.

8 ATTORNEY ANTONUCCI: I would like
9 to take a quick five-minute break if
10 that's okay.

11 THE VIDEOGRAPHER: We are now
12 going off the record. This is the end
13 of media Unit Number 4, and the time
14 is 3:04 p.m.

15 (Recess taken from 3:04 p.m. to
16 3:10 p.m.)

17 THE VIDEOGRAPHER: We are now back
18 on the record. This is the beginning
19 of media Unit Number 5, and the time
20 is 3:10 p.m.

21 BY ATTORNEY ANTONUCCI:

22 Q. Dr. Hu, we are back on the record,
23 and you understand you're still under oath;
24 correct?

25 A. Yes.

1 Q. Did you discuss the substance of
2 your testimony with your attorney during the
3 break?

4 A. I did not.

5 Q. Please turn to Exhibit 3. That's
6 the specific causation initial report for
7 Mr. Kidd.

8 A. Okay. All right.

9 Q. Specifically page 10.

10 A. Okay.

11 Q. Excuse me. I meant page 11. My
12 apologies.

13 A. Okay.

14 Q. Paragraph 5 on page 11 lists risk
15 factors, and I'm just going to read from
16 your report here.

17 It says: Other than TCE, PCE, and
18 benzene as risk factors (that I had
19 discussed in my general causation report),
20 other risk factors for which there is
21 evidence of an association with NHL include
22 being white (versus African-American or
23 Asian American), having family history of a
24 first-degree relative with NHL, previous
25 treatment with cancer chemotherapy drugs,

1 radiation exposure, weakened immune systems
2 (e.g., those weakened by immunosuppressive
3 drugs or HIV/AIDS), certain inherited
4 syndromes associated with immunodeficiency
5 (e.g., ataxia-telangiectasia,
6 Wiskott-Aldrich syndrome), autoimmune
7 diseases (such as rheumatoid arthritis,
8 systemic lupus erythematosus), previous
9 lymphoma, chronic infections that cause
10 continuous immune system activity (e.g.,
11 *Helicobacter pylori*; *Chlamydothila psittaci*,
12 *Campylobacter jejuni*, Hepatitis C), and
13 breast implants.

14 Did I read that correctly?

15 A. Yes. I am impressed.

16 Q. Thanks.

17 So these are -- what I just read
18 were the risk factors for NHL; correct?

19 A. These are the risk factors that
20 appear on the American Cancer Society
21 website.

22 Q. Okay. And are these risk factors
23 for NHL, in general, as a group of cancers?

24 A. Yes.

25 Q. As opposed to any given subtype;

1 correct?

2 ATTORNEY GREENWALD: Objection.

3 Form.

4 THE WITNESS: Correct.

5 BY ATTORNEY ANTONUCCI:

6 Q. So, for example, would you consider
7 H. pylori infection to be associated with
8 diffuse large B-cell lymphoma?

9 A. Specifically? I think I would
10 consider that as a risk factor by default,
11 but if -- you know, I haven't looked
12 intensely into the literature myself.

13 Q. Do you know whether H. pylori
14 infection is a risk factor for diffuse large
15 B-cell lymphoma?

16 A. I don't know if that particular
17 specific question has ever been examined.

18 Q. Do you know whether H. pylori is
19 associated with a different subtype of
20 non-Hodgkin's lymphoma?

21 A. As I sit here today, I don't recall
22 looking at the literature to look at that
23 topic.

24 Q. Okay. Did you consider risk
25 factors for diffuse large B-cell lymphoma in

1 particular?

2 ATTORNEY GREENWALD: Objection.

3 Form, asked and answered.

4 THE WITNESS: Well, these are
5 general risk factors for NHL as they
6 appear on the American Cancer Society
7 website. If Mr. Kidd had evidence of
8 any of these other risk factors, I
9 would have plowed more deeply into the
10 literature, but since he didn't, I did
11 not.

12 BY ATTORNEY ANTONUCCI:

13 Q. Are the causes of diffuse large
14 B-cell lymphoma generally known?

15 ATTORNEY GREENWALD: Objection.
16 Form.

17 THE WITNESS: Well, there's some
18 that are probably listed if you want
19 to get specifically into diffuse large
20 B-cell lymphoma. I don't remember
21 exactly what those risk factors are
22 specifically just for DLBCL, but I
23 know that some of that has been done.

24 BY ATTORNEY ANTONUCCI:

25 Q. In what percent of DLBCL cases is

1 the cause of cancer known?

2 A. I don't think -- I don't recall
3 anybody ever trying to estimate that.

4 Q. Are the causes of MACL known?

5 A. You mean mantle cell lymphoma?

6 Q. Yes, sir.

7 Are the causes of mantle cell
8 lymphoma known?

9 A. Well, some have been -- some risk
10 factors have been identified.

11 Q. Dr. Hu, you agree that the
12 different subtypes of non-Hodgkin's lymphoma
13 have different risk factors; correct?

14 ATTORNEY GREENWALD: Objection.
15 Form.

16 THE WITNESS: I agree that
17 different studies have been done
18 looking at different subtypes. All of
19 these are somewhat rare; so it's --
20 the literature on the epidemiology of
21 each of the subtypes is -- I guess I
22 would say it's sparse.

23 So I wouldn't say that they're
24 known, but there have been various
25 risk factors have been identified in

1 various studies.

2 BY ATTORNEY ANTONUCCI:

3 Q. So my question was that the
4 different subtypes have different risk
5 factors.

6 Do you agree with that?

7 ATTORNEY GREENWALD: Objection.
8 Form, asked and answered,
9 mischaracterizes his testimony.

10 THE WITNESS: I'll say it again.
11 Different risk factors have been
12 identified in studies. That doesn't
13 mean that it is the absolute truth
14 that each subtype of NHL definitely
15 has different risk factors. We don't
16 know that for certain. I can say
17 that, in general.

18 So yes, maybe some study found
19 these risk factors. Some other study
20 found other risk factors for other
21 tumors. But given the rarity of these
22 tumors, I think it's very hard to
23 conclude that the subtypes of NHL
24 either have all the same risk factors
25 or that they're all different.

1 BY ATTORNEY ANTONUCCI:

2 Q. Okay. Earlier you mentioned citing
3 to the American Cancer Society for the list
4 of non-Hodgkin's lymphoma risk factors;
5 correct?

6 A. Correct.

7 ATTORNEY ANTONUCCI: All right.

8 I'm marking for identification
9 Exhibit 21. This is a printout of the
10 American Cancer Society's website
11 titled Non-Hodgkin's Lymphoma Causes,
12 Risk Factors, and Prevention.

13 (Exhibit Number 21 was marked for
14 identification.)

15 THE WITNESS: And what date was
16 that done?

17 BY ATTORNEY ANTONUCCI:

18 Q. It should say at the bottom of the
19 printout.

20 Dr. Hu, I will ask that you review
21 Exhibit 21 and let me know if this is the
22 website that you reviewed when drafting your
23 report.

24 A. It is.

25 Q. So if you look at page 2, there's,

1 again, a list of risk factors; correct?

2 A. Correct.

3 Q. Above that list of risk factors,
4 the American Cancer Society states:
5 Researchers have found several factors that
6 can affect a person's chances of getting
7 non-Hodgkin's lymphoma, NHL. There are many
8 types of lymphoma, and some of these factors
9 have been linked only to certain types.

10 Did I read that correctly?

11 A. You did.

12 Q. Okay. Other than the risk factors
13 listed here, did you consider any other risk
14 factors for NHL?

15 A. No.

16 Q. And to clarify, my last question
17 was referring to page 2 of Exhibit 21.

18 You did not review -- or consider
19 any other risk factors other than those;
20 correct?

21 ATTORNEY GREENWALD: Objection.

22 Form.

23 THE WITNESS: Before I respond,
24 let me also just refer to my general
25 causation report to be certain. Hold

1 on.

2 Okay. Can you repeat your
3 question, please?

4 BY ATTORNEY ANTONUCCI:

5 Q. Other than the risk factors listed
6 on page 2 of Exhibit 21, did you consider
7 any other risk factors in offering your
8 opinions?

9 A. In terms of the differential
10 etiology, no.

11 Q. One of the risk factors on page 2
12 of Exhibit 21 is the history of breast
13 implants; correct?

14 A. Yes.

15 Q. Do you know whether a history of
16 breast implants is associated with all NHL,
17 in general, or just specific types?

18 A. I don't recall. I don't know.

19 Q. Is a history of breast implants a
20 risk for mantle cell lymphoma?

21 A. I don't -- I don't know. I don't
22 recall.

23 Q. Is a history of breast implants a
24 risk factor for any non-Hodgkin's lymphoma
25 that's not situated near the breast?

1 A. Non-Hodgkin's lymphoma can crop up
2 anywhere; so this is not a listing of risk
3 factors for breast cancer. This is a
4 listing of risk factors for non-Hodgkin's
5 lymphoma.

6 So it's not a matter of cancer
7 cropping up in the breast. It's the fact
8 that the exposure here is in the breast.
9 It's the breast implants and their
10 associated chemicals and the reaction that's
11 caused by those chemicals.

12 Q. So I apologize if my question was
13 unclear.

14 I'd like to know if a history of
15 breast implants is a risk factor for a
16 non-Hodgkin's lymphoma that is not near the
17 breast.

18 ATTORNEY GREENWALD: Objection.
19 Form.

20 THE WITNESS: I mean, the fact
21 that it's on this list is a reflection
22 of the fact that breast implants are
23 associated with non-Hodgkin's lymphoma
24 of some type. Non-Hodgkin's lymphoma
25 of any type could have cropped up all

1 over the body, maybe near the breast
2 but typically in lymph nodes or other
3 places where there are collections of
4 lymphocytes.

5 I think I've answered your
6 question, hopefully.

7 BY ATTORNEY ANTONUCCI:

8 Q. Do you know what caused these risk
9 factors to be placed on the American Cancer
10 Society's website?

11 ATTORNEY GREENWALD: Objection.
12 Form.

13 THE WITNESS: The American Cancer
14 Society has a scientific advisory
15 board. They go over the science.
16 They go over research objectives.
17 They actually sponsor some of their
18 own research. They sponsor some
19 research fellowships that some of my
20 trainees have obtained.

21 So I don't know exactly what
22 review body in the American Cancer
23 Society would be responsible for
24 identifying those factors that have a
25 sufficient amount of evidence to show

1 up on their website, but I am pretty
2 certain that they have done it in a
3 systematic way that there's a process
4 for doing that.

5 BY ATTORNEY ANTONUCCI:

6 Q. Okay. And it's true that the
7 American Cancer Society's website says that
8 some risk factors have been linked only to
9 certain types of non-Hodgkin's lymphomas;
10 correct?

11 A. Correct.

12 Q. Dr. Hu, you rolled in exposure to
13 TCE, PCE, and benzene as risk factors in
14 your differential etiology for Mr. Kidd
15 based on Dr. Reynolds' exposure
16 calculations; is that correct?

17 ATTORNEY GREENWALD: Objection.
18 Form.

19 THE WITNESS: I wouldn't quite put
20 it that way. I mean, I considered it
21 a significant risk factor based on my
22 general causation analysis, based on
23 the exposure assessment and the
24 estimates of likely exposure that the
25 Camp Lejeune residents endured, and

1 also the specific exposure assessments
2 done with these plaintiffs.

3 BY ATTORNEY ANTONUCCI:

4 Q. Okay. Did you rule out exposure to
5 TCE, PCE, or benzene as risk factors in your
6 differential etiology for Mr. Kidd?

7 ATTORNEY GREENWALD: Objection.
8 Form.

9 THE WITNESS: Rule it out?
10 Clearly not. I mean, I accepted the
11 exposure assessment that was done on
12 Mr. Kidd.

13 BY ATTORNEY ANTONUCCI:

14 Q. Okay. And because exposure to TCE,
15 PCE, and benzene are risk factors for NHL
16 and because Mr. Carter ultimately got NHL,
17 you opine that the exposure caused his
18 disease; correct?

19 ATTORNEY GREENWALD: Object to
20 form.

21 THE WITNESS: Well, there's a
22 whole number of other factors I looked
23 at. I looked at exposure duration. I
24 looked at latency. I considered these
25 other alternative risk factors. It's

1 a process that needs to be done
2 systematically and in a case where
3 specific causation is proposed.

4 BY ATTORNEY ANTONUCCI:

5 Q. I'd like you to turn to page 7 of
6 Exhibit 21, the American Cancer Society's
7 non-Hodgkin's lymphoma risk factors.
8 Underneath what causes non-Hodgkin's
9 lymphoma, the first sentence states:
10 Researchers have found that non-Hodgkin's
11 lymphoma, NHL, is linked with a number of
12 risk factors, but the cause of most
13 lymphomas is not known. This is complicated
14 by the fact that there are many types of
15 lymphomas are actually a diverse group of
16 cancers which might have different causes.

17 Did I read that correctly?

18 A. You did, and there's a typo there.
19 I think they forgot the word "that."

20 Q. So it's true that the cause of most
21 cases of DLBCL is unknown; correct?

22 ATTORNEY ANTONUCCI: Objection.

23 Form.

24 THE WITNESS: Well, when there's a
25 very -- a person who presents with

1 DLBCL, diffuse B-cell lymphoma, I
2 think it's true that, in many of those
3 cases, there will not be any of the
4 causes listed here identified, and
5 they may not have been exposed to TCE,
6 benzene, or PCE in any meaningful way
7 as well.

8 So that would be someone whose,
9 you know, cancer it's unclear what
10 caused it. Of course, you know, we
11 have limited knowledge about all the
12 genetics, poly genetics. And that's
13 all the subject of current research.

14 BY ATTORNEY ANTONUCCI:

15 Q. Are you familiar with the
16 InterLymph Non-Hodgkin's Lymphoma Subtypes
17 Project?

18 A. I am familiar with the -- one of
19 the non-Hodgkin's lymphomas consortia. It
20 might have the same name. What was the name
21 you mentioned again?

22 Q. InterLymph non-Hodgkin's lymphoma
23 subtypes.

24 A. What is that exactly?

25 Q. I believe what you just described

1 as a consortia.

2 A. Consortia?

3 Q. And you cited to several studies as
4 part of that project in your causation
5 report.

6 A. Right. So I have some familiarity
7 with it.

8 Q. Are you aware that the InterLymph
9 Non-Hodgkin's Lymphoma Subtypes Project
10 concluded that although risk factors for
11 diffuse large B-cell lymphoma have been
12 suggested, that their independent effects
13 modification by sex and association with
14 anatomical sites are largely unknown?

15 A. I'm not aware that they stated
16 that, but I have no reason to disagree with
17 it.

18 Q. Did you consider the fact that most
19 cases of DLBCL is unknown in performing your
20 differential etiology for Mr. Kidd?

21 ATTORNEY GREENWALD: Objection.

22 Form.

23 THE WITNESS: I think what your
24 question poses for me is the concept
25 that since you can't definitively say

1 that one thing is the cause, and there
2 is no way of identifying an exact
3 cause, along with other scientific
4 opinions expressed in the literature,
5 like isn't it true that the cause is
6 just random.

7 And in my view, in the field of
8 cancer epidemiology, what has been
9 debated is the extent to which cancer
10 is random, and I've already discussed
11 that actually in one of my rebuttal
12 reports because, in fact, that view
13 which was expounded by two scientists,
14 I think they were both at Stanford,
15 based on some in vitro studies in math
16 modeling was vigorously debated and
17 disagreed with by the International
18 Agency For Research on Cancer and
19 other scientists based on very
20 reasonable considerations in my view.

21 So back to your question, did I
22 consider whether the cause of
23 Mr. Kidd's cancer was simply unknown?
24 That's a theoretical, but in his
25 particular situation, we know he got

1 cancer. We know that there's some
2 risk factors that are identified. He
3 has a significant risk factor that was
4 identified. He was exposed to that
5 risk factor at a pretty significant
6 level for many years, and many of the
7 other attributes of his clinical
8 course and his personal history are
9 consistent with this being a
10 substantial contributing factor
11 towards his cancer, and that's why I
12 concluded what I concluded.

13 BY ATTORNEY ANTONUCCI:

14 Q. So there was a lot there.

15 A. Yeah.

16 Q. Maybe we -- we'll try to take this
17 one at a time. You mentioned the two
18 scientists at the Stanford that had the
19 random cancer theory.

20 Was that Tomasetti --

21 A. Tomasetti & Vogelstein.

22 Q. -- and Vogelstein?

23 A. Yes.

24 Q. We'll come back to discuss that.

25 So I think really what I was

1 getting at was sort of the last part of the
2 answer you just gave, the fact that Mr. Kidd
3 was exposed, he didn't have other risk
4 factors, the exposure was substantial, and
5 he ultimately got the disease; right?

6 A. Yes.

7 Q. Is that -- is that an accurate
8 characterization of --

9 A. That's an accurate general
10 characterization, although this -- other
11 things that I discussed in the -- in my
12 reasoning.

13 Q. And the fact that the cause of most
14 cases of NHL is unknown is sort of
15 theoretical in your view and not relevant to
16 this analysis; is that right?

17 ATTORNEY GREENWALD: Objection.
18 Form.

19 THE WITNESS: I mean, it's not
20 theoretical. I mean, it's kind of
21 true. We can't explain every case of
22 cancer in a definitive way. There's
23 no epidemiology studies where you put
24 in all the risk factors and you
25 explain 100 percent of the variance of

1 the cancer rate. It just doesn't
2 exist. But that doesn't mean that
3 risk factors that are identified are
4 insignificant or not contributory or
5 irrelevant.

6 BY ATTORNEY ANTONUCCI:

7 Q. I want to take a step back and
8 clarify.

9 When we talk about unknown causes
10 of cancer, is that different from random
11 causes?

12 A. Well, random causes really is a
13 reference to complete randomness. It's not
14 that there's unidentified causes. There's
15 just randomness. And randomness -- and this
16 is part of the -- part of the critique that
17 scientists have made of Tomasetti &
18 Vogelstein is that randomness is not
19 something, for instance, that you would
20 expect to be different in one country versus
21 another or over time with different rates of
22 cancer. And what's really notable about
23 cancer rates is that they do differ between
24 populations, between time periods, between
25 geographic areas, and those are not -- you

1 know, those are not things that one would
2 expect if cancer was just random or if a
3 significant portion of cancer was just
4 random.

5 Q. So ultimately, Dr. Hu, you
6 concluded that the causes of Mr. Carter's
7 and Mr. Kidd's non-Hodgkin's lymphomas were
8 not unknown; correct?

9 A. Well, I concluded -- I'm not sure
10 it's the equivalent that the exposures that
11 were identified at Camp Lejeune were more
12 likely than not a significant contributing
13 factor towards the cancers.

14 Q. Why is that not equivalent to being
15 a known cause?

16 A. I think the way it's phrased, these
17 are a known cause. It makes it ambiguous as
18 to whether it's known because the risk
19 factors that they had and they had proper
20 exposure, et cetera, et cetera, or whether,
21 oh, yeah, we did a test, and it's known that
22 these are the causes; right?

23 So that ambiguity makes me
24 uncomfortable with characterizing their
25 relationship that way; so I feel more

1 comfortable characterizing it the way I just
2 did and in my reports.

3 Q. Dr. Hu, you would agree that even
4 among epidemiological studies that find
5 increased risk of non-Hodgkin's lymphoma,
6 the risk ratios and hazard ratios are very
7 rarely over 2; correct?

8 ATTORNEY GREENWALD: Objection.

9 Form.

10 THE WITNESS: Well, that's often
11 the case with cohort studies. That's
12 not necessarily the case with
13 case-control studies since
14 case-control studies are specifically
15 designed to focus on cancers
16 themselves and less on the population
17 and it's longitudinal experience.
18 Those are general statements and not
19 always true.

20 BY ATTORNEY ANTONUCCI:

21 Q. Which case-control studies indicate
22 an increased risk of non-Hodgkin's lymphoma
23 over 2?

24 A. I'd have to go back and look at my
25 general causation report. Shall I?

1 Are you talking about a specific
2 exposure, TCE, PCE, benzene or just in
3 general?

4 Q. Are you aware of any
5 epidemiological studies that find an
6 increased risk of non-Hodgkin's lymphoma for
7 any of TCE, PCE, or benzene over the risk or
8 hazard ratio of 2?

9 A. So just turning to my general
10 causation report, if you look at the page 38
11 in my Bradford Hill factor analysis of
12 benzene, I was looking at the strength of
13 association criteria -- consideration 1.
14 And among the studies that I discussed was a
15 cohort study by Wong, et al., that found a
16 relative risk of 4.12; a cohort study by
17 Hayes, et al., that found a relative risk of
18 4.7; a case-control study by Fabbro-Peray
19 that found a odds ratio of 5.07; a
20 case-control study by Xu, et al.,
21 that found an odds ratio of 2.78.

22 So that's an example of epi studies
23 that found risk ratios greater than 2 for
24 one of the contaminants in non-Hodgkin's
25 lymphoma.

1 Q. Are those all specific to benzene?

2 A. Yes. And then if you go to my
3 other analyses, let's see. Then in the
4 strength of association consideration for
5 TCE, I discussed the case-control study by
6 Hardell that found an odds ratio for TCE
7 with NHL of 7.2, an occupational cohort
8 study that found that workers exposed to TCE
9 had an elevated standardized incidence ratio
10 of 3.5. So those are some examples for TCE.

11 And as for PCE, let's see if I can
12 find anything. And then for PCE under
13 Strength of Association under page 32, I
14 quoted Antilla, et al., which showed among
15 Finnish workers a standardized incidence
16 rate for PCE and NHL of 2.55; Selden and
17 Ahlorg's cohort study in Swedish dry
18 cleaning workers found a standardized
19 incidence rate of 2.05.

20 And I quoted some other studies
21 too. But those are examples, I guess.

22 Q. Okay. Did any of the studies you
23 just listed evaluate diffuse large B-cell
24 lymphoma or mantle cell lymphoma
25 individually?

1 ATTORNEY GREENWALD: Objection.
2 Form.

3 THE WITNESS: I don't recall, but
4 if you give me the slew of studies I
5 just quoted, I can take a look.

6 BY ATTORNEY ANTONUCCI:

7 Q. Are you, Dr. Hu, confident that the
8 cause of Mr. Carter's non-Hodgkin's lymphoma
9 must have been one of or a combination of
10 the risk factors you listed?

11 ATTORNEY GREENWALD: Objection.
12 Form.

13 THE WITNESS: Well, I mean, I
14 stated my opinion in precisely the way
15 my view exists, which is that -- let's
16 go to the -- make sure I don't
17 misquote myself.

18 It is my opinion to a reasonable
19 degree of medical certainty that the
20 combination of Mr. Carter's exposures
21 to TCE, PCE, and benzene from Camp
22 Lejeune more likely than not was a
23 substantial contributing factor to the
24 causation of mantle cell lymphoma.

25 That is precisely the way I think

1 about it.

2 BY ATTORNEY ANTONUCCI:

3 Q. In your opinion, is it at all
4 possible that Mr. Carter or Mr. Kidd's NHL
5 was caused by a random genetic mutation?

6 A. It is possible.

7 Q. In your opinion, is it at all
8 possible that Mr. Carter or Mr. Kidd's
9 non-Hodgkin's lymphoma was caused by a risk
10 factor other than the ones you listed in
11 your reports?

12 ATTORNEY GREENWALD: Objection.
13 Form.

14 THE WITNESS: Well, let me just
15 back up for a moment. As I said
16 earlier in this deposition, in my
17 view, and I believe this is true of
18 most of the people who are in
19 environmental epidemiology or do
20 research in carcinogenesis -- and
21 carcinogenesis is not only a multistep
22 but multifactorial process.

23 So when I said would I be
24 surprised that, let's say, a genetic
25 factor was the cause, I would say I

1 would not be surprised if it was a
2 cause particularly since most chronic
3 disease outcomes we now believe are a
4 complex interplay between environment
5 and genes.

6 So I would feel comfortable saying
7 it wouldn't surprise me if it was a
8 cause. But would I be surprised if it
9 was the cause and nothing else was the
10 cause? I'd be surprised. Of course,
11 there's no way of knowing.

12 BY ATTORNEY ANTONUCCI:

13 Q. And to be clear, is it your
14 opinion -- strike that.

15 It's not your opinion that everyone
16 who is exposed to contaminated water at Camp
17 Lejeune developed non-Hodgkin's lymphoma;
18 correct?

19 A. Correct.

20 Q. How did you decide which risk
21 factors to consider in performing your
22 differential etiology? Was it based on the
23 ACS list?

24 ATTORNEY GREENWALD: Objection.

25 Asked and answered.

1 THE WITNESS: Yes, and that's
2 because it's a fairly comprehensive
3 list. It's been agreed upon by a
4 sub-guidance body in one of the most
5 prominent, you know, objective
6 rigorous cancer advocacy and research
7 organizations, and I feel that was a
8 reasonable way of approaching it.

9 BY ATTORNEY ANTONUCCI:

10 Q. Okay. So for Mr. Kidd, you
11 wrote -- you listed the risk factors on
12 page 11 of your initial report for Mr. Kidd
13 and continued: Of these risk factors,
14 Mr. Kidd had only one, i.e., he's white.

15 Is that correct?

16 A. Yes.

17 Q. You go on to say: Overall,
18 Mr. Kidd had no clear evidence of any known
19 risk factor for NHL; correct?

20 A. I guess I could have said other
21 than the fact that he was white.

22 Q. Okay. So it is your opinion that
23 being white is a risk factor for NHL?

24 A. Well, that's, you know, what the
25 ACS listed among risk factors. I do believe

1 seen some of that identified in epidemiology
2 that I've read; so I have no reason to
3 disagree with it.

4 Q. Sex and specifically being male is
5 also a risk factor for NHL; correct?

6 A. That is true. He is a male.

7 Q. Did you consider the fact this
8 Mr. Kidd is a male in your differential
9 etiology?

10 A. Well, I mean, yes, but I think
11 that's -- doesn't -- it doesn't undercut the
12 rest of my opinions.

13 Q. How did you weigh Mr. Kidd's sex
14 when performing your differential etiology?

15 A. Well, if it turns out that sex was
16 an overwhelming risk factor, in other words,
17 like testicular cancer, you're not going to
18 get any women. That's only a risk factor
19 for men. Then, of course, I would look at
20 that very strongly, very different.

21 But in this case, yes, being male
22 is associated with a somewhat elevated risk.
23 Okay. But would that undercut the other
24 carcinogenic risk factors that he had? No.

25 Q. Is that explained in your report?

1 A. No, but I'm explaining it to you
2 now.

3 Q. You also noted that Mr. Kidd's
4 exposure to radiation would not be
5 considered an unusually high amount of
6 radiation exposure in relation to the
7 general public; correct?

8 A. Correct.

9 Q. That's because he had X-rays in
10 2009, five to six dental X-rays, and no CT
11 scans; correct?

12 A. Correct.

13 Q. How did you determine Mr. Kidd did
14 not have an unusually high amount of
15 radiation exposure in relation to the
16 general public?

17 A. Well, and one of the things I also
18 do is I review radiation epidemiology and
19 the radiation epidemiology studies that are
20 done looking at radiation as a risk factor,
21 and there has been some really good
22 low-level radiation studies. The typical
23 dose response in a general population is
24 looking at categories of radiation exposure
25 that the lowest exposure level is very

1 typical of the kinds of radiation experience
2 that he had.

3 And the higher levels that are
4 associated with, you know, these elevated
5 risks are typically folks who have had CAT
6 scans, or who have had radiation therapy, or
7 who have had other sources of radiation
8 exposure that are much more high dose than
9 what he experienced.

10 Q. So is it fair to say that you
11 compared Mr. Kidd's radiation doses to
12 radiation doses in epidemiology studies?

13 A. In what I have seen in epi studies
14 of low-level radiation and cancer, yes.

15 Q. Genetic susceptibility loci have
16 also been associated with an increased risk
17 for diffuse large B-cell lymphoma; correct?

18 A. Yes.

19 Q. Did you consider genetics as a risk
20 factor for Mr. Kidd's diffuse large B-cell
21 lymphoma?

22 A. Well, we have no information on
23 that. The genetic loci that you mention
24 come from GWAS studies, so-called
25 genome-wide association studies. And, you

1 know, they have not identified a particular
2 gene that he was either tested for that is
3 commonly tested for that would allow
4 clinicians to understand whether a
5 particular genetic mutation was, in fact, a
6 major factor in his condition.

7 So I considered it, but there was
8 no evidence of that in his case.

9 Q. Is there any evidence that genetics
10 was not involved in the causation of Mr.
11 Kidd's non-Hodgkin's lymphoma?

12 ATTORNEY GREENWALD: Objection.
13 Form.

14 THE WITNESS: No.
15 BY ATTORNEY ANTONUCCI:

16 Q. In Mr. Kidd's initial report, and,
17 again, that's Exhibit 3, page 6, just above
18 subheading 2.

19 A. I think I just mixed up my own
20 file. One second.

21 Q. I think you might have it backwards
22 if you flip it over.

23 A. Yeah. Oh, there it is. Thank you.
24 I just tucked it underneath by mistake.
25 Sorry. Yeah, here we go.

1 What page again?

2 Q. Page 6, Exhibit 3.

3 A. Thank you.

4 Q. For Other environmental history of
5 potential relevance you wrote: Mr. Kidd
6 related that he was occasionally exposed to
7 paint thinners and ammonia bleach. He also
8 occasionally used insecticides to get rid of
9 flies. He also used Roundup, about ten
10 times total.

11 Did I read that correctly?

12 A. Yes.

13 Q. Is exposure to paint thinner a risk
14 factor for NHL?

15 A. Not that I recall.

16 Q. Why did you list it under
17 environmental history of potential
18 relevance?

19 A. Well, when I take a history from a
20 patient, you know, I certainly pay attention
21 to the historical attributes that are
22 relevant to the disease, but I also tend to
23 take a general history just to see if there
24 are other general exposures or general
25 considerations that should be recorded. I

1 try to be thorough.

2 Q. Is exposure to ammonia bleach a
3 risk factor for NHL?

4 A. Not that I recall.

5 Q. Is exposure to insecticide a risk
6 factor for NHL?

7 A. I believe that -- and I have to
8 recheck on this -- that exposure to
9 herbicides, particularly among, for
10 instance, the veterans who are exposed to
11 Agent Orange, is a risk factor for NHL in
12 some epidemiology studies, but that's a
13 recollection I have. But he certainly had
14 no exposures like that.

15 Q. And by "exposures like that," you
16 mean that Mr. Carter was not exposed to
17 Agent Orange?

18 A. Correct.

19 Q. Is exposure to Roundup a risk
20 factor for NHL?

21 A. I haven't looked at the latest
22 epidemiology on that. I haven't looked at
23 the latest epidemiology on that.

24 Q. Do you recall citing Wang 2023
25 Epidemiology and Etiology of Diffuse Large

1 B-Cell Lymphoma in your report for Mr. Kidd?

2 A. Refresh my memory.

3 Q. That is page 9 of your report for
4 Mr. Kidd, footnote 21. That's Exhibit 3.

5 A. Okay.

6 Q. I'm going to hand you Exhibit 22.
7 This is Epidemiology and Etiology of Diffuse
8 Large B-Cell Lymphoma by Sophia S. Wang
9 published in Seminars in Hematology 2023.

10 (Exhibit Number 22 was marked for
11 identification.)

12 BY ATTORNEY ANTONUCCI:

13 Q. Page 262 of Exhibit 22, so on the
14 top of the second column on this page, the
15 second sentence of that paragraph, "In a
16 pooled analysis."

17 Do you see where I am?

18 A. Yes.

19 Q. In a pooled analysis of three large
20 agricultural worker cohorts including 2,430
21 NHL cases across the United States, France,
22 and Norway, a positive association was
23 reported between glyphosate and DLBCL,
24 hazard ratio of 1.48, which was further
25 supported in a meta-analysis of seven

1 studies 1.3 fold DLBCL risk for the highest
2 category of glyphosate exposure.

3 Did I read that correctly?

4 A. You did.

5 Q. Did you consider Mr. Kidd's
6 exposure to Roundup in performing your
7 differential etiology?

8 A. Well, I took note of that, that he
9 had used Roundup. I don't consider his
10 usage of Roundup as being particularly high.
11 I think most adults of his age, just from
12 common sense, have used Roundup to get rid
13 of weeds, unfortunately. I did.

14 So I didn't consider that as a, you
15 know, major high-risk factor if he used it
16 ten times in his life, which is what he
17 reported on history.

18 Q. Okay. So because Mr. Kidd did not
19 use the Roundup product frequently enough,
20 you did not consider it a risk factor in his
21 NHL; is that correct?

22 A. Well, I didn't -- right. I didn't
23 make note of that as a risk factor. But
24 even if I had and said, well, gee, he's been
25 exposed to some Roundup, it's a very low

1 level. Maybe it played a role. It
2 certainly wouldn't undercut, again, the risk
3 factor that were posed by the TCE, PCE, and
4 benzene, in my view.

5 Q. Is it your opinion that Roundup is
6 a risk factor for NHL?

7 ATTORNEY GREENWALD: Objection to
8 form.

9 THE WITNESS: I don't have an
10 opinion.

11 ATTORNEY ANTONUCCI: I'm going to
12 ask again because there was some cross
13 talk, and you can object again.

14 ATTORNEY GREENWALD: I will.

15 BY ATTORNEY ANTONUCCI:

16 Q. Is it your opinion that Roundup is
17 a risk factor for NHL?

18 ATTORNEY GREENWALD: Objection.
19 Form.

20 THE WITNESS: I haven't considered
21 it formally, and I don't have an
22 opinion.

23 BY ATTORNEY ANTONUCCI:

24 Q. Okay. Please turn to Exhibit 4.
25 That's your specific causation report for

1 Mr. Carter. I'd appreciate it if you could
2 look at page 13.

3 So on page 13 on Exhibit 4,
4 paragraph 6, you wrote: Given that MACL is
5 a relatively rare form of NHL, roughly
6 5 percent, research specific to
7 environmental risk factors for MACL is
8 challenging and few such studies exist.
9 2008 Swedish case-control study of NHL found
10 infection by Borrelia, the agent that causes
11 Lyme disease, was associated with a
12 significantly increased risk of MACL;
13 however, such an association has reportedly
14 not been found in studies of Borrelia
15 infection in the USA, and a review of
16 Mr. Carter's medical records has not
17 uncovered evidence of Borrelia infection.

18 Did I read that correctly?

19 A. Yes.

20 Q. You go on to discuss NHL risk
21 factors, which we previously discussed in
22 the context of Mr. Kidd; correct?

23 A. Yes.

24 Q. So the risk factors that you
25 considered for Mr. Carter were his race and

1 low-level and low-duration radiation
2 exposure; correct?

3 ATTORNEY GREENWALD: Objection to
4 form.

5 THE WITNESS: Yes, that's what I
6 wrote later in that paragraph.

7 BY ATTORNEY ANTONUCCI:

8 Q. In addition to his exposure to
9 contamination at Camp Lejeune; correct?

10 A. Correct.

11 Q. And you ruled out Borrelia -- or
12 did not afford weight to Borrelia infection
13 because there's no evidence of it; is that
14 accurate?

15 A. That is accurate.

16 Q. Did you consider family history of
17 a first-degree relative with non-Hodgkin's
18 lymphoma in your differential etiology for
19 Mr. Carter?

20 A. Well, as I wrote on page 6 of that
21 report, I took a family history, and there
22 was no evidence of someone who had lymphoma,
23 although there was someone who died at the
24 age of 76 of what the family described as
25 some type of blood cancer, but without

1 having any other specifics, it's hard to
2 know what that really was.

3 Q. And so you also wrote on page 13 of
4 this report that: Having a family history
5 of a first-degree relative with NHL is a
6 risk factor for which there is evidence of
7 an association with non-Hodgkin's lymphoma;
8 correct?

9 A. Yes.

10 Q. Is there evidence of an association
11 between having a family member with any kind
12 of cancer and non-Hodgkin's lymphoma?

13 A. Not that I'm aware of.

14 Q. Is there evidence of an association
15 between having a family member with blood
16 cancers and non-Hodgkin's lymphoma?

17 A. Assuming that these are hematologic
18 cancers versus lymphoma, I'm not aware of
19 it.

20 Q. So on page 13 -- excuse me,
21 page 14, you cited in footnote 33 the study
22 by Smedby, et al., Epidemiology and Etiology
23 of Mantle Cell Lymphoma; is that correct?

24 A. Yes.

25 ATTORNEY ANTONUCCI: I'm going to

1 fmark that for identification. So
2 this is Exhibit 23, Smedby, et al.

3 (Exhibit Number 23 was marked for
4 identification.)

5 BY ATTORNEY ANTONUCCI:

6 Q. I'd like you to look at page 296
7 under Subsection 5, Family History and
8 Genetic Susceptibility, Smedby, et al.,
9 state: Family history of hematopoietic
10 malignancies has been linked to a twofold
11 increase of MCL -- excuse me.

12 It says: Family history of
13 hematopoietic.

14 Did I read that correctly?

15 A. You did.

16 Q. And hematopoietic malignancies
17 means blood cancers; right?

18 A. Presumably.

19 Q. Did you consider the fact that
20 Mr. Carter's brother also died of a
21 hematopoietic malignancy in your
22 differential etiology for Mr. Carter?

23 ATTORNEY GREENWALD: Objection.

24 Form.

25 THE WITNESS: Without any

1 confirmatory evidence, death
2 certificate, or anything else, it was
3 hard to know what to make of that.

4 BY ATTORNEY ANTONUCCI:

5 Q. Do you normally request a death
6 certificate from patients when you take a
7 family history from them?

8 ATTORNEY GREENWALD: Objection.
9 Form.

10 THE WITNESS: Sometimes. But in
11 this case, it's somewhat vague.

12 BY ATTORNEY ANTONUCCI:

13 Q. If Mr. Carter's brother had died of
14 a hematopoietic malignancy, that would mean
15 Mr. Carter had a first-degree relative with
16 a hematopoietic malignancy; correct?

17 A. Correct.

18 Q. Did you consider the death of
19 Mr. Carter's first-degree relative from a
20 blood cancer as a risk factor?

21 ATTORNEY GREENWALD: Objection.
22 Form.

23 THE WITNESS: It may have been,
24 but if anything, that would have meant
25 he had genetic predisposition that

1 could have interacted with the
2 chemical exposure to increase his risk
3 even more.

4 So let me just come back again to
5 my conclusion that none of the risk
6 factors that we talked about undercut
7 the conclusion that TCE, PCE, and
8 benzene, given all the evidence that
9 we just discussed, was also a
10 substantial contributing factor.

11 BY ATTORNEY ANTONUCCI:

12 Q. It's true that the causes of mantle
13 cell lymphoma are not known; correct?

14 ATTORNEY GREENWALD: Objection.
15 Form.

16 THE WITNESS: I mean, again, it
17 depends on what you mean by causes,
18 but there are risk factors that have
19 been identified, like in this
20 particular review, 14 years ago.

21 BY ATTORNEY ANTONUCCI:

22 Q. Are you referring to the Smedby
23 case you cited?

24 A. Yes.

25 Q. If you look at page 295 of the

1 Smedby case, on the first paragraph, the
2 sentence beginning "MCL."

3 Do you see that?

4 A. I'm sorry, where on page 295?

5 Q. Yes. The paragraph above
6 Subsection 4, about halfway through the
7 paragraph there's a sentence beginning with
8 the abbreviation MCL?

9 A. Oh, yes. I see it.

10 Q. That sentence says: MCL belongs to
11 the group of NHL subtypes for which the
12 etiology is yet to be explained.

13 Did I read that correctly?

14 A. You did.

15 Q. How did you account for the fact
16 that the etiology of MCL has yet to be
17 explained in determining the cause of Mr.
18 Carter's MCL?

19 ATTORNEY GREENWALD: Objection to
20 form.

21 THE WITNESS: This is a general
22 statement, and I don't consider that
23 as necessarily relevant to looking at
24 some of the known risk factors and
25 looking at -- opining on a

1 relationship between the known risk
2 factor and the causation -- the
3 occurrence of cancer in a particular
4 individual. I just don't see how
5 that's relevant to the exercise we're
6 doing today.

7 BY ATTORNEY ANTONUCCI:

8 Q. So on page 7 of your report for
9 Mr. Carter, and that's Exhibit 4.

10 A. What page?

11 Q. I might have the wrong page. On
12 page 7, excuse me, of Exhibit 4.

13 A. Okay.

14 Q. The last sentence of the first
15 paragraph says: In this job, he,
16 Mr. Carter, was known to have had some
17 occupational exposure to asbestos and to
18 have conducted lead paint testing; is that
19 right?

20 A. Yes.

21 Q. Is exposure to asbestos a risk
22 factor for non-Hodgkin's lymphoma?

23 A. I know that's been looked at, and
24 if it is, the evidence is pretty -- what I
25 would say is weak. I don't recall that.

1 Certainly isn't one of the major cancers
2 that has been associated with asbestos.

3 Q. You also noted there that
4 Mr. Carter was known to have conducted lead
5 paint testing.

6 Is exposure to lead a risk factor
7 for non-Hodgkin's lymphoma?

8 A. Not that I know of.

9 Q. And you also noted on page 7 that
10 Mr. Carter maintained a large vegetable
11 garden and used pesticides containing
12 Bifenthrin.

13 Is exposure to Bifenthrin a risk
14 factor for NHL?

15 A. Specifically, not that I'm aware
16 of.

17 Q. You noted on page 14 of Exhibit 4
18 that Mr. Carter smoked cigarettes; correct?

19 A. Yes.

20 Q. How much did Mr. Carter smoke?

21 A. A pack a day between 1974 to 1992
22 when he quit.

23 Q. Do you know how old he was when he
24 started smoking?

25 A. 26.

1 Q. Did Mr. Carter ever quit smoking
2 for any period of time?

3 A. Well, it says here in my history
4 that he quit in 1992 at age 44.

5 Q. Do you know whether Mr. Carter quit
6 at any period before 1992?

7 A. It's possible, but I certainly
8 didn't record it in my report.

9 Q. You noted in your report that
10 epidemiological studies of both mantle cell
11 lymphoma and non-Hodgkin's lymphoma have not
12 identified cigarette smoking as a risk
13 factor for either cancer; correct?

14 A. That is correct.

15 Q. And in support of that, you cited
16 to Smedby, et al.

17 That's Exhibit 23; correct?

18 A. Yes.

19 Q. Okay. Please look at page 296 of
20 Exhibit 23.

21 A. Wait a second.

22 Q. If you'll look at the second
23 column, the second sentence starting with
24 the words "Body mass index."

25 Do you see that?

1 A. Yes.

2 Q. So here Smedby, et al., wrote:
3 Body mass index, cigarette smoking, and
4 alcohol intake were not implicated as risk
5 factors for MCL, but the statistical power
6 to detect any association was limited due to
7 the relatively small sample size of MCL, in
8 spite of the fact that several studies were
9 pooled for these analyses.

10 Did I read that correctly?

11 A. You did.

12 Q. Does that change your conclusion
13 that cigarette smoke is not a risk factor
14 for MACL?

15 ATTORNEY GREENWALD: Objection.

16 Form.

17 THE WITNESS: No.

18 BY ATTORNEY ANTONUCCI:

19 Q. Okay. So I'd like you to turn to
20 Exhibit 5. That's your rebuttal report to
21 Dr. Ambinder for plaintiff Robert Kidd.

22 A. Okay.

23 Q. And on page 1 of this report and
24 also for the page 1 of the rebuttal report
25 to Dr. Ambinder for Ronald Carter, you

1 wrote, quote: My rebuttal report does not
2 contain a response to all of the points that
3 Dr. Ambinder makes in his report with which
4 I disagree, nor should my rebuttal to some
5 portions of his report and not others be
6 viewed as agreement with the portions of his
7 report that I do not rebut below.

8 A. Right.

9 Q. Did I read that correctly?

10 A. You did.

11 Q. And, Dr. Hu, you're aware that
12 you're required to disclose a complete
13 statement of all the opinions you will
14 express in this case and the basis and
15 reasons for them in your reports; correct?

16 ATTORNEY GREENWALD: Objection.
17 Form.

18 THE WITNESS: Yes.

19 BY ATTORNEY ANTONUCCI:

20 Q. Okay. On page 1 of this same
21 exhibit, this is, again, Exhibit 5, you
22 quoted Dr. Ambinder where he states he
23 relied on the expert report prepared by
24 Dr. Goodman.

25 Do you see that?

1 A. Yes.

2 Q. And your comment and response was:
3 I refer to my own general causation report
4 on benzene, TCE, and PCE.

5 Did I read that correctly?

6 A. You did.

7 Q. Have you read Dr. Goodman's report
8 titled Trichloroethylene, Perchloroethylene,
9 Benzene, Vinyl Chloride and Trans-1,2-DCE
10 Exposure and NHL risk, dated February 27,
11 2025?

12 A. Honestly, I don't recall. If you
13 have it, I'll take a look at it.

14 Q. I'll note that this is not on any
15 of your materials considered lists.

16 A. Then I probably did not.

17 Q. Do you know if Dr. Goodman reviewed
18 any studies that you did not?

19 A. In her report?

20 Q. Do you know if Dr. Goodman reviewed
21 any studies in her report that you did not?

22 A. I wouldn't know.

23 Q. Do you know if you reviewed any
24 studies in your reports that Dr. Goodman did
25 not review in hers?

1 A. I don't know.

2 Q. Do you know what methodology Dr.
3 Goodman employed in reaching her
4 conclusions?

5 A. Having not read the report, I do
6 not know.

7 Q. Okay. Is it fair to say that you
8 did not consider Dr. Goodman's report at all
9 in reaching your conclusions on the specific
10 causation of Mr. Kidd and Mr. Carter's
11 diseases?

12 A. Correct.

13 Q. So page 2 of Exhibit 5 here,
14 paragraph 3, you note that Dr. Ambinder
15 quoted Wang 2023 for a list of established
16 risk factors for DLBCL; correct?

17 A. Correct. But I just want to
18 elaborate a little more on the last
19 question, which is, I think, in one of my
20 reports, I discussed Goodman's so-called
21 systemic review of a subject that's relevant
22 to general causation.

23 So I did not see her report in this
24 litigation, but I have seen and critiqued
25 her report on a general causation topic

1 that's relevant to this litigation, a Polish
2 report.

3 Okay. So what's your question this
4 time around?

5 Q. So page 2 of Exhibit 5, paragraph
6 3, you note that Dr. Ambinder quoted Wang
7 2023 for a list of established risk factors
8 for DLBCL; correct?

9 A. Yes.

10 Q. And you noted that the abstract of
11 that study states, quote: There is strong
12 evidence for multiple environmental
13 exposures in DLBCL etiology, including
14 exposure to trichloroethylene, benzene, and
15 pesticides and herbicides, with recent
16 associations noted with glyphosate; correct?

17 A. Yes.

18 Q. Okay. I would like you to please
19 turn to Exhibit 22. That's the Wang 2023
20 Epidemiology and Etiology of DLBCL study.

21 A. This is not the same study as I
22 quoted in the text you just read. The text
23 you just read, I quoted Wang study of
24 Epidemiology and Etiology of Diffuse Large
25 B-Cell Lymphoma.

1 Exhibit 23 is epidemiology and
2 etiology of mantle cell lymphoma and other
3 NHLs.

4 Q. I'm sorry. I meant to say
5 Exhibit 22.

6 A. Oh, okay.

7 Q. Thanks for the correction.

8 A. Yeah.

9 Q. I appreciate that.

10 A. Okay.

11 Q. So if you could please look at
12 Exhibit 22, page 261, at the last
13 paragraph under the heading Environmental
14 and Occupational Exposures.

15 Do you see where I am?

16 A. Yes.

17 Q. This reads: Although there is
18 relatively robust evidence linking
19 occupations and environmental exposures to
20 increased risk of hematopoietic
21 malignancies, the evidence that links these
22 exposures specifically to DLBCL remains
23 limited due to the nature of the studies.
24 Much of the epidemiologic data is derived
25 from occupational cohorts that follow

1 high-exposed populations for years but
2 accrue relatively small numbers of NHLs, of
3 which evaluating NHL subtypes is
4 challenging. Nested case-control studies
5 are also used to directly measure
6 environmental exposures in biospecimens, but
7 due to the expense in cost and biospecimen
8 of measuring exposures, these studies are
9 also typically limited in sample size.

10 Did I read that correctly?

11 A. You did.

12 Q. Wang goes on to state on page 262:
13 Trichloroethylene (TCE) is classified as a
14 probable carcinogen (group 2A) by IARC. A
15 meta-analysis of 14 occupational cohort and
16 four case-control studies linked TCE to
17 elevated NHL risk but lacked NHL subtype
18 data; however, a pooled analysis of 3,788
19 NHL cases and 4,279 controls within four
20 participating InterLymph Consortium studies
21 evaluated the association between TCE based
22 on occupation categories and NHL risk and
23 found DLBCL risk to be elevated in the
24 highest category of exposure intensity;
25 correct?

1 A. Correct.

2 Q. How does that highest category of
3 occupational exposure intensity compare to
4 Mr. Kidd's exposures at Camp Lejeune?

5 A. Well, let's pull out the study, and
6 let's talk about it.

7 Q. Are you aware of what the level is
8 sitting here today?

9 A. Well, I read the study, and I think
10 I quoted it in my general causation report;
11 so I'm aware of it. But I -- you know,
12 sitting here, I can't specifically recall
13 exactly how they defined highest category
14 exposure intensity.

15 Q. Did you remember at the time you
16 wrote your specific causation report?

17 A. I mean, I was looking at the study;
18 so sure. You know, this is another example
19 of where, you know, you need to look at
20 these high dose studies in order to address
21 the issue of general causation because if
22 you did a power calculation of the number of
23 people you need to study in order to get a
24 statistically significant increased risk in
25 a study that's properly designed, that would

1 be -- typically involve many more
2 individuals than is typically possible in a
3 epidemiology study.

4 So we're often relying on
5 occupational studies to give us a sense of
6 what the general risk is whether it's cancer
7 or other chronic disease outcomes. We used
8 those studies to understand general
9 causation and oftentimes project what the
10 risks are at lower levels of exposure.

11 THE VIDEOGRAPHER: Can we go off?

12 We are now going off the record.

13 This is the end of media Unit

14 Number 5, and the time is 4:23 p.m.

15 (Recess taken from 4:23 p.m. to
16 4:34 p.m.)

17 THE VIDEOGRAPHER: We are now back
18 on the record. This is the beginning
19 of media Unit Number 6, and the time
20 is 4:34 p.m.

21 BY ATTORNEY ANTONUCCI:

22 Q. Dr. Hu, we're back on the record.

23 You understand you're still under
24 oath; correct?

25 A. Yes.

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

4 A. No.

5 Q. Okay. It's your opinion that
6 Mr. Kidd's exposure to TCE, PCE, and benzene
7 from Camp Lejeune increased the risk of and
8 constituted the cause or a substantial
9 contributing cause to his non-Hodgkin's
10 lymphoma; correct?

11 A. Yes.

12 Q. Did Mr. Kidd's exposure to
13 contaminated water at Camp Lejeune increase
14 the risk of his NHL, or did it cause his
15 NHL?

16 A. I would simply say it increased the
17 risk.

18 Q. Okay. So increasing the risk of
19 something and causing it are two different
20 things; correct?

21 A. Well, the fact that he got NHL, in
22 my view, ends up being a reason for
23 concluding that the exposures at Camp
24 Lejeune were a substantial contributing
25 factor that caused his NHL.

1 Q. Increasing the likelihood of
2 disease is different from actually causing
3 such a disease; correct?

4 A. Of course.

5 Q. And it's your opinion that
6 Mr. Kidd's exposure to contaminated water at
7 Camp Lejeune did both; is that right?

8 A. Yes.

9 Q. How much does the risk of NHL have
10 to be increased by exposure to contamination
11 to say that the exposure was a substantial
12 contributing cause to the NHL?

13 ATTORNEY GREENWALD: Objection.
14 Form, asked and answered.

15 THE WITNESS: I mean, I think that
16 that's a specific causation question,
17 and that always requires a in-depth
18 analysis of the specific circumstances
19 of the individual, the cancer, the
20 exposure, timing, et cetera, other
21 potential causes which, as I've said
22 many times in this deposition, is a
23 process that has to be undertaken
24 rigorously and relatively thoroughly,
25 and that's what I believe I performed.

1 BY ATTORNEY ANTONUCCI:

2 Q. In your rebuttal to Dr. Ambinder
3 for Mr. Kidd, that is Exhibit 5, you wrote
4 on pages 2 and 3 that Dr. Ambinder ignores
5 the fact that cancer causation is a
6 multi-step and multi-factorial phenomenon;
7 correct?

8 A. Yes. On page 3, I wrote that:
9 Ignores the fact that as research on the
10 process of carcinogenesis continues to make
11 progress, it has become clear that cancer
12 causation is a multi-step and
13 multi-factorial phenomenon that is heavily
14 influenced by extrinsic (i.e.,
15 environmental) factors.

16 That's my opinion, and it remains
17 so today.

18 Q. And you wrote that Dr. Ambinder's
19 critique of your discussion of differential
20 etiology essentially implies that position;
21 correct?

22 ATTORNEY GREENWALD: Objection.
23 Form.

24 BY ATTORNEY ANTONUCCI:

25 Q. If you look at page 3 under the

1 subheading Comment, second paragraph: The
2 second comment by Dr. Ambinder essentially
3 implies two positions.

4 A. Yeah, no, I understand that. So
5 I'm sorry. Can you repeat your question?

6 Q. I'd like you to look at Exhibit 9,
7 please. That is your rebuttal report to
8 Dr. Ambinder for Mr. Carter.

9 A. Okay.

10 Q. On page 2 of Exhibit 9 in the -- at
11 the very top of the page, you write: I
12 would agree with Dr. Ambinder that
13 Mr. Carter likely had a higher risk of
14 stroke given his family history of
15 myocardial infarctions and strokes; correct?

16 A. Yes.

17 Q. Do you know what percentage of
18 non-Hodgkin's lymphoma patients die from
19 strokes?

20 A. Not as I sit here today.

21 Q. Do you agree that the vast majority
22 of non-Hodgkin's lymphoma patients do not
23 die from strokes?

24 ATTORNEY GREENWALD: Objection.

25 Form.

1 THE WITNESS: That sounds
2 plausible, but without actually
3 reviewing the evidence, I couldn't say
4 for sure.

5 BY ATTORNEY ANTONUCCI:

6 Q. You noted in your report for
7 Mr. Carter that he had a history of coronary
8 artery disease, angina, a myocardial
9 infarction in 2013, and glucose intolerance;
10 correct?

11 A. I'm sorry. Where are you?

12 Q. That's going to be in Exhibit 4 on
13 page 6. Excuse me. That's specifically on
14 pages 4 and 5.

15 A. Okay. Can you repeat that
16 question? Sorry.

17 Q. You're aware that Mr. Carter had a
18 history of coronary artery disease, angina,
19 myocardial infarction in 2013, and glucose
20 intolerance; right?

21 A. Yes, I am.

22 Q. And you're aware that Mr. Carter's
23 father died at the age of 69 of a heart
24 attack; correct?

25 A. That's what was reported by the

1 family.

2 Q. Were you aware the family also
3 reported Mr. Carter's son had two heart
4 attacks and a stroke?

5 A. I don't recall that and did not
6 record that in my report; so no.

7 Q. Okay. And it's your opinion that
8 having non-Hodgkin's lymphoma increased
9 Mr. Carter's risk of stroke; correct?

10 A. Yes.

11 Q. And that having non-Hodgkin's
12 lymphoma was a substantial contributing
13 factor to Mr. Carter's stroke?

14 A. I actually did not render a formal
15 opinion on that in either my specific
16 causation expert report or the rebuttal to
17 Dr. Ambinder. I was simply responding to
18 his critique of my report that was -- you
19 know, essentially tried to take issue with
20 my history.

21 Q. Okay. So it is not an opinion that
22 you're offering in this litigation that
23 having non-Hodgkin's lymphoma was a
24 substantial contributing factor to
25 Mr. Carter's stroke?

1 ATTORNEY GREENWALD: Objection.

2 Form.

3 THE WITNESS: Well, his exposures
4 clearly were a substantial
5 contributing factor to his cancer, and
6 the immediate cause of death on his
7 death certificate was a stroke, and I
8 have discussed how non-Hodgkin's
9 lymphoma is known to be associated
10 with an increased risk of stroke.

11 So it would be logical to conclude
12 that if that's true, that the
13 exposures at Camp Lejeune indirectly
14 were a cause of his death through the
15 causation of his lymphoma. I didn't
16 explicitly say that in my report, but
17 one could draw that conclusion based
18 on what I've written in my rebuttal to
19 Dr. Ambinder.

20 BY ATTORNEY ANTONUCCI:

21 Q. Okay. So yes or no: Are you
22 offering the opinion that Mr. Carter's NHL
23 was a substantial contributing factor to his
24 stroke?

25 ATTORNEY GREENWALD: Objection.

1 Form, asked and answered.

2 THE WITNESS: You know, I'd have
3 to look at my primary sources and
4 review the literature again before I
5 furnish a formal opinion on that.

6 BY ATTORNEY ANTONUCCI:

7 Q. Okay. I'm going to reserve the
8 right to hold the deposition open to
9 question you on the causation of
10 Mr. Carter's stroke based on your review of
11 the literature.

12 I'd like to move on to your
13 rebuttals to Dr. Bailey. That's Exhibits 10
14 and 11. On page 1 of both Exhibits 10 and
15 11, I'll give you a moment to find those.

16 A. Okay. I've got it. 10 and 11.
17 Okay.

18 Q. Dr. Hu, you agree that the linear
19 no-threshold mechanism of action is applied
20 in regulatory cancer risk evaluations where
21 there is no known threshold dose below which
22 exposure to a carcinogen is not expected to
23 lead to some level of risk even if it is
24 very low; correct?

25 A. Yes.

1 Q. And you agree that a threshold
2 model for deriving toxicity criteria is
3 based on the concept that there is some dose
4 below which no adverse effects are expected;
5 right?

6 A. For some toxicity relationships,
7 that's true.

8 Q. And the linear no-threshold model
9 likely overestimates exposures at very low
10 doses; correct?

11 A. What do you mean "overestimates
12 exposures"?

13 Q. Do you believe it's biologically
14 possible that exposure to one molecule of
15 TCE can increase an individual's risk for
16 developing NHL?

17 ATTORNEY GREENWALD: Objection.
18 Form.

19 THE WITNESS: It's theoretically
20 impossible -- it's theoretically
21 possible but, obviously, unlikely
22 given the multi-hit hypothesis, the
23 multi-cause hypothesis, and the
24 existence of defense mechanisms in the
25 body.

1 BY ATTORNEY ANTONUCCI:

2 Q. Do you believe it's biologically
3 plausible that exposure to one molecule of
4 PCE can increase an individual's risk for
5 developing NHL?

6 ATTORNEY GREENWALD: Objection.
7 Form.

8 THE WITNESS: I'm going to go back
9 to my general causation report for a
10 moment. Excuse me.

11 Okay. Can you repeat the
12 question?

13 BY ATTORNEY ANTONUCCI:

14 Q. Do you believe it's biologically
15 plausible that exposure to one molecule of
16 PCE can increase an individual's risk for
17 developing NHL?

18 A. It's theoretically possible but,
19 obviously, unlikely for the reasons I
20 mentioned for the other scenario you posed.

21 Q. Do you believe it is biologically
22 plausible that exposure to one molecule of
23 benzene can increase an individual's risk
24 for developing NHL?

25 ATTORNEY GREENWALD: Objection.

1 Form.

2 THE WITNESS: Same response.

3 BY ATTORNEY ANTONUCCI:

4 Q. So, in general, you agree that the
5 human body is, at least in some
6 circumstances, able to repair DNA damage;
7 correct?

8 A. Yes.

9 Q. And you agree that the body's
10 normal defense mechanisms can eliminate low
11 concentrations of a mutagenic or
12 carcinogenic substance and repair DNA damage
13 that exposure to that substance may have
14 caused; right?

15 A. In certainly the majority of cases
16 but not necessarily all.

17 Q. Is DNA damage a cause of
18 non-Hodgkin's lymphoma?

19 ATTORNEY GREENWALD: Objection.

20 Form.

21 THE WITNESS: I believe it is part
22 of the intrinsic mechanism by which
23 NHL is caused in many cases.

24 BY ATTORNEY ANTONUCCI:

25 Q. And, Dr. Hu, you conclude that PCE

1 causes -- exposure to PCE causes NHL through
2 genotoxicity based on evidence of PCE's
3 genotoxicity and peripheral blood
4 lymphocytes; right?

5 A. I think that's one of the
6 mechanisms that may be at play, yes.

7 Q. Peripheral blood lymphocytes are
8 not involved in the development of NHL;
9 correct?

10 A. Probably not. But they are, I
11 guess you could say, a reflection of what
12 may be happening in the bone marrow or in
13 lymph nodes.

14 Q. You also conclude that PCE causes
15 NHL through genotoxicity based on evidence
16 of PCE's genotoxicity in leukocytes; right?

17 A. Right.

18 Q. Leukocytes are not involved in the
19 development of NHL; correct?

20 A. No. Those are from the adaptive
21 immune response, but they are also a
22 reflection of what might be happening in
23 cells of that origin.

24 Q. So what is your -- what is your
25 methodology for concluding that -- that PCE

1 causes NHL based on evidence of genotoxicity
2 in cells unrelated to the development of NHL
3 like peripheral lymphocytes and leukocytes?

4 A. To the extent that it's a
5 reflection of what's happening in the bone
6 marrow itself and in lymph nodes and other
7 lymph-containing organs.

8 Q. Is there any evidence that PCE
9 causes genotoxicity on the cells related to
10 the development of NHL?

11 ATTORNEY GREENWALD: Objection.
12 Form.

13 THE WITNESS: There may be some in
14 vitro studies that have been done, but
15 I'd have to go back and look at the
16 scientific basis -- scientific
17 evidence basis to answer that question
18 accurately.

19 BY ATTORNEY ANTONUCCI:

20 Q. Dr. Hu, you're aware that there's a
21 debate in the scientific community regarding
22 the linear no-threshold model; correct?

23 A. Yes.

24 Q. Is it your belief that there is a
25 consensus in the scientific community around

1 the linear no-threshold model?

2 A. Well, it depends how you define
3 consensus, but I think a majority of, I
4 guess I would say, mainstream environmental
5 epidemiologists and my colleagues would
6 consider the linear no-threshold model as
7 still operative.

8 Q. All right. On Exhibit 10, that's
9 your rebuttal to Dr. Bailey for Mr. Kidd.
10 At the bottom of page 3, last
11 paragraph starting with the word "Finally."

12 Are you with me?

13 A. Yes.

14 Q. You write: Finally, despite these
15 limitations, I point out there have been
16 several key epidemiological studies that
17 have leveraged extraordinarily large sample
18 sizes to provide evidence of the cancer risk
19 at very low levels of exposure to chemical
20 carcinogens; correct?

21 A. Yes.

22 Q. And going on to page 4, you cite to
23 Wang 2024, Long-Term Exposure to Low
24 Concentrations of Ambient Benzene and
25 Mortality in a National English Cohort;

1 correct?

2 A. Correct.

3 Q. And to Yu 2025, Long-Term Exposure
4 to Low-Level Ambient BTEX and Site-Specific
5 Cancer Risk: A National Cohort Study in the
6 UK Biobank as examples of such studies;
7 correct?

8 A. Yes.

9 Q. Are there any other studies you're
10 aware of that have leveraged large sample
11 sizes to provide evidence of the risk of
12 cancer at very low levels of exposure to
13 chemical carcinogens that you're aware of?

14 ATTORNEY GREENWALD: Objection.
15 Form.

16 THE WITNESS: Not this large, no.
17 BY ATTORNEY ANTONUCCI:

18 Q. Okay. On page 4 of your rebuttal
19 to Dr. Bailey for Mr. Kidd -- and, again,
20 that is Exhibit 10 -- you wrote in the
21 second paragraph about halfway through the
22 paragraph starting with "Of note."

23 You wrote: Of note is that,
24 according to EPA toxicokinetic models,
25 inhalation of air contaminated with benzene

1 at a level of 0.18 parts per billion benzene
2 would give rise to the same internal dose
3 (i.e., level of benzene in blood) as
4 ingesting drinking water with benzene at a
5 level of 4.5 parts per billion. As noted in
6 my report on Mr. Kidd, the exposure
7 assessment by Dr. Reynolds resulted in an
8 estimated time-weighted average exposure for
9 Mr. Kidd of 9.6 parts per billion, which is
10 over twice the level of benzene at which
11 point the Yu, et al., study found direct
12 epidemiological evidence of the risk of
13 cancer increasing.

14 Did I read that correctly?

15 A. You did.

16 Q. Dr. Hu, how did you determine that
17 the inhalation of air contaminated with
18 benzene at a level of 0.18 parts per billion
19 would give rise to the same internal dose
20 for a level of benzene in blood as ingesting
21 drinking water with benzene at a level of
22 4.5 parts per billion?

23 A. I consulted an EPA toxicokinetic
24 model exercise.

25 Q. You ran an EPA toxicokinetic model

1 to determine the 4.5 parts per billion value
2 that you cite here on page 4 of Exhibit 10?

3 A. Yes.

4 Q. Which one?

5 A. I didn't cite it here, but I'd have
6 to go back to my computer to figure it out.

7 Q. Do you remember what it's called?

8 A. Oh, God. No.

9 Q. How did you decide which
10 toxicokinetic model to run?

11 A. This is a very simple model. It
12 was a conversion model that went from air
13 modeling to what the likely level would be
14 in a person's blood given some basic
15 parameters of -- I think it was a
16 75-kilogram male breathing this
17 concentration of benzene for a certain
18 amount of time, and there was some other
19 conditions as well. That's what I recall.

20 Q. Did you review EPA's risk summary
21 for the derivation of toxicity values?

22 A. Which publication are you talking
23 about?

24 Q. Did you perform -- or did you
25 review an EPA risk summary that included

1 simple route-to-route extrapolation?

2 ATTORNEY GREENWALD: Objection.

3 Form.

4 THE WITNESS: I think that's

5 exactly what I consulted.

6 BY ATTORNEY ANTONUCCI:

7 Q. Okay.

8 A. Right.

9 Q. So is that actually a toxicokinetic
10 model, or is it --

11 A. Well, it's based on a toxicokinetic
12 model.

13 Q. Okay. And you mentioned that some
14 of the inputs to your model were the
15 75-kilogram male --

16 A. That's not my model. Their model.

17 Q. Sure.

18 Some of the inputs to
19 route-to-route extrapolation were the
20 individual 75-kilogram male; correct?

21 A. That's what I recall, but don't
22 quote me on that. I don't remember the
23 exact conditions that they leveraged.

24 Q. You input the amount of benzene
25 inhaled in the air, the concentration of

1 benzene inhaled; correct?

2 A. That's correct.

3 Q. Did you input the amount of time
4 which the benzene is inhaled over?

5 A. I don't think the model -- at least
6 the model that I consulted asked for that.
7 This is a steady-stated model that they had
8 where you could simply put in the air and
9 get out what the blood was. They have
10 assumptions on how long the inhalation was
11 for.

12 Q. I see.

13 A. It's not like that was a variable
14 that you had to put into the model.

15 Q. Do you know what that assumption
16 was for how long the inhalation was?

17 A. It's like all the things we're
18 talking about here. I don't specifically
19 recall what those assumptions are.

20 Q. And you mentioned there were other
21 conditions which bear on the outputs of the
22 model; correct?

23 A. It's a vague memory. I don't
24 remember exactly what their assumptions
25 were. I'm sorry.

1 Q. And you don't remember the other
2 conditions that --

3 A. No.

4 Q. Okay. Did you check any literature
5 sources to decide which model to select?

6 A. Well, since it was the EPA, and
7 this is their job, I relied on the EPA for
8 that derivation.

9 Q. Okay. And did you document the
10 process of running the model anywhere?

11 A. I may have it on my computer, and
12 I'm sorry I didn't put a footnote in here
13 like I usually do for that.

14 Q. If you did have that file on your
15 computer, would you be able to produce it to
16 counsel?

17 A. I can certainly do my best.

18 ATTORNEY ANTONUCCI: We're going
19 to request, after this deposition in a
20 formal request, that we receive those
21 files.

22 THE WITNESS: That's fine.

23 ATTORNEY GREENWALD: We'll get a
24 letter. Don't worry. If you want to
25 make your own note, go ahead.

1 THE WITNESS: I'm going to make my
2 own note and stick it in my pocket.

3 BY ATTORNEY ANTONUCCI:

4 Q. Did the toxicokinetic model that
5 you consulted provide you with a confidence
6 interval for the 4.5 parts per billion value
7 generated?

8 A. I don't think so. It just churned
9 out a number.

10 Q. Did it provide you with a P value
11 for the 4.5 parts per billion value it
12 generated?

13 A. That's a research metric, not a
14 model metric; so no, it did not.

15 Q. What's your level of confidence in
16 the results of this toxicokinetic model you
17 ran?

18 A. Well, I mean, it's the best
19 estimate, I think, that's available from one
20 of the best potential sources for generating
21 those kinds of estimates; so my degree of
22 confidence is high.

23 Q. Okay. And the inhalation value of
24 0.18 parts per billion benzene was taken
25 from the Wang and Yu studies; correct?

1 A. Yes.

2 Q. And so here you're comparing the
3 inhalation of 0.18 parts per billion benzene
4 in a long-term study with Mr. Kidd's
5 exposure; correct?

6 A. That's correct.

7 Q. Mr. Kidd was on base for about
8 17 months; correct?

9 A. Yes.

10 Q. Do you know how long the exposure
11 duration of the Wang and Yu studies was?

12 A. Wait. Did you say 17 months?

13 Q. Yes.

14 A. I just want to make sure that was
15 right. Okay. Yes, that was correct.

16 So what was your question again?
17 I'm sorry.

18 Q. Dr. Reynolds stated that Mr. Kidd
19 was on base for about 17 months; correct?

20 A. Yes.

21 Q. Okay. And so with this 4.5 parts
22 per billion value that you created, you're
23 comparing the results of the long-term Wang
24 and Yu studies with 17 months of exposure
25 for Mr. Kidd; correct?

1 A. That's correct.

2 Q. You're also comparing a population
3 study with exposure for one individual;
4 correct?

5 A. Yes.

6 Q. Wang in 2024 and Yu in 2025
7 evaluated the same cohort; correct?

8 A. Yes.

9 Q. And that was using the U.K.
10 Biobank?

11 A. Yes.

12 Q. Okay. I'm going to provide you
13 with the Wang study. This is Exhibit 24.

14 (Exhibit Number 24 was marked for
15 identification.)

16 BY ATTORNEY ANTONUCCI:

17 Q. I'd appreciate if you could please
18 turn to page 993. If you look in the center
19 column, it's the third paragraph starting
20 with "However."

21 Do you see where I am?

22 A. Yes.

23 Q. Wang 2024 write: However, the
24 study also has certain limitations that must
25 be acknowledged. First, this observational

1 study could not fully control for all
2 unknown or unmeasured confounding factors
3 and was unable to demonstrate a causal
4 relationship between benzene and mortality.

5 Did I read that correctly?

6 A. You did.

7 Q. Now I'm going to provide you with
8 Yu 2025, and that will be Exhibit 25.

9 (Exhibit Number 25 was marked for
10 identification.)

11 BY ATTORNEY ANTONUCCI:

12 Q. The Yu 2025 study titled Long-Term
13 Exposure to Low-Level Ambient BTEX and
14 Site-Specific Cancer Risk: A National Cohort
15 Study in the UK Biobank focuses on low-level
16 ambient exposure to BTEX; right?

17 A. Yes.

18 Q. Meaning it studied lower exposure
19 doses than an occupational study would;
20 correct?

21 A. I would say that's -- yes.

22 Q. It also evaluates long-term
23 exposures; correct?

24 A. Yes.

25 Q. Do you know how long?

1 A. Well, the study's methodology
2 section notes that their exposure data come
3 from measurements taken from 2006 to 2021;
4 however, in their paper, they do not
5 actually go over the results of that testing
6 for benzene, toluene, and xylene during that
7 time. So it's impossible to know whether
8 the exposures were meaningful in 2006 or
9 whether they were low or high or what the
10 trend was, and I think you'd have to go to
11 some of the other information they've
12 published to answer your question
13 accurately.

14 Q. What other information would you
15 have to look at? Is that the Wang study?

16 A. Well, they said in their first
17 sentence the annual mean levels of benzene,
18 toluene, xylenes from 2006 to 2021 were
19 derived from a three-dimensional
20 chemistry-climate model, and then they cited
21 reference 16 through 18. And it's kind of
22 hard to tell whether those are references
23 that might have gone over the data on
24 exposure.

25 So it just -- I can't answer your

1 question based on the information provided
2 in this paper.

3 Q. Okay. Do you know if the Yu 2025
4 study controls for co-exposures?

5 A. I mean, they have -- because it's a
6 cohort study and it's UK Biobank, they do
7 have data, as they mentioned, in term of
8 covariates, demographic, socioeconomic,
9 lifestyle, health-related information.
10 Passive smoking, smoking, exposure to PM2.5,
11 nitrogen dioxides.

12 So they have quite a bit of
13 additional individual information among the
14 covariates that they have to control for,
15 which they did.

16 Q. Do you know if the Yu 2025 study
17 controls for confounding factors?

18 A. Well, everything that I mentioned
19 could potentially be a confounder.

20 Q. It's true that Yu 2025 could not
21 rule out residual confounding by unmeasured
22 factors that could affect exposure and
23 cancer incidents; correct?

24 ATTORNEY GREENWALD: Objection.

25 Form.

1 THE WITNESS: That is an
2 acknowledgment they made, and that's
3 an acknowledgment every epidemiologist
4 has to make in just about every
5 epidemiology study. It's impossible
6 to control for absolutely everything.

7 BY ATTORNEY ANTONUCCI:

8 Q. Okay. The Yu 2025 study does not
9 present data on NHL subtypes; correct?

10 I would direct you to page 4 for
11 the Figure 1 Associations of Long-Term
12 Exposures to BTEX with Risk of Overall and
13 Site-Specific Cancer.

14 A. Do you have a magnifying glass?
15 Okay. I can make out some of it. Hold on a
16 moment.

17 Yeah, they just list non-Hodgkin's
18 lymphoma but not the subtypes.

19 Q. Okay. And Yu 2025 is an ecological
20 study; correct?

21 A. It's a what?

22 Q. Ecological study; correct?

23 A. No, it's not.

24 Q. Can you explain why Yu 2025 is not
25 an ecological study?

1 A. Because they have individual level
2 data on the subjects. Yes, the exposure
3 data is extrapolated based on the claimant
4 model that they used, but that's a very
5 common technique for doing any air pollution
6 type of epidemiology. But the fact that
7 this is a cohort that's very well
8 characterized and followed over years and
9 they have individual level data on all these
10 other potential confounders, in my book,
11 that is a perspective cohort study.

12 Q. The Yu 2025 study estimates
13 population-based exposure information;
14 correct?

15 A. That's correct.

16 Q. Okay. And that's, again, based on
17 a climate model; right?

18 A. Yes.

19 Q. Population-based exposure
20 information was not measured in the Yu 2025
21 study; correct?

22 A. I think you just contradicted
23 yourself. Say that again.

24 Q. If the information was estimated by
25 the model, it wasn't measured by air

1 sampling; correct?

2 A. Oh, they did plenty of air
3 sampling, but then they used the air
4 sampling using a geospatial model to
5 estimate what the individual level
6 concentrations were. They didn't just pluck
7 these, you know, estimated air pollution
8 measures out of the air and just try to
9 model what they were based on nothing. They
10 actually had measurements.

11 Q. Okay. On page 5, the
12 paragraph above Subsection 5, Conclusion,
13 the sentence starting with "The exposure
14 assessment."

15 This reads: The exposure
16 assessment based on residential addresses
17 could not capture activity patterns of
18 individuals, thus potential exposure
19 misclassification might exist. Moreover,
20 despite the adjustment of a series of
21 confounders, we could not rule out residual
22 confounding by other unmeasured factors that
23 might affect the exposure and cancer
24 incidence. Finally, indoor emissions are an
25 important source of BTEX. The lack of data

1 on individual indoor exposure is a common
2 limitation in environmental epidemiological
3 research, and the results should be
4 interpreted with caution.

5 Did I read that correctly?

6 A. You did.

7 Q. The study authors are saying here
8 that there's potential for exposure
9 misclassification based on their exposure
10 assessment; correct?

11 A. Correct.

12 Q. And Yu used a climate -- Yu 2025
13 used a climate model that predicted
14 atmospheric benzene concentrations in boxes
15 with horizontal dimensions of about
16 100 kilometers by 80 kilometers; right?

17 A. Yes.

18 Q. They then assigned average benzene
19 concentrations to study participants with
20 residential addresses at a
21 1-kilometer-by-1-kilometer resolution;
22 right?

23 A. Yes.

24 Q. The 1-kilometer-by-1-kilometer
25 resolution was superimposed on the

1 10-kilometer-by-80-kilometer boxes that
2 their model created; right?

3 A. Correct.

4 Q. And the study authors compared
5 modeled BTEX concentrations with on-site
6 measurements from European countries, and
7 the comparisons showed a normalized mean
8 bias of plus or minus 25 percent; right?

9 A. Yes.

10 Q. What is normalized mean bias?

11 A. Read that sentence again.

12 Q. So I'm looking at 2.2, Exposure
13 Assessment, on page 2: We compared modeled
14 BTEX concentrations with on-site
15 measurements from European countries, and
16 the comparison showed a normalized mean bias
17 of plus or minus 25 percent.

18 A. Yeah, so they -- okay. So what
19 they did here is that they used their
20 modeling methodology to model BTEX
21 concentrations in European countries where
22 there are actual on-site measurements in the
23 locations where they used the BTEX modeling
24 to model it.

25 Then in order to compare them and

1 not compare apples to oranges, they
2 normalized, in other words, they
3 standardized the measurements between the
4 two geographic locations so that they're in
5 the same metric, and then when they're on
6 the same metric, now they can calculate the
7 coefficient variation, which is essentially
8 this calculation of plus or minus 25 percent
9 which is that -- you know, the precision of
10 their estimates compared to actual
11 measurements was about plus or minus
12 25 percent. That's the meaning of that
13 sentence.

14 Q. And that's an average; correct?

15 A. Yes.

16 Q. And so they tested their bias in
17 European countries rather than in the UK;
18 correct?

19 A. Well, I mean, it's -- you know,
20 it's testing a methodology. It's not so
21 much the dependent on the actual
22 measurements of pollutants in each of these
23 countries. They're testing the measurement
24 methodology approach. Let's put it that
25 way.

1 Q. Isn't the normalized mean bias a
2 measure of its actual ability to predict
3 BTEX values?

4 A. Yeah, it's, like I said, a
5 measurement of the coefficient of variation.
6 It's like you want to take a measurement of
7 something, and the true measurement is 10.
8 Sometimes you get 9 or 8 or 11 or 12. But
9 on average, it comes out to be about a 10;
10 so there's no bias, but there is what is
11 known as noise, and that's also known as
12 non-differential misclassification.

13 Q. So the normalized mean bias has a
14 value of plus or minus 25 percent.

15 What does it mean for the mean bias
16 to be plus or minus 25 percent?

17 A. It means the true value might be a
18 little bit higher, might be a little bit
19 lower.

20 Q. So plus or minus means
21 approximately?

22 A. Yeah.

23 Q. Okay. And I believe you just
24 testified that the Yu study has the
25 potential for differential bias; correct?

1 A. I don't think I --

2 ATTORNEY GREENWALD: Objection.

3 Form.

4 THE WITNESS: I don't think I
5 testified that. Differential bias?

6 BY ATTORNEY ANTONUCCI:

7 Q. I'm sorry. Did you say
8 non-differential bias?

9 A. Non-differential bias.

10 Q. Pardon me.

11 So you testified that the Yu study
12 has the potential for non-differential bias;
13 correct?

14 A. Non-differential misclassification
15 in terms of exposure, yes.

16 Q. Okay. And is that based on
17 classifying exposures based on residences
18 and not occupations?

19 A. I mean, it's based on these using
20 modeling and air pollution measurements to
21 estimate what the exposures were for a
22 population where they -- as the authors
23 themselves noted, they didn't have indoor
24 measurements. They didn't know how much
25 people actually worked over here but resided

1 over there for how many hours of the day.

2 Those are all the kinds of things
3 that happen in exposure measurement
4 limitations that can end up, as I mentioned
5 before, diluting the apparent effect of the
6 exposure you're trying to study.

7 Q. So if the methodology dilutes the
8 apparent effect of the exposure we're
9 studying, that would mean that Yu is
10 probably reporting artificially low
11 associations at high exposures; correct?

12 A. Well, these are all low exposures.
13 None of them -- these are environmental
14 exposures that are residential community,
15 et cetera. So the fact that there's
16 non-differential misclassification, as I
17 mentioned earlier in this deposition,
18 typically means that the effect estimate is
19 an underestimate of the true magnitude of
20 the association between the exposure of
21 interest, in this case benzene, and the
22 outcome of interest, risk of cancer.

23 Q. So conversely accepting as true
24 that benzene causes NHL, people who got NHL
25 because they were exposed to high doses of

1 benzene will probably be shown in Yu as
2 having been exposed to lower doses than they
3 were really exposed to because of the
4 dilution effect; correct?

5 ATTORNEY GREENWALD: Objection.
6 Form.

7 THE WITNESS: Whoa. That made no
8 sense to me. Can you repeat the
9 question.

10 BY ATTORNEY ANTONUCCI:

11 Q. Sure. We're discussing the effect
12 of dilution on the exposure
13 classifications --

14 A. That's an epidemiology concept.
15 That's pertinent to interpreting the results
16 of an epidemiology study, not a, you know,
17 specific causation analysis.

18 Q. Dr. Hu, I know you probably didn't
19 mean to, but please allow me to finish my
20 question.

21 A. Okay. Sorry.

22 Q. So the people who were exposed to
23 high doses of benzene may be modeled as
24 having been exposed to low doses because of
25 dilution; correct?

1 ATTORNEY GREENWALD: Objection.

2 Form.

3 THE WITNESS: Correct. And the
4 other way around. Some people who had
5 low exposures may have actually had
6 higher exposures.

7 BY ATTORNEY ANTONUCCI:

8 Q. That means that the Yu study had
9 potential for differential bias too;
10 correct?

11 A. No. No. What we just discussed is
12 non-differential bias. That is, high may go
13 to low; low may go to high. But there's no
14 reason to suspect that oh, in this study,
15 the ones who were -- had, for instance,
16 higher cancer rates were much more likely to
17 have been estimated to have higher
18 exposures. You know, that -- on a
19 differential basis, that's not true.

20 These are -- whatever
21 misclassification there is, is kind of
22 random because you don't have data on who
23 moved or, you know, whether air pollution is
24 a little bit higher in one place than the
25 other, et cetera, et cetera.

1 So non-differential
2 misclassification is what they're talking
3 about here. I don't think anybody is
4 talking about differential
5 misclassification.

6 Q. The Yu study found a positive
7 association between every cancer and every
8 contaminant that it evaluated; correct?

9 And for that, you can look to
10 page 4, Figure 1.

11 A. Yes. For each of the contaminants
12 and for each of these cancer outcomes,
13 except for a few, the point estimates of
14 risk seem to be above 1, and the 95 percent
15 confidence, the lower bound of the
16 95 percent confidence limit seem to be above
17 1 for the majority of the cancers.

18 Q. Are there any cancers that the Yu
19 study evaluated that did not find a hazard
20 ratio of above 1?

21 A. So they selected a number of cancer
22 sites which they called major cancer sites
23 for this analysis. They didn't look at all
24 cancers.

25 Q. However, for all of the cancers

1 they evaluated, they found an increased
2 hazard ratio; correct?

3 A. That's what I'm trying to figure
4 out, whether this actually is a listing of
5 all the cancers they looked at, or whether
6 it's just the listing of the cancers for
7 which the risks exceeded 1. I'm not sure.
8 I'm just trying to discern that from the
9 paper.

10 Well, they didn't say explicitly
11 so, but my assumption from reading the paper
12 is that they selected a bunch of cancers
13 that they identified as major cancers, and
14 all of the results, it seems, were presented
15 in Figure 1. And in Figure 1, it does
16 appear that all of the risks exceeded 1, and
17 many of them exceeded 1 with the lower bound
18 confidence interval exceeding 1 as well.

19 Q. Okay. On page 3 of Exhibit 10,
20 which is your rebuttal to Dr. Bailey for
21 Mr. Kidd.

22 A. Yes.

23 Q. Here you cite the Bove, et al.,
24 2024 cancer incident study as evidence that
25 there's direct epidemiological evidence of

1 risk at very low levels of carcinogen
2 exposure; correct?

3 A. Correct.

4 Q. Do you believe that individuals
5 stationed at Camp Lejeune between 1953 and
6 1987 were exposed to very low levels of
7 carcinogen exposure?

8 A. Well, I mean, low is a qualitative
9 term, but they certainly weren't exposed to
10 the kind of, what I would say, high levels
11 of these chemicals as would happen in people
12 who were occupationally exposed to those
13 particular solvents, trichloroethylene,
14 perchloroethylene, and benzene.

15 Q. You specifically reference, on
16 pages 4 and 5, the Bove, et al., cancer
17 incident studies adjust hazard ratios for
18 myeloid cancers, acute myeloid leukemia,
19 myelodysplastic and myeloproliferative
20 syndromes; correct?

21 A. Yes. Those are the ones I
22 discussed in that paragraph where they found
23 elevated adjusted hazard ratios.

24 Q. If you will please turn to
25 Exhibit 11, that's your specific causation

1 rebuttal report for Dr. Bailey for
2 Mr. Carter.

3 A. Okay. What page?

4 Q. Page 5 of Exhibit 11.

5 A. Okay.

6 Q. You commented on Dr. Bailey's
7 statement that there's a high likelihood of
8 exposure misclassification in ATSDR's Camp
9 Lejeune studies; right?

10 A. Yes.

11 Q. And you wrote that
12 misclassification is common in
13 epidemiological studies; right?

14 A. Yes.

15 Q. And you wrote that: However,
16 unless there's reason to suspect a
17 differential exposure misclassification that
18 would introduce a specific bias that
19 increases the likelihood of finding an
20 association (where none truly exists),
21 non-differential (i.e., random) exposure
22 misclassification typically results in a
23 bias to the null, i.e., a dilution of any
24 apparent effect.

25 Did I read that correctly?

1 A. You did.

2 Q. Do you have any reason to believe
3 the exposure misclassification in ATSDR's
4 Camp Lejeune studies was non-differential?

5 A. Well, it's the other way around.
6 There's no evidence that the exposure
7 misclassification was differential. In the
8 absence of that, any evidence to show that
9 there's differential exposure
10 misclassification, then the typical exposure
11 misclassification that occurs in a setting
12 like this is non-differential.

13 Q. If Dr. Bove's 2024 cancer incident
14 study used exposure doses that were
15 uniformly 10 percent higher than reality
16 across all exposure classifications, that
17 would constitute a non-differential
18 misclassification bias; correct?

19 ATTORNEY GREENWALD: Objection.
20 Form.

21 THE WITNESS: No. No. Because
22 non-differential misclassification has
23 to -- that affects the results of a
24 study. In terms of general causation
25 has to be differential between those

1 who develop cancer and these who
2 didn't, and that's how you can get a
3 false impression of a elevated risk of
4 cancer. But if the overall estimates
5 of exposure are just 10 percent
6 higher, 10 percent lower, it's
7 10 percent higher or lower for
8 everybody, you know, whether they
9 develop cancer or not, and that would
10 not, theoretically, lead to a type of
11 error that would create a false
12 impression of an association between
13 exposure and disease.

14 BY ATTORNEY ANTONUCCI:

15 Q. If Dr. Bove's study used exposure
16 doses where some were higher than in reality
17 and some were lower in random proportions,
18 that would constitute a differential
19 misclassification bias; correct?

20 A. No.

21 ATTORNEY GREENWALD: Objection.

22 Form.

23 THE WITNESS: I'm sorry.

24 ATTORNEY GREENWALD: That's okay.

25 THE WITNESS: No, because, like

1 you just said, it's random. So it's
2 random; it's not differential.

3 BY ATTORNEY ANTONUCCI:

4 Q. If they were not random or
5 differentiated in that some doses were
6 higher than in reality and some were lower
7 than in reality, that would constitute
8 differential misclassification; right?

9 A. Only if there was a systematic bias
10 where people who had cancer had a different
11 exposure measurement based on the
12 methodology used than the ones who didn't
13 develop cancer.

14 Q. Do you have an opinion on whether
15 ATSDR's water models calculated accurate
16 historical concentrations of contaminants in
17 drinking water?

18 ATTORNEY GREENWALD: Objection.
19 Form.

20 THE WITNESS: All I know is that
21 I'm not aware of any better model.

22 BY ATTORNEY ANTONUCCI:

23 Q. Can you accurately assess the
24 misclassification bias of Dr. Bove's studies
25 without understanding the accuracy of

1 ATSDR's water models?

2 ATTORNEY GREENWALD: Objection.
3 Form.

4 THE WITNESS: I can't estimate
5 that, but like I said, what's most
6 important from a general causation
7 point of view is whether there's
8 not -- where there's differential
9 classification and that -- I've not
10 seen any evidence for that.

11 ATTORNEY ANTONUCCI: Okay. I'd
12 like to take one final break.

13 THE VIDEOGRAPHER: We are now
14 going off the record. The time is
15 5:38 p.m.

16 (Recess taken from 5:38 p.m. to
17 5:50 p.m.)

18 THE VIDEOGRAPHER: We are now back
19 on the record. The time is 5:50 p.m.

20 BY ATTORNEY ANTONUCCI:

21 Q. Dr. Hu, we're back on the record.

22 You understand you're still under
23 oath; correct?

24 A. Yes.

25 Q. Did you discuss the substance of

1 your testimony during the break?

2 A. No.

3 Q. Dr. Hu, has the hypothesis that
4 benzene causes all NHLs because it's
5 associated with pluripotential stem and cell
6 mutation being tested?

7 ATTORNEY GREENWALD: Objection.
8 Form.

9 THE WITNESS: Tested. I think you
10 were referring to research that would
11 either take the form of epidemiology,
12 which would be very difficult to do
13 unless you did bone marrow biopsies on
14 people and followed them prospectively
15 over time, or some type of
16 experimental toxicology study, and I'm
17 not aware of any such research that
18 exists.

19 BY ATTORNEY ANTONUCCI:

20 Q. Dr. Hu, have your opinions as an
21 expert witness ever been excluded by a
22 judge?

23 A. No.

24 Q. Have portions of your opinions as
25 an expert witness ever been excluded by a

1 judge?

2 A. Not that I'm aware of.

3 Q. Dr. Hu, are you aware that you were
4 once excluded from offering testimony that
5 you had diagnosed a plaintiff with multiple
6 chemical sensitivity?

7 A. What year was that?

8 Q. 2000. Do you recall Anello versus
9 Shaw Industries, Inc.?

10 A. Nope. That's 25 years ago.

11 Q. Your opinions have been excluded in
12 more recent cases too, though, haven't they?

13 A. You'd have to refresh my memory
14 because I'm not aware of that.

15 Q. Do you recall testifying in the
16 case Butler versus Mallinckrodt, LLC, in
17 2022 Eastern District of Missouri?

18 A. Yes.

19 Q. Your opinions were excluded in that
20 case, were they not?

21 A. I believe that the opinion of the
22 exposure assessment expert I relied on was
23 excluded, but my opinion was not excluded
24 based on my science but rather on the fact
25 that I relied on his testimony.

1 Q. Okay. And also McMunn versus
2 Babcock and Wilcox Power Generation Group,
3 Inc., in 2013.

4 Do you recall that case?

5 A. I think it was a very similar case
6 with the same exposure assessment expert.

7 Q. Okay. Are there any answers you've
8 given to my questions during this deposition
9 that you wish to change before we end it?

10 ATTORNEY GREENWALD: Objection.

11 Form.

12 THE WITNESS: No.

13 BY ATTORNEY ANTONUCCI:

14 Q. Is there any information I asked
15 about that you didn't recall at the time but
16 now remember?

17 ATTORNEY GREENWALD: Objection.

18 Form.

19 THE WITNESS: I'm sorry. What was
20 that question again.

21 BY ATTORNEY ANTONUCCI:

22 Q. Is there any information I asked
23 about that you didn't recall at the time but
24 now remember?

25 A. Not that I recall.

1 Q. Do you have any other opinions that
2 you've not testified to today?

3 ATTORNEY GREENWALD: Objection.
4 Form.

5 THE WITNESS: Not that are
6 relevant to this case, as far as I
7 know.

8 BY ATTORNEY ANTONUCCI:

9 Q. Once it's ready, you'll be provided
10 with the transcript of this deposition. I
11 ask that you carefully read, correct, and
12 sign the transcript.

13 Do you understand?

14 A. I do.

15 Q. Thank you for your patience in
16 answering my questions today, Dr. Hu.

17 ATTORNEY ANTONUCCI: I pass the
18 witness.

19 ATTORNEY GREENWALD: We're going
20 to take a little break.

21 THE VIDEOGRAPHER: We're now going
22 off the record. The time is 5:54 p.m.

23 (Recess taken from 5:54 p.m. to
24 6:11 p.m.)

25 THE VIDEOGRAPHER: We are now back

1 on the record. The time is 6:11 p.m.

2

3 EXAMINATION

4 BY ATTORNEY GREENWALD:

5 Q. Dr. Hu, I just have a couple of
6 questions to ask you, and it relates to the
7 questions that counsel was asking you about
8 Mr. Carter's stroke. Okay?

9 A. Yes.

10 Q. Do you have an opinion whether the
11 diagnosis of non-Hodgkin's lymphoma
12 increased Mr. Carter's risk of having a
13 stroke?

14 A. Yes.

15 Q. And what is that opinion?

16 A. It does increase the risk of
17 stroke.

18 Q. Okay.

19 A. Including Mr. Carter's stroke.

20 Q. Okay. Can you go to your expert
21 report, your original report, your specific
22 causation, not the rebuttal, which is
23 Exhibit 4, I believe.

24 A. Yes.

25 Q. And go to page 14, please.

1 Are you there?

2 A. Yes.

3 Q. And you say in the sentence above
4 the stars: Thus, given my general causation
5 assessment and the factors reviewed above,
6 it is my opinion to a reasonable degree of
7 medical certainty that the combination of
8 Mr. Carter's exposure to TCE, PCE, and
9 benzene from Camp Lejeune more likely than
10 not was a substantial contributing factor to
11 the causation of his mantle cell lymphoma.

12 And that is still your opinion
13 today; correct?

14 A. Correct.

15 Q. You go on under those lines to say:
16 This concludes my evaluation of Mr. Ronald
17 Carter with respect to the relationship
18 between exposures of TCE, PCE, and benzene
19 that he experienced from Camp Lejeune and
20 the mantle cell lymphoma that he developed
21 (and that was the proximate cause of his
22 death).

23 Do you see that?

24 A. Yes.

25 Q. Do you still hold the opinion that

1 Mr. Carter's exposure to TCE, PCE, and
2 benzene at Camp Lejeune and his mantle cell
3 lymphoma is the proximate cause of his
4 death?

5 A. Yes.

6 ATTORNEY GREENWALD: I don't have
7 any other questions.

8

9 FURTHER EXAMINATION

10 BY ATTORNEY ANTONUCCI:

11 Q. Really brief redirect.

12 First of all, Dr. Hu, since we've
13 come back on the record, you understand that
14 you're still under oath; correct?

15 A. Yes.

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

19 A. I did.

20 Q. What you did you discuss?

21 ATTORNEY GREENWALD: I'm not
22 letting him testify.

23 I'm instructing you not to answer.

24 That's privileged communication.

25 We've gone over this with your

1 lawyers before. You can take it up
2 with the judge if you like.

3 BY ATTORNEY ANTONUCCI:

4 Q. Are you going to listen to your
5 attorney's instruction not to answer my
6 question?

7 A. I will.

8 Q. Okay. I am going to hold the
9 deposition open for the purpose of
10 determining what you discussed during the
11 last break of this deposition under 4th
12 Circuit law.

13 Dr. Hu, you testified that it is
14 now your opinion that Mr. Carter's stroke --
15 the risk of Mr. Carter's stroke was
16 increased by his non-Hodgkin's lymphoma;
17 correct?

18 ATTORNEY GREENWALD: Objection.
19 Form.

20 THE WITNESS: I never testified
21 that wasn't true.

22 BY ATTORNEY ANTONUCCI:

23 Q. When I asked earlier whether you
24 were offering that opinion, you said you had
25 to review the literature in order to offer

1 an opinion on that.

2 What's changed?

3 A. What's changed is the level of
4 certainty. I mean, I quoted two scientific
5 studies in my report -- or this is actually
6 my rebuttal to Dr. Ambinder. And when I was
7 reminded of those studies and their rigor,
8 one was a national study looking at cancers
9 and risk of stroke that included
10 specifically NHL, I was persuaded that, in
11 fact, that's a level of evidence that I
12 could rely on.

13 Q. And you reviewed that study during
14 the break?

15 A. I was reminded of it when I reread
16 the report and et cetera.

17 Q. Okay. So what is proximate cause?

18 A. Proximate cause is essentially the
19 disease process that likely was essentially
20 the most impactful influence on whatever the
21 outcome you're trying to look at.

22 Q. Are you aware that proximate cause
23 is a legal term of art?

24 ATTORNEY GREENWALD: Objection.

25 Form.

1 THE WITNESS: Well, I'm just using
2 my understanding of how we use it in
3 medicine.

4 BY ATTORNEY ANTONUCCI:

5 Q. Okay. So on page 14 of your report
6 for Mr. Carter, when you said that the
7 relationship between exposures to TCE, PCE,
8 and benzene that he experienced from Camp
9 Lejeune and the mantle cell lymphoma that he
10 developed, and that was the proximate cause
11 of his death, you do not mean proximate
12 cause in the legal sense; is that correct?

13 ATTORNEY GREENWALD: Objection.
14 Form.

15 THE WITNESS: I don't even know
16 what the proximate cause means in a
17 legal sense. I just used it from my
18 understanding of how we use it in
19 medicine.

20 ATTORNEY ANTONUCCI: Okay. No
21 further questions.

22 ATTORNEY GREENWALD: I don't
23 either.

24 THE VIDEOGRAPHER: We are off the
25 record at 6:16 p.m., and this

1 concludes today's testimony given by
2 Dr. Howard Hu. The total number of
3 media used was six and will be
4 retained by Golkow, a Veritext
5 division.

6 THE CERTIFIED STENOGRAPHER: Ms.
7 Greenwald, do you need to order a
8 rough draft?

9 ATTORNEY GREENWALD: No.

10 (Whereupon the deposition
11 concluded at 6:16 p.m.)
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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

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.

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E R R A T A S H E E T

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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.

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