

Exhibit 165

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

Case No.
IN RE: 7:23-CV-00897
CAMP LEJEUNE WATER LITIGATION

This Document Relates to:
ALL CASES

April 11, 2025

Videotaped deposition of
KATHLEEN M. GILBERT, PH.D., held at Weitz
and Luxenberg, 700 Broadway, New York, New
York, commencing at 9:30 a.m. EDT, on the
above date, before Marie Foley, a
Registered Merit Reporter, Certified
Realtime Reporter and Notary Public.

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A P P E A R A N C E S :

ON BEHALF OF PLAINTIFF :

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ALSO PRESENT REMOTELY VIA ZOOM:

Matt Quinn

Jim Roberts

Patrick Telan

VIDEOGRAPHER:

Ingrid Rodriguez

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9:32 a.m. EDT
New York, New York
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THE VIDEOGRAPHER: We are now on the record.

My name is Ingrid Rodriguez. I'm a videographer for Golkow Litigation Services.

Today's date is April 11th, 2025, and the time is 9:32 a.m.

This video deposition is being held at Weitz and Luxembourg, PC, New York, New York, in the matter of In Re: Camp Lejeune Water Litigation, in the United States District Court for the Eastern District of North Carolina.

The deponent is Dr. Kathleen M. Gilbert.

Would counsel please state your appearances for the record.

MS. GREENWALD: Robin Greenwald for the plaintiffs.

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MS. GJONAJ: Diana Gjonaj for the plaintiffs.

MS. McKEEVER: Traci McKeever for the United States.

MS. KONSTANTOPOULOS: Melanie Konstantopoulos for the United States.

THE VIDEOGRAPHER: The court reporter is Marie Foley and will now swear in the witness.

THE STENOGRAPHER: If I could ask you to raise your right hand, please.

Do you swear or affirm the testimony you give will be the truth, the whole truth, and nothing but the truth today?

THE WITNESS: I do.

THE STENOGRAPHER: Thank you.

- - -

KATHLEEN M. GILBERT, PH.D., the Witness herein, having been first duly sworn by a Notary Public in and of the State of New York, was examined and testified as follows:

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EXAMINATION BY

MS. McKEEVER:

Q. Good morning again, Dr. Gilbert. We met briefly this morning. Again, I'm Traci McKeever from DOJ.

Have you had your deposition taken before?

A. Yes.

Q. Okay. So this will be very similar to that. I'm just going to go through a few ground rules before we get started.

I will ask questions, and please answer them to the best of your ability, but if you don't understand a question, please let me know and I will rephrase the question. If you answer the question, I will assume you understood it.

Can we agree to that?

A. Yes.

Q. In a normal conversation, it is typical that you may understand what I'm asking before I complete the question, but for the court reporter and the record,

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I'll just ask that you let me finish my question and I will let you finish your answer.

Can we agree to that?

A. Yes.

Q. And you're doing a great job so far, but when you answer a question, please say your answers with a yes or a no rather than nodding or shaking your head, so that the court reporter can get it on the record.

Can we agree to that?

A. Yes.

Q. Wonderful.

Can you please state your full name for the record?

A. Kathleen Mary Gilbert.

Q. And what is your current business address?

A. It's 800 Shire Court, Fort Collins, Colorado.

Q. This is a court proceeding, even though we're not in a courtroom, and you took an oath just a moment ago.

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You understand that you're obligated to tell the truth?

A. Yes.

Q. Please talk at a reasonable pace so that the court reporter can record everything, and again, we should try not to interrupt or speak over one another.

During the deposition, you may hear one of the other attorneys say "objection." Unless your attorney instructs you not to answer the question, please answer the question after the objection has been made.

Is there any reason why you are unable to give your most truthful and accurate testimony today?

A. No.

Q. Is there any reason your memory might be impaired today?

A. No.

Q. And finally, you may ask for breaks. I will be taking some breaks. I will just ask that you answer any question before we take the break.

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Do you understand?

A. Yes.

Q. All right, wonderful.

MS. McKEEVER: I'm going to start with Exhibit 1.

For the record, this is Notice of Deposition and Request For Production of Documents to Kathleen M. Gilbert, Ph.D.

(Gilbert Exhibit 1, Notice of Deposition and Request For Production of Documents to Kathleen M. Gilbert, Ph.D., was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. So, just take a moment and look at that.

Have you seen this document before?

A. Yes.

Q. And you understand that it requires your presence at the deposition today?

A. Yes.

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Q. And it also requests some documents.

A. Yes.

Q. Did you bring any documents with you in response to the subpoena?

A. The invoices that you requested.

Q. Okay, wonderful.

MS. GREENWALD: (Handing.)

I have an extra.

MS. McKEEVER: We'll look at these during the break.

BY MS. McKEEVER:

Q. Turn to Attachment A, please. And I'm just going to go through these.

It will be a little bit of reading, but I'm just going to go through these quickly.

Attachment A asks: Pursuant to the Federal Rules of Civil Procedure 32 B 2 and 45, United States makes the following request for the production of non-privileged documents, communications and materials, including but not limited to any electronically stored information,

1
2 data, technical files and photographs
3 within your possession, custody, or
4 control.

5 And then number 1 requests: All
6 emails, letters, correspondence, text
7 messages, conversations, chats,
8 voicemails, data, technical files or other
9 communications pertaining to Camp Lejeune
10 sent or received prior to your retention
11 as an expert in this matter, including but
12 not limited to, from or with.

13 And A is Morris Maslia and B is
14 David Savitz.

15 Did you have anything in
16 response to question 1?

17 A. No.

18 Q. And then number 2 asks for a
19 similar series of documents from a number
20 of people: Robert Faye, Jason Sautner,
21 Rene Suarez-Soto, Susan Martel, Scott
22 Williams, Frank Bove, Mike Partain, Jerry
23 Ensminger, Lori Freshwater, and Paul
24 Rosenfeld.

25 Did you have anything in

1
2 response to question 2?

3 A. No.

4 Q. All right. And number 3 asks
5 for all emails, letters, correspondence,
6 text messages, conversations, chats,
7 voicemails, or other communications to or
8 from or with any individual who has filed
9 a claim with the department of the Navy or
10 the eastern districts of north core line
11 pursuant to the Camp Lejeune justice act
12 of 2022.

13 Do you have anything in response
14 to number 3?

15 A. No.

16 Q. And then am I correct in
17 understanding that the invoices your
18 counsel handed me a moment ago are in
19 response to number 4?

20 A. Correct.

21 Q. All right. And again I will
22 review those during a break, and we may
23 come back and ask a few questions about
24 those.

25 Am I correct that you have been

1
2 retain by the plaintiffs' leadership group
3 as an expert witness in the Camp Lejeune
4 water litigation?

5 A. Yes.

6 Q. When were you hired?

7 A. I think it was about -- with
8 regard to the plaintiffs' leadership, it
9 was about a year ago.

10 Q. What do you mean when you say
11 "with regard to the plaintiffs'
12 leadership"?

13 A. I did some general consulting
14 with some of the law firms that eventually
15 ended up being associated with the Bell
16 Legal Group.

17 Q. Okay. And that general
18 consulting was related to Camp Lejeune?

19 A. It was more general consulting
20 with regard to the health effects of TCE.

21 Q. And do you recall which law
22 firms?

23 A. Pat Telan.

24 Q. That's the only one?

25 A. Yes.

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Q. Okay.

Who hired you to be an expert in this case?

A. What do you mean "hired"?

Q. When you transition from the general consulting work to understanding that you are going to be an expert in the Camp Lejeune water litigation, who hired you for that position?

MS. GREENWALD: Objection; form. You can answer.

A. I don't remember receiving a specific invitation from someone.

Pat Telan eventually ended up being part of Bell Legal, and the -- my work just continued.

Q. Okay.

Before being retained as an expert and before your consulting work that you mentioned a moment ago, had you heard about Camp Lejeune?

A. Camp Lejeune in general?

Q. Camp Lejeune water contamination.

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A. Yes.

Q. Okay. And what did you know about that?

A. As someone who's studied trichloroethylene for many, many years, I am aware of Camp Lejeune. It's an example of environmental exposure.

Q. And when did you first become aware of Camp Lejeune water contamination?

A. I don't remember. It's years ago.

Q. Did you do anything to prepare for your deposition today?

A. Reviewed some of the reports, looked over some of the studies.

Q. And which reports are you referring to?

A. My three expert reports.

Q. The three expert reports you produced in this case?

A. Yes.

Q. And which studies are you referring to?

A. I can't remember all of them.

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Q. Numerous studies?

A. Numerous studies.

Q. Other than reviewing some reports, your reports and some studies, did you do anything else to prepare?

MS. GREENWALD: Objection; form.

A. What do you mean did I do anything else to prepare?

Q. Did you meet with anyone?

A. I met with these two attorneys yesterday.

Q. Okay.

How many times did you meet with your attorneys?

A. With regard to this deposition?

Q. Yes.

A. Just the once.

Q. And how long was the meeting?

A. Maybe six or seven hours.

Q. Was anyone else other than the attorneys present during the meeting?

A. No.

Q. Did you speak to anyone other than the attorneys about preparing for

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your deposition?

A. No.

Q. Have you spoken to a plaintiff in this lawsuit about your deposition?

A. No.

Q. Have you reviewed any of the other depositions taken in this case?

A. I've looked at some of them.

Q. Which ones?

A. I can't remember off the top of my head.

I've looked at the Bove deposition.

Q. How about Morris Maslia?

A. No.

Q. And he was deposed in 2024. You haven't reviewed that deposition?

A. I don't remember reviewing that.

Q. And he was deposed in 2010. You haven't reviewed that deposition?

A. Correct.

Q. The deposition of Christopher Rennix?

A. No.

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Q. The deposition of Susan Martel?

A. No.

Q. Can you think of any other deposition you reviewed other than Dr. Bove?

A. I can't remember any other.

MS. McKEEVER: Let's move on to the next exhibit. This is -- sorry, let me just -- I will represent that this is what was attached to your reports as your CV, and it says at the top of the page "Kathleen M. Gilbert, Ph.D., Immunotoxicologist."

(Gilbert Exhibit 2, CV of Kathleen M. Gilbert, Ph.D., Immunotoxicologist, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. You could just take a moment to look at that.

A. Okay.

(Witness reads document.)

Q. Do you recognize this document?

A. Yes.

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Q. And what is it?

A. It is my CV.

Q. Is this a complete and accurate copy of your CV?

A. This is a complete and accurate copy of the shorter version of my CV.

Q. And I take it from that answer that there is a longer version of your CV?

A. There is a version that one uses when you're applying for tenure that has all the comments from students and all that sort of thing that really is not relevant for most what I'm doing now.

Q. I understand.

Is there anything relevant to this litigation that you need to add to your CV today?

A. I don't believe so.

Q. Is there anything you need to change?

A. No.

Q. Page 21 lists some litigation history.

So, you have prepared expert

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reports before, correct?

A. Yes.

Q. And how many times?

A. I'd have to go through this list and add them up.

Q. Well, let's just look at the first one listed, Jodelle L. Kirk.

Did you prepare an expert report in that case?

A. Yes, I did.

Q. And were you working on behalf of the plaintiffs or the defendants?

A. The plaintiffs.

Q. Okay.

And then the Hostetler case, it looks like you wrote an expert report in that case as well.

A. Yes.

Q. And were you working on behalf of the plaintiff or the defendant?

A. The plaintiff.

Q. And then in the Asher case, I can't tell, did you prepare an expert report in that case?

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A. That was just an emergency hearing. So I didn't prepare, I don't believe, a expert report for that one.

Q. It just says: Wrote affidavit concerning human health effects of --

A. Right.

Q. -- chloride.

A. Right.

Q. So you prepared an affidavit?

A. Right.

Q. And was that on behalf of the plaintiff or defendant?

A. Plaintiff.

Q. And the next one is the Millman case, and it looks like you wrote an expert report in that case, correct?

A. Yes.

Q. And was that on behalf of the plaintiff or defendant?

A. Plaintiff.

Q. And on the next page, Houlihan, it looks like you wrote an expert report in that case, correct?

A. Yes.

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Q. And was that on behalf of plaintiff or defendant?

A. Plaintiff.

Q. Okay.

Funderburk is the next one. It looks like you wrote an expert report in that case, correct?

A. Yes.

Q. And was that on behalf of plaintiff or defendant?

A. Plaintiff.

Q. And Taylor, I can't tell if you wrote an expert report in that case.

A. I did not.

Q. You did not, okay.
And why not?

A. As it was explained to me by counsel, in the State of Missouri, you don't have to write a expert report in order to be deposed.

Q. Okay. But you gave a deposition?

A. Yes.

Q. And was that on behalf of the

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plaintiff or defendant?

A. Plaintiff.

Q. Okay. And then it just says:
Preliminary research 2022. And then:
Generated report on epidemiological
studies linking trichloroethylene exposure
and human health.

Was that with respect to a
particular case?

A. They were deciding about whether
to get involved in a certain case. So
they asked me to do some preliminary
research for them.

Q. Okay.

Did you produce an -- an
official expert report?

A. No.

Q. And was this work on behalf of a
plaintiff's law firm or a defendant's law
firm?

A. Plaintiff.

Q. Okay.

And then the last entry says
again "Preliminary Research 2022" for the

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Romanucci law firm. And again it just says: Performed risk assessments for 19 plaintiffs.

But no expert report?

A. Correct.

Q. Okay.

And have you ever testified at trial?

A. One time.

Q. And can you identify which case that was?

A. The Jodelle Kirk case.

Q. Have you otherwise been involved in litigation?

A. What do -- what do you mean?

Q. I mean personal litigation.

A. No.

Q. Any class action or products liability litigation?

A. That I was involved in personally?

Q. Yes.

A. No.

Q. How long have you been serving

1
2 as an expert witness?

3 A. About eight years.

4 Q. And why did you begin working as
5 an expert witness?

6 A. The law firm representing
7 Jodelle Kirk contacted me and asked me to.

8 Q. Does your payment depend on the
9 outcome of this case?

10 A. No.

11 Q. And I'll come back to some of
12 the billing questions later.

13 Has your testimony ever been
14 excluded, to your knowledge?

15 A. One time one of the Indiana
16 cases my testimony was, I don't know if
17 you use the word "excluded." It was
18 limited.

19 Q. And what was the basis of that
20 limitation?

21 A. It was whether you could use
22 regulatory values to talk about future
23 risk as opposed to having actual values
24 that the plaintiffs were exposed to.

25 Q. Individual exposure values?

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A. Mm-hm. Yes.

Q. Thank you.
Which Indiana case?

A. I honestly don't remember.

Q. Has any of your work focused on
the impact of TCE on cancer?

A. You mean my actual experimental
work?

Q. Yes.

A. Indirectly, yes.

Q. And what do you mean by
"indirectly, yes"?

A. Well, because I worked on the
effects of TCE on the immune system, and
since the immune system is such a large
part of carcinogenesis, then you can say
that it is indirectly related.

Q. Okay. Let's move on to your
reports.

MS. McKEEVER: I'm going to go
ahead and introduce all three.

The first one is General
Causation Expert Report of Kathleen M.
Gilbert, Ph.D., TCE, PCE, Benzene and

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Bladder Cancer.

And I think this is Exhibit 3.

(Gilbert Exhibit 3, General Causation Expert Report of Kathleen M. Gilbert, Ph.D., TCE, PCE, Benzene and Bladder Cancer December 9, 2024, was marked for identification, as of this date.)

MS. McKEEVER: The second one is General Causation Expert Report of Kathleen M. Gilbert, Ph.D. TCE, Non-Hodgkin's Lymphoma, and Leukemia. Exhibit 4.

(Gilbert Exhibit 4, General Causation Expert Report of Kathleen M. Gilbert, Ph.D. TCE, Non-Hodgkin's Lymphoma, and Leukemia, was marked for identification, as of this date.)

MS. McKEEVER: And the third report is General Causation Expert Report of Kathleen M. Gilbert, Ph.D. TCE and Kidney Cancer, Exhibit 5.

(Gilbert Exhibit 5, General Causation Expert Report of Kathleen M.

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Gilbert, Ph.D. TCE and Kidney Cancer, was marked for identification, as of this date.)

MS. McKEEVER: I'm also going to mark some materials we received separate from your report, and I'm going to put them all together in one exhibit.

(Pause.)

MS. McKEEVER: All right. I'm including four different documents in this exhibit.

The first document is Dr. Kathleen Gilbert Additional Materials Considered Bladder Cancer.

The second one is Dr. Kathleen Gilbert Additional Materials Considered General Causation Expert Report of Kathleen M. Gilbert, Ph.D., TCE, Non-Hodgkin Lymphoma and Leukemia, dated December 9th, 2024.

The third is a long reference list just called December 2024 General Causation Expert Report of Kathleen M.

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Gilbert, Ph.D., TCE, Non-Hodgkin
Lymphoma and Leukemia and Materials
Considered List.

And the last one is Dr. Kathleen
Gilbert Additional Materials
Considered Kidney Cancer.

And I'm going to mark those as
Exhibit 6.

(Gilbert Exhibit 6, additional
supplemental materials, was marked for
identification, as of this date.)

MS. McKEEVER: Yesterday evening
after the close of business I received
some additional supplemental materials
for all three reports. I have not
reviewed those yet, so I'm reserving
the right to continue the deposition,
if necessary, due to the late
supplement, but we're going to move
forward for now.

BY MS. McKEEVER:

Q. Okay. If you could take a
moment to look at Exhibit 3, which I
believe is your bladder cancer report.

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Okay. Have you had a moment?

A. (No verbal response.)

Q. Is that your signature on page 34?

A. Yes.

Q. Okay.

So, going forward, I will probably refer to Exhibit 3 as your bladder cancer report.

And then on Exhibit 4, if you'll take a moment to look at that. It should be the leukemia/NHL.

Is that your signature on the cover page?

A. Yes.

Q. And so going forward, I will probably refer to this as your NHL, leukemia report.

And then Exhibit 5, do you recognize this document?

A. Yes.

Q. And is that your kidney cancer report?

A. Yes.

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Q. And is that your signature on page 34?

A. Yes.

Q. And so moving forward, I will refer to this as your kidney cancer report.

Okay. If you could turn to page 8 of the leukemia/NHL report.

Okay. I am reading from the third full paragraph and it says: With respect to the causation standard I employed in reaching my opinions, I have reviewed the Camp Lejeune Justice Act (CLJA) and am aware the causation standard under the CLJA explains that the plaintiffs in this case must show the relationship between exposure to the water at Camp Lejeune and the harm is: A, sufficient to conclude that a causal relationship exists, or B, sufficient to conclude that a causal relationship is at least as likely as not.

ATSDR Assessment of Evidence referenced above considers the at least as

1
2 likely as not standard to be the
3 functional equivalent of its category for
4 equipoise and above. Although my opinions
5 in this case are expressed to the higher
6 more likely than not standard, the ATSDR's
7 definition of equipoise and above served
8 as guidance for me in this case. These
9 classifications and categories are
10 consistent with my education, training,
11 and experience with the sciences to which
12 they relate.

13 Did I read that correctly?

14 A. Yes.

15 Q. Okay.

16 Would you use the phrase "at
17 least as likely as not" in a scientific
18 manuscript?

19 MS. GREENWALD: Objection to
20 form.

21 A. My scientific manuscripts are
22 all about basic science. So if I talk
23 about epidemiology, I'm referring to
24 someone else's conclusions in those
25 things.

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So I -- I don't use that expression. I have never used that expression in my basic science research.

Q. What about "more likely than not"?

MS. GREENWALD: Same objection.

A. Neither. I mean, I have not used that one either.

Q. You wouldn't use the phrase "equipoise" in a publication on your research, would you?

MS. GREENWALD: Objection; form.

A. Equipoise as far as I understand it is more of a legal term than a scientific term. And no, I have never used that.

Q. Were you provided with the burdens and standard of proof section of the Camp Lejeune Justice Act?

A. Yes.

Q. Did you review the complete language of that statute?

A. I looked at it. I don't remember it in detail.

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Q. And you're not a lawyer,
correct?

A. Correct.

Q. And you don't have legal
training?

A. Correct.

Q. You don't have a law degree?

A. Correct.

Q. Have you ever done research on
the topic of legal burdens of proof?

A. No.

Q. Have you used "more likely than
not" in any of your other expert opinions?

MS. GREENWALD: Objection; form.

A. I don't honestly remember.

Q. Going back to the equipoise
language we discussed a moment ago. You
said: Although my opinions in this case
are expressed to a higher more likely than
not standard, the ATSDR's definition of
equipoise and above served as a guidance
for me in this case.

Again did I read that correctly?

A. Yes.

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Q. What do you mean by "the ATSDR's definition of equipoise and above served as guidance for me in this case"?

A. I took it into consideration when I was looking at the -- the material.

Q. In what way?

A. Comparing whether a particular study met, or the cumulative exposures, met that criteria.

Q. Met the equipoise criteria?

A. Yes.

Q. Are you aware that the ATSDR's 2017 Assessment of Evidence, which I believe you've cited to in your reports, took its classification scheme of equipoise from the IOM scheme for VA presumption which gives the veteran the benefit of the doubt?

MS. GREENWALD: Objection; form.

A. I don't remember that.

Q. Have you used equipoise in your non-litigation work to prove the probability of association?

A. Once again, I think equipoise is

1
2 more of a legal term than a basic science
3 term. So I don't believe I've ever used
4 it in my own basic science research.

5 Q. You reviewed Dr. Bove's
6 deposition, correct?

7 A. I didn't read it in depth, but I
8 looked at parts of it, yes.

9 Q. If Dr. Bove testified that
10 equipoise and above gives the subject the
11 benefit of the doubt, you wouldn't
12 disagree with that, would you?

13 MS. GREENWALD: Objection; form.

14 A. Could you repeat that question?

15 Q. If Dr. Bove testified that
16 equipoise and above gives the subject the
17 benefit of the doubt, you wouldn't
18 disagree with that?

19 A. I just --

20 MS. GREENWALD: Objection; form.
21 Go ahead.

22 A. I just don't know enough about
23 it to be able to comment on that.

24 Q. Fair enough.

25 Let's see. On page 8 of the

1
2 leukemia/NHL report, Exhibit 4. Yes,
3 thank you.

4 I am now reading from the
5 paragraph -- the paragraph above the one I
6 just read and it says: Similar to the EPA
7 and the ATSDR, I used a modified Bradford
8 Hill approach to derive my opinions. This
9 means I considered results from
10 epidemiological, animal, and mechanistic,
11 sometimes in vitro studies. The
12 importance of these three components in
13 determining a causal relationship will be
14 described below.

15 Did I read that correctly?

16 A. Yes.

17 Q. And now let me ask for the
18 ATSDR.

19 MS. McKEEVER: All right.

20 I'm marking as the next exhibit
21 ATSDR Assessment of the Evidence For
22 the Drinking Water Contaminants At
23 Camp Lejeune and Specific Cancers and
24 Other Diseases, dated January 2017.

25 (Gilbert Exhibit 7, ATSDR

1
2 Assessment of the Evidence For the
3 Drinking Water Contaminants At Camp
4 Lejeune and Specific Cancers and Other
5 Diseases, dated January 13, 2017,
6 Bates CLJA_HEALTHEFFECTS-0000044276-427,
7 was marked for identification, as of
8 this date.)

9 MS. McKEEVER: Did I say the
10 wrong exhibit? Exhibit 7.

11 BY MS. McKEEVER:

12 Q. If you could turn to page 8,
13 please: And I'm looking at the language
14 beginning in the very last sentence of
15 page 8 and continuing to page 9. It says:
16 Instead, ATSDR assessment of the
17 epidemiological evidence considered some
18 of the viewpoints associated with Hill.
19 1, temporal relationship; 2, magnitudes of
20 the effect, e.g. risk ratio, odds ratio,
21 and standardized mortality ratio; 3,
22 consistency of findings; 4, exposure
23 response relationship, although the
24 relationship could be non-linear or
25 non-monotonic; and 5, biological

1
2 plausibility (Hill 1965). When
3 considering the magnitude of the effect
4 estimate, an effect estimate was
5 considered near the null value if less
6 than or equal to 1.10 and elevated if
7 greater than 1.10. Also considered were
8 the effects of biases and particular
9 exposure misclassification, healthy worker
10 effect, and confounding.

11 Do you see what I'm talking
12 about?

13 A. Yes.

14 Q. So, is it your understanding
15 that this is the modified Hill approach?

16 A. What do you mean "the modified
17 Hill approach"?

18 Q. Well, you said: Similar to the
19 EPA and ATSDR, I used a modified Bradford
20 Hill approach to drive -- to derive my
21 opinions.

22 Is this the modified Hill
23 approach that you used?

24 A. I don't know that it was
25 strictly the same as the ATSDR's modified

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Bradford Hill approach.

Q. In what way was yours a modified Bradford Hill approach?

A. So, Bradford Hill is much more of a list of suggestions to include in your causal determination. So I took that into consideration and used some of them as -- and even Dr. Hill in his -- in his speech explains why some of them are more important than others and that you don't have to be rigorously found that each one of these are important.

So, in that respect, I used a modified Bradford Hill calculation.

Q. Do you normally in your research consider studies that do not show linear exposure response relationship to be evidence of causality?

MS. GREENWALD: Objection; form.

A. Could you say that again?

Q. Do you normally in your research consider studies that do not show a linear exposure relationship to be evidence of causality?

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MS. GREENWALD: Same objection.

A. Linear relationships are very difficult to find in epidemiology studies. So I think there are plenty of reasons why you do not have to be tied strictly to that criteria. Even Dr. Hill says that in his report.

Q. And so is the answer yes, you do consider studies in your research that don't show a linear relationship?

A. Yes.

Q. And do you in your research consider studies that have a non-monotonic dose response relationship as evidence of causality?

MS. GREENWALD: Objection; form.

A. Once again, that is a criteria that is very difficult to meet, as Dr. Hill says himself. So yes, I do consider studies that are not strictly monotonic.

Q. Do you give greater weight to studies that show a linear response?

MS. GREENWALD: Objection; form.

A. Since you almost never find

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studies that show a linear response, I can't really answer that.

Q. And what about studies that show a monotonic response, would you give greater weight to those studies?

MS. GREENWALD: Objection; form.

A. Greater weight than what?

Q. Than a study that does not show a monotonic response.

A. There's so many different --

MS. GREENWALD: Objection; form.

Sorry, go ahead.

A. There's so many different aspects of that that I can't answer it as a yes or no.

Q. Okay. Let's go to page 12 of the leukemia/NHL report, Exhibit 4.

I'm on page 12 of Exhibit 4, and I'm looking at the fifth full paragraph which says: For inclusion in the epidemiological assessment of NHL and leukemia that I performed, studies had to demonstrate, 1, a temporal relationship between chemical exposure and negative

1
2 health effects, i.e. exposure precedes
3 toxicity; and 2, convincing positive
4 associations represented by a risk ratio
5 (RR), odds ratio (OR), standardized
6 mortality ratio (SMR), or standardized
7 incident ratio (SIR) greater than 1.1; 3,
8 biological plausibility; 4, adult
9 exposure; 5, exposure durations of more
10 than one month; and 6, ability to
11 distinguish TCE specific effects.

12 Did I read that correctly?

13 A. Yes.

14 Q. Does this same statement apply
15 to the epidemiological assessments you
16 performed in your other two reports?

17 A. I believe so. I -- I believe
18 that paragraph was in the other reports.

19 Q. I believe so too. I was just
20 trying to avoid asking you the same
21 question about each of the reports.

22 Regarding number 2: Convincing
23 positive associations represented by a
24 risk ratio, odds ratio, standardized
25 mortality ratio, or standard incident

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ratio greater than 1.1.

Is it your opinion that a risk ratio at 1.1 or above represents a strong association?

MS. GREENWALD: Objection; form.

A. That is a tricky question when you're talking about epidemiology. Because so many particular diseases are relatively rare, sometimes you are -- you have to pay attention to anything over 1.1.

Q. What level of increased risk reflects a modest association, in your opinion?

A. You can't answer that question.

Q. Are you aware that David Savitz has written in his book "Epidemiology and the Law" that 1.2 is a modest association?

MS. GREENWALD: Objection; form.

A. I was not aware of that.

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

MS. GREENWALD: Objection; form.

A. Once again, impossible to

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answer.

Q. And why is it impossible to answer?

A. Because there's so many aspects that go into each epidemiological study that there is no -- I would find it difficult to describe a strict number that reflects a medium or a high odds ratio as being more important than something else.

Q. And what are some of the factors you're considering when you say it's impossible to know?

A. It has to do with the number of -- of subjects, a lot of it. It has to do with how much is known about the actual exposure.

There are many factors that go into it.

Q. Are you aware that David Savitz has written his become -- in his book "Epidemiology and the Law" that 2.5 is a sizable association?

MS. GREENWALD: Objection; form.

A. I was not aware.

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Q. Would you disagree with that?

A. Once again, it's not something I've really sat down and considered and come up with a -- a -- a gradation in that regard.

Q. Why did you use the 1.1 odds ratio as a benchmark?

A. Because that's what the -- the is the most common in terms of finding something that is at least memorable.

Q. What do you mean by "most common"?

A. It is a number that is -- I have seen used in many studies as their cutoff point.

Q. Their cutoff point for what?

A. Determining whether a response had occurred.

Q. And have you used the 1.1 odds ratio as a benchmark in your other expert reports?

A. Yes.

Q. Which ones?

A. These other two.

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Q. These other two.

Outside of this case, have you used 1.1 as a benchmark in any of your other expert reports?

A. I don't remember.

Q. Have you used the 1.1 odds ratio as a benchmark in any peer-reviewed publication that you have authored or co-authored?

MS. GREENWALD: Objection; form.

A. Since my work does not involve epidemiology as far as what we conducted, I have never used that in my own research.

Q. I don't think I understood what you just said. You said in your work.

A. Sorry.

So, my work is basic science. The 1.1 odds ratio has to do with epidemiology.

Q. I understand.

So in your research, the tests that you perform, you don't use the 1.1 odds ratio because it's an epidemiological concept?

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A. Correct.

Q. Are you an expert in epidemiology?

A. I'm an expert in TCE health effects, and in order for that to be true, I have to know a lot about epidemiology.

Q. What sort of epidemiology training do you have?

A. It is -- it centers on my years of reading the literature.

Q. Okay.

So would you consider yourself an epidemiologist?

A. I am not an epidemiologist.

Q. Are you aware that the EPA has characterized an odds ratio of between 1.3 and 2.0 as evidence of a positive association?

MS. GREENWALD: Objection; form.

A. No, I was not aware.

Q. Do you agree with that characterization?

MS. GREENWALD: Objection; form.

A. I think we've already discussed

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that.

Q. And I assume if I ask you are you aware that the EPA has characterized an OR of over 3.0 as evidence of a strong, you're aware of that. Is that correct?

MS. GREENWALD: Objection; form.

A. That's true.

Q. And just for the record, OR stands for odds ratio.

Okay. Going back to your assessment in this case. Looking at, we're still on the leukemia/NHL report, and I have turned to page 14, table 1 "Epidemiological Studies Showing That TCE and Other Chemicals Cause NHL". And this is Exhibit 4.

When you were putting together -- did you put together this table?

A. Yes.

Q. When you were putting together this table, does the risk ratio or odds ratio have to be at least 1.1 to be included in the table?

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A. Yes.

I think there might have been a couple of exceptions, but generally, yes.

Q. Did the confidence interval have to fall within a certain range for you to have included it in this table in your report?

A. No.

Q. Did you consider any studies that did not show an association in your epidemiological assessment?

A. Of course.

Q. And are those included in this table?

A. They are not.

Q. And why not?

A. Because I made that with the causation standard associated with the Camp Lejeune Act in mind. So, they wanted to -- information to show that this was sufficient to conclude that a causal relationship exists. So I used the studies that did that.

Q. Okay.

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And you're -- when you mention the causation standard, you're talking about the Camp Lejeune causation standard?

A. Yes.

Q. And that's a legal standard, correct?

A. I believe so.

Q. So, on page 12 under Section A "Epidemiological Studies," the next to last sentence says: I used Pub Med, the free online NIH-sponsored database of medical science studies, to identify epidemiological studies published between 2020 and 2024.

Right?

A. Yes.

Q. And when you did that search, did you find any studies that did not show an association?

A. Yes.

Q. But those studies are not included in your table 1 on page 14, correct?

A. Correct.

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Q. And are you aware that David Savitz in his book "Epidemiology and the Law" has said that: An overall assessment considers the full range of studies that provide pertinent information regardless of their results and integrates the full range of relevant studies?

A. I was --

MS. GREENWALD: Objection; form.
Go ahead.

A. I was not aware of that statement.

Q. Do you agree with the statement?

MS. GREENWALD: Objection; form.

A. Could you read the statement again?

Q. An overall assessment considers the full range of studies that provide pertinent information regardless of their results and integrates the full range of relevant studies.

MS. GREENWALD: Same objection.

A. Yes, I would agree in general that's true.

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Q. But that wasn't the purpose of your table 1, correct?

A. I looked at all the studies and took into account the positive ones and negative ones. So in essence, I did what he was suggesting.

I just put the ones in the table that satisfied the -- what I thought was the need to show a causal relationship.

Q. Okay.

MS. McKEEVER: If we could take a short break here.

THE WITNESS: That would be great.

THE VIDEOGRAPHER: The time right now is 10:19 a.m.

We're off the record.

(Recess taken.)

THE VIDEOGRAPHER: The time right now is 10:48 a.m.

We're back on the record.

BY MS. McKEEVER:

Q. All right. Welcome back. I'm going to ask you some

1
2 questions about your reports, starting
3 with the bladder cancer report which was
4 Exhibit 3, I believe.

5 Ready?

6 A. Ready.

7 Q. Did you prepare all the opinions
8 in this report?

9 A. Yes.

10 Q. Is there anything in this report
11 that you believe is incorrect?

12 MS. GREENWALD: Objection; form.

13 A. No.

14 Q. Is there anything in this report
15 that needs to be updated?

16 A. Can't say for sure about that.
17 I mean, there are new studies and things
18 that come out all the time. So I
19 can't --

20 Q. That you're aware of today?

21 A. I believe we sent you that
22 supplemental report about bladder cancer.
23 So would you call that an update?

24 Q. Let's look at, what's the
25 exhibit number for the supplements?

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MS. KONSTANTOPOULOS: 6.

BY MS. McKEEVER:

Q. 6.

So, there are -- in Exhibit 6 there are a number of supplemental materials listed.

So, if you look at your report, the materials cited in your report, and the supplemental materials, and understanding that we got an additional supplement last night that we haven't reviewed, is there anything else that you are aware of today that would need to be updated in your report?

A. I don't believe so.

Q. Okay.

Does your report contain a complete statement of all the opinions that you will offer at trial or to the court on bladder cancer?

MS. GREENWALD: Objection; form.

A. I'm not sure I know what that -- what you're asking.

Q. Well, we covered that you

1
2 prepared all the opinions in your report
3 and that sitting here today you can't
4 think of anything that needs to be
5 updated, right?

6 A. Correct.

7 Q. And so, as far as you're aware
8 today, your report is a complete statement
9 that you would offer at trial. Is that
10 right?

11 MS. GREENWALD: Objection; form.

12 BY MS. McKEEVER:

13 Q. You haven't supplemented your
14 report?

15 A. I have not supplemented my
16 report.

17 Q. Okay.

18 Have you reviewed or considered
19 any other expert reports in this case?

20 A. I believe I looked at the -- the
21 Goodman and the McCabe expert reports.

22 Q. Did you review the rebuttal
23 report of David Savitz?

24 A. No.

25 Q. Did you review the rebuttal

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report of Madigan?

A. No.

Q. Let me ask you to turn to page 3 of the bladder cancer report.

In the second full paragraph you say: My report will focus on TCE since that is the contaminant that has been the nexus of my own research. However, PCE and benzene will be discussed as sources of added toxicity.

Did I read that correctly?

A. Yes.

Q. Do you feel that you're qualified to give an opinion on benzene?

A. I feel that I am qualified to have an opinion on benzene as how it may interact with trichloroethylene.

Q. And do you feel that you're qualified to give an opinion on PCE?

A. The same answer.

Q. And that answer is how it interacts with trichloroethylene?

A. Well, I know a bit more, quite a bit more about PCE because it is very

1
2 similar to TCE and because I was on the
3 SAC committee that reviewed the risk
4 assessment for PCE, and I actually wrote a
5 lot of the human -- the -- the reports
6 as -- not the reports. The reviews of the
7 human health aspects on that particular
8 risk assessment.

9 Q. Okay.

10 Turning to page 10, your opinion
11 1 says: The contaminants in the drinking
12 water at Camp Lejeune more likely than not
13 caused bladder cancer.

14 Right?

15 A. Yes.

16 Q. Which contaminants?

17 A. Well, as I -- it's hard to break
18 them down. Be -- I don't know that I
19 could specify that one causes more than
20 the other.

21 Obviously perchloroethylene has
22 a lot of epi's showing that it causes
23 bladder cancer, and so the bottom -- the
24 bottom line is that I don't know that I
25 could break it down for you.

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Q. When you say "perchloroethylene," the abbreviation for that is PCE, correct?

A. Yes.

Q. You have not offered an opinion in your report that vinyl chloride causes bladder cancer, correct?

A. Correct.

Q. And is it your opinion that PCE alone causes bladder cancer?

A. Well, like I just said, it's -- it's really hard to say for sure.

Q. And is it your opinion that benzene alone causes bladder cancer?

A. Same answer.

Q. And is it your opinion that TCE alone causes bladder cancer?

A. Difficult to say.

Q. Turning to page 4. You've mentioned that you relied on the ATSDR Assessment of Evidence for the drinking water contaminants at Camp Lejeune.

Correct?

A. Where do you see that on this

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page?

Q. It is among your materials listed --

A. Okay.

Q. -- in numerous places, is that right?

A. Yes.

Q. The ATSDR Assessment of Evidence 2017?

A. Yes.

Q. And on page 16, you have said that: ATSDR concluded that there was not sufficient evidence for causation for TCE and bladder cancer.

Right?

A. Yes.

Q. And you acknowledge that, also on page 16: ATSDR concluded that there was not sufficient evidence for causation for benzene and bladder cancer.

A. Yes.

Q. And then it says: But it should be noted that evidence for benzene exposure as a risk factor for bladder

1
2 cancer has been published since the 2017
3 ATSDR evaluation.

4 What evidence are you referring
5 to?

6 A. I would have to go back and look
7 at that table. I don't remember off the
8 top of my head.

9 Q. And which table are you
10 referring to?

11 A. The epi table.

12 Q. The epi table in this report,
13 table 1 of the bladder cancer report?

14 A. Correct.

15 Q. Okay. Let's go --

16 MS. McKEEVER: Excuse me. Could
17 we just take a break for just a
18 moment?

19 THE VIDEOGRAPHER: The time
20 right now is 10:57 a.m.

21 We're off the record.

22 (Recess taken.)

23 THE VIDEOGRAPHER: The time
24 right now is 10:57 a.m.

25 We're back on the record.

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BY MS. McKEEVER:

Q. If you could look at Exhibit 7, please, which is the ATSDR Assessment of Evidence. And on page 95, that's where the ATSDR says there is below equipoise causation for TCE and bladder cancer and below equipoise evidence for vinyl chloride and benzene and bladder cancer, correct?

A. Yes.

Q. And I'm going to refer you to pages 6 and 7 of the ATSDR. And I'm looking at the classification scheme categories, and I'm looking at the bottom of page 6, continuing on to page 7 on the classification scheme for equipoise and above evidence for causation.

And it says: The evidence is sufficient to conclude that a causal relationship is at least as likely as not, but not sufficient to conclude that a causal relationship exists. This category would be met, for example, if:

1. The degree of evidence from

1
2 human studies is less than sufficient but
3 there is summary evidence from animal
4 studies and/or mechanistic studies that
5 supports causality, or,

6 2. A meta-analysis does not
7 provide convincing evidence (e.g., the
8 summary risk estimate is close to the null
9 value of 1.0, i.e. less than 1.1), or if
10 the meta-analysis observes a non-monotonic
11 exposure-response relationship) but there
12 is at least one epidemiological study
13 considered to be of high utility occurring
14 after the meta-analysis has been
15 conducted, in which an association between
16 the exposure and increased risk of the
17 disease of interest has been found and in
18 which chance and biases can be ruled out
19 with reasonable confidence.

20 And approximate. A
21 meta-analysis has not been conducted, but
22 there is at least one epidemiological
23 study considered to be of high utility in
24 which an association between the exposure
25 and increased risk of the disease of

1
2 interest has been found and in which
3 chance and biases can be ruled out with
4 reasonable confidence.

5 Do you see what I'm reading?
6 Did I read that correctly?

7 A. Yes.

8 Q. If you relied on the ATSDR
9 Assessment of Evidence to form your
10 opinions and the ATSDR found below
11 equipoise for TCE and bladder cancer, how
12 are you able to opine that TCE more likely
13 than not causes bladder cancer?

14 MS. GREENWALD: Objection; form.

15 A. Several studies have come out
16 since the 2017 ATSDR assessment.

17 Q. And what are those studies?

18 A. Well, there are several studies
19 in that table that talk about that have
20 increase risk or odds ratios for TCE and a
21 couple for benzene.

22 Q. And you are referring to the
23 table beginning on page 11 of your bladder
24 cancer report?

25 A. Consider.

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Q. Epidemiological Studies of Contaminant Exposure and Bladder Cancer.

And which studies are you referring to?

A. Well, there's as you can see in column 3, there are studies that looked at TCE, and there are a couple that looked at benzene?

Q. And can you be more specific?

A. You want me to name all the ones that --

Q. I -- you said that you -- more recent studies came out since the 2017 Assessment of Evidence, and I'm asking which ones those are.

A. I don't remember the dates that these are published.

Q. Well, for example, the first one Anttila looks at TCE, but when I check your reference 36, that study was July 1995, right?

A. Okay.

Q. So I'm asking which ones are you relying on that came after the ATSDR

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Assessment of Evidence.

MS. GREENWALD: Objection; asked
and answered.

A. I would have to go back and look
through all of these and figure out which
ones specifically. But as I recall, even
though it's not on here, I think the
incidence, the Bove incidence report from
2024 had increased risk for bladder
cancer.

Q. Okay. We will discuss the Bove
reports as well.

And then are you able to
identify which studies you were referring
to on benzene that came out since the
ATSDR Assessment of Evidence in 2017?

A. I would have to go through this
and figure that out.

Q. Okay. Moving on then.

MS. McKEEVER: I am handing you
the next exhibit, and it is a printout
from the IARC website, that's capital
I-A-R-C, International Agency For
Research on Cancer. And it says:

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Agents Classified by the IARC
Monographs, Volumes 1 through 137.

(Gilbert Exhibit 8, Agents
Classified by the IARC Monographs,
Volumes 1-137, was marked for
identification, as of this date.)

MS. GJONAJ: Exhibit 8?

MS. KONSTANTOPOULOS: 8.

BY MS. McKEEVER:

Q. You are familiar with IARC,
correct?

A. Reasonably.

Q. You cited at least one IARC
source in some of your reports, I believe.
Is that right?

A. Yes.

Q. I just wanted you Exhibit 8, and
have you seen this document before?

A. I don't know that I've reviewed
this particular document.

Q. Did you review the report of
Peter Shields?

A. No.

Q. If you look at page 9 of this

1
2 exhibit it lists a cancer site and
3 carcinogenic agents with sufficient in
4 humans and agents with limited evidence in
5 humans.

6 And I'm looking at urinary
7 bladder cancer, and you can see that TCE
8 is not listed among the agents with
9 sufficient evidence in humans for bladder
10 cancer, right?

11 MS. GREENWALD: Objection; form.

12 A. I can see that it's not on this
13 table, yes.

14 Q. And TCE is also not listed among
15 the agents with limited evidence in humans
16 for bladder cancer, right?

17 MS. GREENWALD: Objection; form.

18 A. According to this table, yes.

19 Q. And benzene is not listed among
20 the agents with sufficient evidence in
21 humans for bladder cancer?

22 MS. GREENWALD: Objection; form.

23 A. I don't really -- I haven't
24 looked at this particular document. I
25 don't know when it was published.

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I -- I would really need to know more to decide how useful this is.

Q. Right. But I'm just asking whether you see benzene on the list.

A. I do not see benzene on the list.

Q. And benzene is not listed among the agents with limited evidence for humans and bladder cancer, right?

MS. GREENWALD: Objection; form.

A. In this document, yes.

Q. Okay. Let's move on to some of the Bove studies you mentioned.

MS. McKEEVER: Can I have Cancer Incidence?

(Pause.)

MS. McKEEVER: All right.

I am marking Cancer Incidence among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water at US Marine Corps Base Camp Lejeune: A Cohort Study, October 2024.

(Gilbert Exhibit 9, Cancer

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incidence among Marines and Navy personnel and civilian workers exposed to industrial solvents in drinking water at US Marine Corps Base Camp Lejeune: a cohort study, Bove 2024, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. You've reviewed the ATSDR's epidemiological studies of the Camp Lejeune population, right?

A. Which document are you referring to?

Q. Well, you cited to a number of them in your reports. You cited to this one, the 2024 cancer incidence study.

A. So you're -- you're calling this an ATSDR study.

Q. Moving forward I will refer to it at the Bove cancer incidence study 2024 because we'll be discussing a lot of reports and it will get confusing.

You have reviewed this document before?

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A. Yes.

Q. And would you agree that those studies, this study, examined the potential effects of the Thomas in the Camp Lejeune water in persons exposed to water at Camp Lejeune?

A. Yes.

Q. On page 27 of your bladder cancer report at the bottom of the page you say: For example, Bove et al. noted that increased incidence of bladder cancer at Camp Lejeune was ex -- was associated with exposures of 7 to 10 quarters for Marines and greater than 21 quarters for civilians.

Correct?

A. That's what it says, yes.

Q. Were any of these findings statistically significant?

A. I don't remember.

Q. I think if you look at tables 5 and 6.

MS. GREENWALD: I just want to state an objection, the supplements

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aren't included here, so.

MS. McKEEVER: Okay.

BY MS. McKEEVER:

Q. If you look at table 5 which is Cancer outcomes by duration stationed at Camp Lejeune compared with Camp Pendleton between 1975 and 1985, Marines Navy personnel subgroup.

And we're looking at the bladder cancer results. Were any of those findings statistically significant?

A. I have to find where the bladder cancer is on this table.

They don't actually specify statistical significance here. So we would have to use the confidence intervals.

Q. Right.

A. And the confidence intervals are -- the lower confidence interval has -- supposedly has to be of a 1, and both of those are very close to 1.

Q. But is it statistically significant?

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A. Well, as I said, they don't actually spell out whether they're statistically significant. So you would have to look at the results and compare and look at the confidence intervals Andrew your own conclusions as to how important that data is.

Q. And if you look at table 6 which is Cancer Outcomes By Duration Employed At Camp Lejeune Compared With Camp Pendleton, October 1972 to December 1975 Among Civilian Workers, bladder cancer, urinary bladder is about -- it's in the top half of the page.

A. Right.

Q. And the high duration HR for Camp Lejeune is listed as 1.09.

Right?

A. Yes.

Q. And is that result statistically significant?

A. Once again, they didn't calculate that, but based on the odds ratios -- I mean, confidence intervals, it

1
2 seems unlikely.

3 Q. And are these results linear
4 from looking at the low to medium duration
5 to the high duration?

6 A. There's only two values. So
7 talking about linear is very difficult.

8 Q. But you would agree that the
9 low-medium duration shows a higher hazard
10 ratio than the hazard ratio for the
11 high duration, correct?

12 A. They're -- the numbers are very
13 similar, and you don't have that many
14 cases. So I don't know that you could
15 draw any specific conclusions about
16 that -- the linearity of that response.

17 MS. McKEEVER: Okay.

18 Let's look at a different Bove
19 study. This one is 2024 mortality.

20 All right. I am handing you
21 Evaluation of mortality among Marines,
22 Navy personnel, and civilian workers
23 exposed to contaminated drinking water
24 at USMC base Camp Lejeune: a cohort
25 study, 2024.

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(Gilbert Exhibit 10, Evaluation of mortality among Marines, Navy personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study, Bove 2024, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. And I will try to refer to this as the Bove 2024 mortality study as opposed to the cancer incidence study.

Have you seen this document before?

A. Yes.

Q. And you have listed this document in some of your reports, but not in the bladder cancer report.

Is that right?

A. I guess so.

Q. I do not see it in table 1 of your bladder cancer report.

A. Okay.

Q. Would you disagree with that?

A. No.

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Q. All right.

If you look at table 3 which is the Standardized mortality ratios (SMR) Poisson regression risk ratios, and the 95 percent confidence intervals (CI) for Camp Lejeune and Camp Pendleton civilian employees: underlying cause of death.

So, for urinary bladder cancer, which is in the top half, the risk ratio for Camp Lejeune versus Camp Pendleton is .65, right?

A. Yes.

Q. So this means that there were fewer deaths at Camp Lejeune due to bladder cancer among civilian employees, right?

A. Yes.

MS. McKEEVER: Okay.

Now let's look at another Bove study, 2014 civilian.

Now I'm looking at the Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective

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cohort study. And this is dated 2014.
(Gilbert Exhibit 11, Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study, Bove 2014 Bates CLJA_HEALTHEFFECTS-0000291324-336, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. I will try to refer to this as the 2014 Bove civilian study.

(Pause.)

MS. McKEEVER: May I see that exhibit back, please, just to make sure you have the right one?

THE WITNESS: (Handing.)

MS. McKEEVER: Yeah, civilian.

BY MS. McKEEVER:

Q. You've seen this study before, correct?

A. Yes.

Q. If you look at table 3, table 3 Standardized Mortality Ratios (SMRs)

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underlying cause of death for bladder cancer, you'll see that the SMR is .53.

Is that right?

A. You also see that the number of cases is very low, so it's hard to draw too much inference from that stick value.

Q. Okay.

A. But that is what it says.

Q. All right.

And on the table 4 on the next page, Camp Lejeune versus Camp Pendleton Hazard Ratios and 95 Percent Confidence Intervals Adjusted By Sex, Race, Occupation (blue collar versus white collar), and Education 10-year lag, the hazard ratio is .65.

Is that right?

A. To really understand what that means, though, we have to acknowledge that -- that the Marines especially in this study were very young and bladder cancer is most often found in people over 65, and almost nobody in the study was of that age.

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Q. But this is comparing two sets of Marines, correct?

A. Correct.

Q. And the hazard ratio comparing Camp Lejeune to Camp Pendleton was .65 in this study?

A. Yes.

MS. McKEEVER: Let's look at one more study, the Bove 2014 Marines.

Will this be 12?

THE STENOGRAPHER: Yes.

MS. McKEEVER: Okay. I'm going to hand you one more study: Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study, 2014.

(Gilbert Exhibit 12, Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study, Bove 2014, Bates

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CLJA_HEALTHEFFECTS-0000141103-116, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. You've seen this study before?

A. Yes.

Q. And if you look at table 4 standardized mortality ratios (SMRs) underlying cause of death for bladder cancer, you'll see comparing Camp Pendleton to Camp Lejeune that the SMR is .84, right?

A. I misspoke a minute ago. I was thinking we were talking about the Marines as opposed to the civilians, even though the civilians were also relatively young. But once again, the age of the -- of the Marines in this case would make it fairly unlikely that you would see any kind of bladder cancer mortality.

So this number is not un -- is not surprising.

Q. And in both studies, the 2014 Marine study, were comparing Marines at

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Camp Pendleton to Marines at Camp Lejeune, right?

A. Yes.

Q. And in the civilian study we are comparing civilian employees at Camp Pendleton to civilian employees at Camp Lejeune, correct?

A. Yes.

Q. And if you look at table 5 Camp Lejeune versus Camp Pendleton hazard ratios and 95 percent confidence intervals adjusted by sex, rank, and education 10-year lag, you'll see that the hazard ratio comparing Camp Lejeune to Camp Pendleton is .76.

Is that right?

A. Yes. Once again, you would -- it would be very surprising to see many -- any kind of camp -- bladder cancer deaths at this point in their lives.

Q. This study isn't listed on - I'm going to find the page number - in your bladder cancer report page 11 and 12 the epidemiological studies of Camp Pendleton

1
2 exposure to bladder cancer, right? The
3 25240 Marine study --

4 MS. McKEEVER: I'm sorry,
5 strike.

6 Q. The 2014 Marine study and the
7 2014 civilian study, neither one of those
8 are listed on your table in pages 11 and
9 12.

10 A. Well, there is the Bove with the
11 reference 40.

12 Q. I believe that will be a
13 reference to the 2024 cancer incidence
14 study, if you'd like to check it.

15 (Pause.)

16 A. Yes.

17 Q. And so, the 2014 civilian study
18 and the 2014 Marine study are not listed
19 on your table on pages 11 and 12 the
20 bladder cancer study.

21 A. That's correct.

22 Q. And you have listed those
23 studies in other reports, in your kidney
24 cancer report for example.

25 A. Yes.

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Q. Why weren't they included in the bladder cancer report?

A. Because this table was a -- a -- was designed to show the -- the studies that provided evidence that, or that -- that supported my opinions as far as the bladder cancer, and the problem with the bladder cancer, there's not that many of them and they don't usually occur in civilians or Marines that were that young at the time of the 2014 studies. So it didn't make sense to put them in the table.

Q. In your kidney cancer report on page 14 table 1 epidemiological evidence that TCE and other VOC cause kidney cancer, you do include these studies?

A. Okay.

Q. The Bove Marines 2014, the Bove civilians 2014.

Do people develop kidney cancer at a much older age?

A. It has -- okay.

Kidney cancer is also something

1
2 that is developed later in age -- later in
3 life, but I believe that the incidence may
4 be higher so that you would -- it's not
5 surprising that you would see more of an
6 effect in those earlier studies.

7 Q. So why were these two studies
8 included in your kidney cancer report but
9 not in the bladder cancer report?

10 MS. GREENWALD: Objection; form;
11 asked and answered.

12 A. I think I just answered that.

13 Q. I don't think I understand the
14 answer. I --

15 A. I can't explain why for sure
16 you -- they detected an increased
17 incidence of kidney cancer and not bladder
18 cancer, but the findings that they didn't
19 find it in bladder cancer are not unusual
20 and unexpected considering several
21 parameters. And since this table was, as
22 I explain in -- in the introduction to the
23 table, was to show the association between
24 bladder cancer and TCE, they didn't get
25 included.

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Q. Because the odds ratio is not above 1.1 for the bladder cancer?

A. Correct.

Q. And it is above 1.1 for these studies and the kidney cancer?

A. Correct.

Q. Okay. I understand.

I'm looking again at page 11 of your bladder cancer report. There is a column for Confounder Adjustment.

If that is blank, does that mean there was no confounder adjustment for smoking?

A. It means it -- that the -- whatever was done with confounding -- either they didn't do it or it wasn't sufficient.

Q. And that would be true every time that column -- every time that space is blank?

A. Yes.

Q. On page 13 you agree with your statement that: There are no animal studies showing that TCE can cause bladder

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cancer.

Right?

MS. GREENWALD: Objection; form.

A. The studies haven't been done.

Q. Right.

Your statement is: Although there are numerous animal studies showing that TCE causes cancer in the closely associated kidney, the ability of TCE or other contaminants to cause bladder cancer specifically has not been tested.

Is that correct?

A. That's what I -- that's what it says, yes.

Q. Are there human studies showing that TCE can cause bladder cancer?

A. Well, we just talked about one of them. We talked about the Bove, and there's some other ones listed in here in the table that suggest that TCE does cause bladder cancer.

Q. And in your opinion, the Bove studies show that TCE causes bladder cancer?

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A. The incidence report.

Q. The cancer -- the Bove 2024 cancer incidence report shows that TCE can cause bladder cancer?

A. Shows an increased risk odds ratio.

Q. And is an increased risk the same as cause?

A. Causation takes many different things into effect, but that is one of the components is to show that you see an epidemiological association.

Q. And just to be clear, on page 11 you reference in your table the Bove cancer incidence report 2024 and the numbers you listed are an odds ratio of 1.2 for Marines and 1.18 for civilians.

Is that right?

A. Yes.

Q. And that's what you're relying on?

A. When you say that's what I'm relying on, as far as the Bove studies go, or?

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Q. Well, I asked whether there were human studies that showing -- that show TCE can cause bladder cancer, and you referenced this study.

A. Well, yes, but there are other ones on this table as well.

Q. Okay. Which others?

A. Anttila, Blair, Hansen, Morgan, Pesch, I don't know how you pronounce her name, Raaschou Nielsen. Several of them.

Q. And it's your opinion that these studies show an increased risk. Is that right?

MS. GREENWALD: Objection; form.

A. They show an increased odds ratio, yes.

Q. Do they show cause?

A. They were not designed to show cause. They were designed specifically as epi studies.

Q. Okay.

Going back to the statement on page 13 at the top of the page: Although there are numerous animal studies showing

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that TCE causes cancer in the closely associated kidney.

What do you mean by "closely associated"?

A. Anatomically.

Q. Does that mean that the urothelial cells of the bladder are closely associated with renal cells?

A. What do you mean "closely associated with"?

They're certainly in proximity. They're, like I said, anatomically.

Q. So by "anatomically" you mean in close proximity to one another?

A. Yes.

Q. And that's what you mean by "closely associated"?

A. Yes.

Q. Okay.

And on page 15 at the bottom of the page you acknowledged that the ability of TCE, PCE and benzene to induce genotoxicity to cause bladder cancer has not been studied, right?

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A. Could you show me where on that page it shows that?

Q. It's at the very bottom of the page in the last paragraph: Although the role of TCE and PCE and benzene-induced toxicity in causing bladder cancer specifically has not been studied, the toxicity of the TCE has been documented in the physically proximal kidney where these chemicals get metabolized.

Right?

A. That's what it says, yes.

Q. And are you saying that TCE, but not PCE or benzene toxicity has been demonstrated in the kidney, and therefore some TCE metabolites can travel from the kidney to the bladder where genotoxic events occur?

MS. GREENWALD: Objection; form.

A. That's not what it says. It just says that genotoxicity of the TCE has been documented in the kidney.

Q. Right, but it also says: Although the role of TCE and PCE and

1
2 benzene-induced genotoxicity in causing
3 bladder cancer specifically has not been
4 studied.

5 And that's where I'm trying to
6 figure out how you're reaching your
7 conclusion.

8 A. The conclusion is that TCE
9 has -- genotoxicity has been shown in the
10 kidney. That's the --

11 Q. But what about for PCE?

12 A. This partly has to do with the
13 supplemental material that we sent you.

14 Q. That was sent to me last night?

15 A. No. That was sent to you, I
16 don't know when exactly it was sent to
17 you.

18 Q. Is it listed in any of these
19 supplemental materials?

20 A. I'm sure it's in -- what -- was
21 that 6?

22 (Pause.)

23 Q. Exhibit 6.

24 A. Yes.

25 Q. And which page of Exhibit 6 are

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you referring to?

A. The first page.

Q. And what is the title of the first page?

A. Dr. Gilbert Additional Materials Considered Bladder Cancer.

Q. Okay.

And what are the two studies that are -- or the studies that are listed?

A. Because it's just one study. The Ge and Kramer and Pereira study.

Q. And this is what you're referring to when you say "additional materials"?

A. Yes.

Q. Okay.

But this study is dated 2001, right?

A. Yes.

Q. Looking at page 11 again in your bladder cancer report, do you know what a confidence interval ratio is?

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A. I know that odds ratios are used to develop confidence intervals.

Q. Do you know how to calculate a confidence interval ratio?

A. What do you mean by confidence interval ratio?

Q. It is referenced by Dr. Bove and I'm asking if you're familiar with it, and if you are, then I will ask you some questions about it, but if you're not familiar with it, then I won't.

MS. GREENWALD: Objection; form.

A. I'm just a little confused by the terminology.

Like I said, I know what confidence intervals are and they are used based on odds ratios. I don't know what you mean specifically by calling it a confidence interval ratio.

Is that the same thing?

Q. No. A confidence interval ratio is when you take the high end of the confidence interval and you divide it by the low end and you get a confidence

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interval ratio.

A. Okay.

Q. Is that something you have ever used in your research?

A. No.

Q. All right.

Okay. Let's take a look at Exhibit 4, your leukemia -- well, let me just double check.

Yes, Exhibit 4.

Okay. Going back to Exhibit 4, did you prepare all the opinions in this report?

A. Yes.

Q. Is there anything in this report that you believe is incorrect?

A. No.

Q. Other than the supplemental materials that are contained in Exhibit 6 and the one that we received last night that I haven't reviewed, is there anything else in the report that needs to be updated?

MS. GREENWALD: Objection; form.

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A. That's kind of a open-ended question.

As -- as far as I know, the answer is no. I mean, I don't know for sure if some study has come out extremely recently that I haven't seen.

Q. Okay.

And on page 4, second paragraph you say: My report will focus on TCE since that is the contaminant that has been the nexus of my own research.

Right?

A. Mm-hm, yes.

Q. And you have not offered any opinion in this report that PCE causes NHL?

A. I can't remember if I have a section at the end where we were talking about co-exposures. I don't remember. I'd have to look.

(Witness reads document.)

Yes, so I do talk about other contaminants.

Q. And you're talking about that in

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the context of your opinion 5?

A. Yes.

Q. You haven't offered an opinion in your report that PCE alone causes NHL?

A. I don't be -- there is some evidence in the epidemiological table showing that PCE has been linked to NHL, but I don't believe I had any specific opinions in regard to that.

Q. Opinion 1 on page 12 is: TCE more likely than not causes NHL and leukemia.

Right?

A. Yes.

Q. And you haven't offered an opinion in this report that PCE causes leukemia?

MS. GREENWALD: Objection; asked and answered.

MS. McKEEVER: I asked about NHL. This is leukemia.

A. Well, the same answer applies. The only part of this report that addresses that is the opinion 5 where

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we're talking about co-exposures.

Q. Okay. And you haven't offered an opinion that PCE alone causes leukemia?

A. Not in this report, no.

Q. And you haven't offered an opinion in this report that benzene alone causes NHL?

A. I have no opinions on that in this report.

Q. And when I say "NHL" I'm referring to non-Hodgkin lymphoma.

A. Okay.

Q. And you haven't offered an opinion in this report that benzene alone causes leukemia, correct?

MS. GREENWALD: Objection; form.

A. The answer's always the same. The only part of this report that deals with those two -- with those chemicals are in that cumulative exposure part in opinion 5.

Q. And you haven't offered an opinion in this report that vinyl chloride causes NHL, correct?

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A. That's correct.

Q. And you haven't offered an opinion in this report that vinyl chloride alone causes leukemia?

A. Correct.

Q. Would you agree that lymphomas and leukemias are a vast collection of diseases that have different risk factors?

MS. GREENWALD: Objection; form.

A. I think "vast" is overstating it, and I think they share some of the same risk factors. There are some differences.

Q. On page 11 you've noted that: NHL consists of a group of over 60 specific types of blood cancer.

Right?

A. Where are you reading that?

Q. Page 11.

A. Okay. Yes.

Q. Non-Hodgkin lymphoma consists of a group of over 60 specific types of blood cancer.

Correct?

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A. Correct.

Q. And then also on page 4 in the next section you have noted the four main subtypes of leukemia as acute lymphoblastic, acute myelogenous, chronic lymphocytic and chronic myelogenous, correct?

A. Correct.

Q. But other than noting that, your report doesn't break down the other subtypes of leukemia, correct?

A. Correct.

Q. And it doesn't break down the different subtypes of NHL?

A. Correct.

I think as far as the NH -- the lym -- the NHL, there may be 60, but I think there's two that are -- consist of the -- most of the cases. So it's a little bit incorrect to think of them as -- as needing to specify the effects on all 60 of these particular cancers, some of which I'm sure are extraordinarily rare.

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Q. When you say "two subtypes" and "most cases," are you referring to most cases of NHL generally or most cases of NHL at Camp Lejeune?

A. Most cases of NHL generally.

Q. Do you have a medical degree?

A. I do not.

Q. Okay. I'm going back to Exhibit 8. This is the IARC exhibit. And on page 11 leukemia is listed, and you can see that TCE is not listed among the agents with sufficient evidence in humans for any of the leukemia subtypes listed here, right?

MS. GREENWALD: Objection; form.

A. Once again, this is really hard to interpret out of context, and I -- I don't even know when this was published.

I can attest that that's what it says on the table, but I don't know the significance of that without more context.

Q. Okay.

A. I also note that it shows benzene in two of those.

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Q. Right. And you did not offer an opinion on benzene alone, correct?

A. Correct.

Q. Okay. Let's move to, I'm going to have to try to keep up with the exhibit numbers, the Bove 2014 Marine study?

MS. McKEEVER: Which was exhibit.

MS. KONSTANTOPOULOS: 12.

Q. 12.

On table -- I'm sorry, on page 14 of your NHL/leukemia report.

A. Okay.

Q. And your table of epidemiological studies shows that TCE and other chemicals cause NHL, and you have reference 36 Bove Marines, which I will rep to you is the 2014 study.

Where do you get these numbers for the odds ratio, risk ratio?

A. I don't remember. And I also do not see the data from the supplemental tables. Some of the information came from the supplemental tables.

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Q. I think if you look at this study on the first page you'll see that the 1.97 number for TCE is for Hodgkin lymphoma, correct?

MS. GREENWALD: Objection; form.

A. I'm sorry, where -- where are you reading this?

Q. I am on the first page of Exhibit 12 where it says non-hodg -- it says Hodgkin lymphoma and trichloroethylene and the HR is 1.97. And if you look at the number you listed for TCE in your report that says 1.97.

A. Well, it is possible that I made a mistake and put that -- the Hodgkin lymphoma in -- in non-Hodgkin lymphoma table.

Q. I think if you look at table 7 of Exhibit 12 for Hodgkin lymphoma you will see that the numbers for TCE, VC, vinyl chloride, benzene, and TVOC correspond with the numbers you listed in your table on page 14.

A. Okay. I must have made a

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mistake and put it in the wrong table.

Q. And then on page 9 of your report.

MS. McKEEVER: No, I'm sorry.

Strike that.

Q. If you listed the wrong data here, does that change your opinion?

MS. GREENWALD: Objection; form.

A. There is plenty of data showing that TCE exposure causes NHL, even if you left out the -- my incorrect numbers from this particular study.

Q. If you look at table 5 of Exhibit 12, Camp Lejeune versus Camp Pendleton hazard ratios and 95 percent confidence intervals, adjusted by sex, race, rank and education, 10-year lag, you'll see that the hazard ratio for NHL is .81, correct?

A. Yes. I'm just trying to see where it has the double asterisks by it.

Q. I believe it is because they are representing that NHL stands for non-Hodgkin lymphoma.

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A. Okay.

Once again, we have the same issue, that especially the -- the Marines at this point to have a lot of mortality information to use would be very unusual, and so it's not that unusual that you don't see it at this point.

Q. Based on what you said earlier about not using studies with risk ratios of less than 1.1, you would not include this study, Exhibit 12, on your list for NHL, correct?

A. It would depend. Like I said, I -- sometimes there -- there's information in the supplemental tables that show that there is an increase within you're looking -- within the cohort at camp -- for example, Camp Lejeune where you would look at unexposed to medium exposed or high exposed. But I don't remember specifically on this -- on this one.

But yes. Otherwise, I would not have included it if there was just that

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one number to go by.

Q. Okay. Let's move to the Bove civil study 2014 which is Exhibit 11.

If you look at table 3 Standardized Mortality Risk Ratios (SMRs) underlying cause of death for NHL, you'll see that the SMR is .6, correct?

A. That's what it says, yes.

Q. So there were fewer deaths at Camp Lejeune versus Camp Pendleton, that's what that means, right?

A. There were very few deaths at either place.

Q. And the Bove civilians 2014 study is not listed on your table for NHL in your report.

A. Okay.

Q. Is that correct?

A. I don't remember --

Q. You can check.

A. -- but I believe -- I'll trust you on that.

Q. And on the table 4 of Exhibit 11, Camp Lejeune versus Camp

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Pendleton hazard ratios, you'll see that the hazard ratio for NHL is .83, right?

A. Yes.

Q. Okay. Let's look at the 2024 Bove mortality study which is Exhibit 10.

And this study Exhibit 10 is not listed in your report for NHL, right?

A. I would have to go back and check, but I will -- yes, that's true.

Q. And if you look at table 2 of this report, this study Standardized Mortality Ratios (SMR) for Marines Underlying Cause of Death for NHL, you'll see that the Camp Lejeune versus Camp Pendleton, the risk ratio is .087, right?

A. That's what it says, but you have to put this in context again, that less -- I think it was, like, 2 percent of the Marines even at this point in the study were over 65.

Q. But again we're comparing Marines to Marines, right?

A. Yes, but if none of them are old enough to get NHL, it doesn't really

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matter.

Q. And looking at table 3 of the same exhibit, you'll see that the risk ratio for Camp Lejeune versus Camp Pendleton for NHL is .98, right?

A. That's what it says, but as I -- as I just pointed out, it's very difficult to know what that really means in view of the age of the -- the subjects.

Q. And in table 4 of the same exhibit, looking at NHL, hazard ratios for the Marines and Navy Personnel Subgroup Camp Lejeune versus Camp Pendleton Underlying Cause of Death, again the HR is .87, right?

A. I am looking for where NHL is.

Q. It's in the lower half?

A. That's what it says, yes.

Q. And finally, table 5 Comparison of Camp Lejeune and Camp Pendleton Civilian Workers Underlying Cause of Death for NHL, the hazard ratio is .95, right?

A. The fact that you don't have an age population which is going to be likely

1
2 to have non-Hodgkin lymphoma, sort of
3 negates the -- the reasons to look at all
4 these different breakdown categories.

5 Q. You have not cited this study in
6 the NHL report, but you have cited to this
7 study in other reports, like for example
8 kidney cancer, correct?

9 A. Yes.

10 Q. Let's go back to the cancer
11 incidence study 2024, Exhibit 9. Thank
12 you.

13 Okay. On page 32 of your
14 NHL/leukemia report. Are you there?

15 A. Yes.

16 Q. I'm under Section C TCE Disease
17 Latency, you list the NHL odds ratio of
18 1.31 and the leukemia odds ratio of 1.15.

19 Right?

20 A. Yes.

21 Q. And if you look at the cancer
22 incidence study Exhibit 9, table 6. This
23 is the table for cancer outcomes for
24 civilians. And the 1.31 number that you
25 listed for NHL comes from the low/medium

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duration at Camp Lejeune.

Is that right?

A. I have to find NHL.

Yes.

Q. But the number at the high duration for NHL is 1.05, right?

A. Yes.

Q. And if you turn to table 5 which is Cancer Outcome by Duration at Camp Lejeune Compared With Camp Pendleton for Marines, you'll see that you used the adjusted hazard ratio for leukemia of 1.15 from the high duration category, right?

A. Hold on.

Yes.

Q. So why use the number, the hazard ratio in the high duration category for one disease and in the low to medium duration category for the other disease?

MS. GREENWALD: Objection; form.

A. As I explain when I'm discussing the tables, the odds ratio need to be over 1.1. So I use the values that were.

Q. Understood.

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Would you agree that you used the highest hazard value regardless of the level of duration?

A. I don't think I could make a statement on that without asking you a little bit more about that. What -- what do you mean?

Q. At least in this instance, would you agree that you used the highest hazard ratio regardless of duration?

A. I don't know if I would call it regardless of duration.

I used the value that showed the odds ratio of greater than 1.1.

Q. Okay. Let's go to the Bove mortality study of 2024, Exhibit 15. And I'm looking at table 3 of Exhibit 10 Standardized Mortality Ratios (SMR) for Camp Lejeune and Camp Pendleton Civilian Employees Underlying Cause of Death.

For leukemias, the risk ratio of Camp Lejeune versus Camp Pendleton is 1.0, correct?

A. In that table, yes.

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Q. Does that indicate a null result?

A. Yes.

Q. And on the table 5 Comparison of Camp Lejeune and Camp Pendleton Civilian Workers Cause of Death, the hazard ratio for leukemia is again 1.0, correct?

A. Well, as you can see, some of the individual leukemias have hazard ratios which are greater than 1.1.

Q. And CLL, chronic lymphocytic leukemia, has a hazard ratio of .85, correct?

A. That's what it says, yes.

Q. And AML, acute myeloid leukemia, has a hazard ratio of .97.

A. Yes.

Q. And CML has a hazard ratio of 1.26.

A. Yes.

Q. With a confidence interval of .29 to 5.48, right?

A. Yes.

Q. Let's go back to your report,

1
2 and again we're on the leukemia/NHL
3 Exhibit 4.

4 Page 14 for NHL, 14 and 15 and
5 16, excuse me, and then page 17 are the
6 epidemiological studies for leukemia, page
7 17 and 18.

8 If the confounder adjustment is
9 blank, does that mean that there was no
10 confounder adjustment for smoking?

11 A. For both of those tables, or are
12 you asking --

13 Q. Well, I can ask separately
14 for --

15 A. Don't. Please don't.

16 Q. I assume the answer would be the
17 same --

18 A. Yes.

19 Q. -- for both.

20 A. Yes. You're right. You're
21 correct.

22 Q. I am correct that there's no
23 confounder adjustment for smoking if -- if
24 the -- if the table is blank?

25 A. Not that I could find.

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Q. If you look on page 22 of your NHL/leukemia report under the section of Leukemia, you have acknowledged that TCE exposure in leukemia has not been study as -- as rigorously.

Is that right?

A. Yes.

Q. And at the bottom of that paragraph, certainly there was enough data to convince the ATSDR that there is equipoise and above evidence for causation for all types of leukemia. There are also some admittedly limited animal and mechanistic data to support the equipoise designation.

Right?

A. That's what it says.

Q. And are you relying on the ATSDR's finding of equipoise to support your opinion?

MS. GREENWALD: Objection; form.

A. I took it into account, but I don't know that I -- I can't remember if I used it specifically.

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MS. McKEEVER: Before we move on to kidney cancer, do you all want to break for lunch?

THE WITNESS: I'm hungry.

THE VIDEOGRAPHER: The time right now is 12:16 p.m.

We're off the record.

(Luncheon recess taken.)

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A F T E R N O O N S E S S I O N

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THE VIDEOGRAPHER: The time right now is 1:37 p.m.

We're back on the record.

MS. McKEEVER: Welcome back from lunch.

BY MS. McKEEVER:

Q. When we left off, we had gone through your -- excuse me. We had gone through your bladder cancer report and your NHL/leukemia report, and now I'm going to ask you a few questions about your kidney cancer report.

A. Okay.

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Q. Which is Exhibit 5.

Did you prepare all the opinions in this report?

A. Yes.

Q. And is there anything in the report that you believe is incorrect?

A. No.

Q. And your opinion 1 says: TCE causes kidney cancer.

Right?

A. What page is that on?

Q. Looks like 11.

A. Yes.

Q. You have not offered an opinion in your report that PCE alone causes kidney cancer.

A. I think I talk about it as perhaps potentiating things in the co-exposure.

Q. My question is you haven't offered an opinion about whether PCE alone causes kidney cancer.

A. I -- let me just check all the --

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Q. Sure.

A. -- opinions. It's been a while since I...

(Witness reads document.)

Yes, that's true.

Q. Yes, you have not offered an opinion that PCE alone causes kidney cancer?

A. Yes.

Q. And you have not offered any opinion in your report that benzene alone causes kidney cancer?

A. Yes.

Q. And you have not offered any opinion in your report that vinyl chloride alone causes kidney cancer?

A. Yes.

Q. So, opinion 1 is phrased as: TCE causes kidney cancer.

And that's different from your bladder cancer and NHL/leukemia reports where you phrased it as "more likely than not." Right?

A. They --

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MS. GREENWALD: Objection; form.
I'm sorry, go ahead.

A. So, what your question is that I -- I verbalized it differently in one versus the other?

Q. Yes. In opinion 1 in the kidney cancer report you say: TCE causes kidney cancer.

Right?

A. Mm-hm, yes.

Q. And then in the NHL/leukemia report, you say in opinion 1: TCE more likely than not causes NHL and leukemia.

Right?

A. Okay.

Q. And then in the bladder cancer report, I believe it says the same language as in the NHL report. It says: The contaminants in the drinking water at Camp Lejeune more likely than not causes bladder cancer.

So, I'm asking is your opinion that there is stronger evidence that TCE causes kidney cancer or bladder cancer or

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NHL and leukemia?

MS. GREENWALD: Objection; form.

A. There's more extensive evidence. There has been more work on it. So that's the difference.

Q. Look at page 11 in the kidney cancer report.

I'm looking at the first full paragraph towards the end where it says: However, these risk factors account for much less than 50 percent of causes. Thus making it clear that other environmental factors such as toxic and exposure, must play a role in tumorigenesis.

A. I'm sorry, where are you reading from?

Q. Page 11.

A. Yes.

Q. First full paragraph towards the bottom.

A. Okay.

Q. Last full sentence.

A. Okay.

Q. (Reading) However, these risk

1
2 factors account for much less than 50
3 percent of cases. Thus making it clear
4 that other environmental factors, such as
5 toxic and exposure, must play a role in
6 tumorigenesis.

7 Did I read that correctly?

8 A. Yes.

9 Q. And you were identifying
10 smoking, obesity, and hypertension as the
11 risk factors?

12 A. Yes.

13 Q. How much could genetics play a
14 role in kidney cancer?

15 A. I -- didn't I address that
16 somewhere?

17 I don't remember.

18 Q. Would you agree that genetics
19 play a role in kidney cancer?

20 A. I'd have to look and find what I
21 said about it.

22 MS. GREENWALD: Objection; form.

23 A. It says right here: Less than
24 10 percent of kidney cancer cases appear
25 to involve a genetic predisposition.

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Q. What about age, does age play a role in kidney cancer?

A. Yes.

Q. And what about just bad luck, random mutations that arise during DNA replication in normal non-cancerous stem cells?

MS. GREENWALD: Objection; form.

A. That one's really hard to answer because what exactly is bad luck. There's still so many components to that even though you may have a random mutation, even if your immune system has been altered such that now it turns into a consider. It's not based purely on bad luck.

Q. Let me ask you page 14.

This is your epidemiological evidence that TCE and other VOC cause kidney cancer.

And I have the same question that I have asked about the other reports, that if the column for the confounder adjustments blank, does that mean that

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there was no confounder adjustment for smoking?

A. None that I could find.

Q. You're familiar with the National Research Council, right?

A. Sure, yes.

Q. And the National Academy of Sciences?

A. Yes.

Q. And you've done some work with the National Research Council?

A. Yes.

Q. Are you aware that the purpose of the National Academies is to produce and promote the adoption of independent, authoritative, trusted scientific advice for the benefit of society?

MS. GREENWALD: Objection; form.

A. I did not know that was their mission statement.

MS. McKEEVER: I'm sorry, we're going to have to take a short break because we left our exhibits in the other room.

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THE VIDEOGRAPHER: The time
right now is 1:45 p.m.
We're off the record.
(Recess taken.)

THE VIDEOGRAPHER: The time
right now is 1:46 p.m.
We're back on the record.

MS. McKEEVER: Okay. Sorry
about that.

BY MS. McKEEVER:

Q. And you're familiar with the
epidemiologist David Savitz, right?

A. I was not familiar with him
until involvement in this particular case.

Q. Are you aware that he is an
expert for the plaintiffs?

A. I -- yes, I am aware that.

MS. McKEEVER: If I hand you
this it's going to be the exhibit. I
don't know that we want to make it the
entire, for your sake.

So, I'm going to mark the fewer
number of passages as the exhibit and
I'll give you the full document, if

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you want to review it. But for the court reporter's sake, I'm not going to mark the entire report.

So, I am marking an excerpt from the National Academies Science Engineering and Medicine. This is entitled "Contaminated Water Supplies At Camp Lejeune: Assessing Potential Health Affects 2009."

(Gilbert Exhibit 13, "Contaminated Water Supplies At Camp Lejeune: Assessing Potential Health Affects 2009, was marked for identification, as of this date.) Pellets.

MS. McKEEVER: What exhibit are we on?

THE STENOGRAPHER: 13.

BY MS. McKEEVER:

Q. Are you familiar with this document?

A. I don't think I am.

Q. It's listed in your materials considered for the NHL/leukemia report.

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A. Okay.

Q. Number 113, I believe.

A. Okay.

I certainly have not evaluated it extensively.

(Pause.)

MS. McKEEVER: I'll have to come back to that.

(Pause.)

MS. McKEEVER: I'm short on paper.

BY MS. McKEEVER:

Q. Let's see. On page 1 -- so, is it your recollection that you considered this or you didn't consider this?

A. I may have glanced at it. It certainly was not a big part of my decision-making.

Q. Okay.

On page 1 you can see the charge to the committee in the second paragraph says: The charge had several elements. One was to review the scientific evidence about the kinds of adverse health effects

1
2 that could occur after exposure to TCE,
3 PCE, and other contaminants.

4 The second was to: Evaluate
5 studies that were performed or that are
6 underway on former residents of the base
7 and to consider how useful it will be to
8 conduct additional studies.

9 The third element was to:
10 Identify scientific considerations that
11 could help the Navy set priorities on
12 future activities.

13 The responsibility of the
14 committee was to address its charge in a
15 dispassionate, expert, and unbiased way.
16 Analyses and findings were neither subject
17 to oversight, nor influenced by the agenda
18 of the entities with responsibilities for
19 Camp Lejeune, former or current residents
20 of Camp Lejeune, or any other entity.

21 Did I read that correctly?

22 A. I'm sorry, I'm still trying to
23 find where you're -- and you're not
24 talking about the preface, right?

25 Q. No, I'm talking about actual

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page 1.

A. Okay, so.

Q. There are a lot of paper, I apologize.

A. Okay.

Q. Do you see what I just read there?

A. Are you talking about the second paragraph?

Q. The second paragraph which begins with "The charge."

A. Just give me a moment.

Q. I'll give you a moment to read it.

A. Thank you.

(Witness reads document.)

Okay.

Q. And now we're back in the preface. If you see on page V that David Savitz is the chair of the committee.

A. Okay.

Q. Do you see that?

A. Yes.

Q. If you could turn to page 8,

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actual page 8 of the report.

A. Is that in here?

(Pause.)

Q. It's the box.

A. Okay.

Q. You see it?

A. I just didn't see a number.

Oh, there it is. It's under the staple, okay.

Q. I'm looking at page 8, box 2 of Exhibit 13.

You can see that the categorization of health outcomes reviewed in relation to -- are reviewed in relation to TCE, PCE, or solvent mixtures, right?

A. That's what it says, yes.

Q. And the categories are:
Sufficient evidence of a causal relationship; sufficient evidence of an association; limited suggestive evidence of an association; or inadequate/insufficient evidence to determine whether an association exists; limited or suggestive evidence of no

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association.

Correct?

A. Yes.

Q. Are you familiar with this categorization scheme?

A. With variations of it, yes.

Q. Do you see that the National Research Council committee considering the Camp Lejeune water contaminants with respect to TCE, PCE, and solvent mixtures did not list any diseases in the category of "Sufficient evidence of a causal relationship"?

MS. GREENWALD: Objection; form.

A. It's really hard to me -- for me to interpret this without having read the -- or really studied the -- that study.

Q. All right. And my question is you can see that under the category "Sufficient evidence of a causal relationship" it lists no outcomes, right?

A. That's what it says, yes.

Q. Is there any reason you wouldn't

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have considered this? It says contaminated water splice at Camp Lejeune assessing potential health effects.

MS. GREENWALD: Objection; form.

A. There's certainly no reason that -- that I saw it and rejected it. I don't remember being aware of this particular federal document.

Q. And you can see in box 2 that under the categorization "Sufficient evidence of an association" no outcomes is listed, correct?

A. That's what it says.

Q. And kidney cancer is listed in the "Limited suggestive evidence of association."

A. This -- this particular document is not in agreement with several of the other federal documents, but this particular one says that.

Q. Right.

And bladder cancer is listed in the "Limited suggestive evidence of association" with respect to PCE.

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A. In this particular study, yes.

Q. And adult leukemia is listed in the "Suggested evidence of an association" with respect to solvent mixtures, right?

A. That's what it says.

Q. And non-Hodgkin lymphoma is listed in the "inadequate/insufficient evidence to determine whether an association exists" in the last category.

A. Again, it's in -- it's totally different from other federal documents.

Q. But that's what it says here, correct?

A. That's what it says here.

Q. Okay.

A. So, this -- this report came out of public meetings held in 2007 and 2008.

Is that correct?

Q. That's what it says, yes.

A. So once again, there's a lot of data that's come out after that. So putting too much weight on this particular document does not seem warranted to me.

Q. And do you recall whether you

1
2 considered it or not?

3 A. I know that -- I think someone
4 sent it to me as something that should be
5 considered, and I don't know that I spent
6 that much time looking at it.

7 MS. McKEEVER: Okay.

8 I am showing you another
9 document now. This is the preprint of
10 the 2024 cancer incident study, and
11 it's called "Evaluation of cancer
12 incidence among Marines and Navy
13 personnel and civilian workers exposed
14 to contaminated drinking water at USMC
15 Base Camp Lejeune: a cohort study."
16 And it was used in Dr. Bove's
17 deposition. It has some Bates stamps
18 numbers at the bottom.

19 (Gilbert Exhibit 14, Evaluation
20 of cancer incidence among Marines and
21 Navy personnel and civilian workers
22 exposed to contaminated drinking water
23 at USMC Base Camp Lejeune: a cohort
24 study, Bove 2024, Bates
25 CLJC_ATSDR_BOVE-0000060101-164, was

1
2 marked for identification, as of this
3 date.)

4 MS. McKEEVER: Is this 14?

5 MS. KONSTANTOPOULOS: Yes.

6 BY MS. McKEEVER:

7 Q. Okay. We discussed several of
8 the Bove studies of the Camp Lejeune
9 public -- population earlier, and I'd like
10 to -- have you seen this study before, the
11 preprint version?

12 A. Preprint version, is this --
13 this isn't the 2018 --

14 Q. No.

15 A. -- screening?

16 Q. It's the preprint version of the
17 2024 cancer incident study.

18 A. I have not seen the preprint
19 version of it.

20 Q. Okay.

21 I'm going to direct you to table
22 2, and it is towards the very end of the
23 report.

24 Find it?

25 A. Yes. Table 2 I see.

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Q. Okay. Table 2 is called:
Standardized incident rates and Poisson
aggression results Marines/Navy personnel
subgroup.

And this compares the standard
incidence rates of disease in the Camp
Lejeune and Camp Pendleton population
adjusted for sex, race and age in
five-year increments. Would you agree?

A. Just a second.

(Witness reads document.)

Where are you seeing the part
about the --

Q. The next page.

A. Okay.

Q. Would you agree?

A. Yes.

Q. And let's look at the diseases
that you've offered general causation
opinions on.

For urinary bladder cancer, the
standard incidence rates of disease in the
Camp Lejeune --

MS. McKEEVER: I'm sorry, strike

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that.

Q. For urinary bladder cancer the standard incident rate for the Camp Lejeune cohort is .90.

Do you see that?

A. I don't understand why you would look at a preprint versus the actual published peer-reviewed document.

So, the significance of that number doesn't mean a whole lot to me.

Q. Would you agree that it shows that the -- there are 10 percent fewer bladder cancers in the Camp Lejeune cohort than in the general population?

A. Since a preprint implies that there's -- there's further evaluation and further work on the report before it goes into final publication, talking about numbers in a early version is -- is somewhat unwarranted.

I can say that that's what the number says on that page. I don't know the significance of that.

Q. And for kidney and renal pelvis

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cancer, it shows the standard incidence rate for the Camp Lejeune cohort is 1.03.

Is that what it says?

A. That's what it says. But once again, I don't know the significance of that number.

Q. And for non-Hodgkin lymphoma it shows that the standard incidence rate for Camp Lejeune is .86, right?

A. That's what it says on the page.

Q. And that would indicate that there are 14 percent fewer non-Hodgkin lymphomas at Camp Lejeune than in the general population.

MS. GREENWALD: Objection; form.

A. I don't know that you can strictly say it's 14 percent. I don't know how you -- I mean, I understand where you got that number, but I don't know if that's accurate way of discussing it.

And plus, I said talking about a preprint is -- doesn't make sense.

Q. And for leukemias, the standard incidence rate for camp -- standard

1
2 incidence rate for Camp Lejeune is .0 87,
3 right?

4 A. That's what it says on this
5 table.

6 Q. Let's go back to your report. I
7 am looking at the NHL/leukemia report
8 which is Exhibit Number 4. And I'm
9 turning to -- I'm turning to page 26.

10 And this is a table of studies,
11 correct?

12 A. One, two, three, four, five,
13 six, seven, yes.

14 Q. I'm just going to go through and
15 list the authors' names so that we can ask
16 questions about this without having to do
17 it for every single report.

18 So, the authors are: Lan,
19 Hosgood, Lee, L-E-E, Zheng, Li, L-I, and
20 Kamijina, and then Jia, J-I-A.

21 And so when I am asking
22 questions about these seven studies --
23 these -- first of all, these seven studies
24 were used in all three of your reports.
25 Is that right?

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A. Yes.

Q. The same seven studies?

A. Yes.

Q. Okay.

So if I refer to the seven studies, are you going to know what I'm talking about?

A. Yes.

Q. Okay, good.

Do you agree with the follow statement: Statistical significance does not indicate importance. A statistically significant result doesn't necessarily imply a scientifically or practically important effect and vice versa.

MS. GREENWALD: Objection; form.

A. That's a very convoluted statement. I don't know that I could answer that.

Q. The purpose of these -- well, let me just, you cited these seven studies that reported the effects of occupational TCE exposure on various immune markers.

Is that a fair characterization?

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A. Yes.

Q. And again, the seven studies are used in all three of your reports, right?

A. Yes.

Q. And so, you think these same seven studies are relevant to your opinions concerning kidney cancer?

A. They all have -- they all speak to the effect that TCE has in inducing immuno toxicity. And since that is relevant to all three of the diseases, they are included in all three of the reports.

Q. Okay. And I'm just going to clarify for the record so that I don't have to go through each report and ask you the same questions.

So, the seven studies are relevant to your opinions concerning kidney cancer, right?

A. Yes.

Q. And bladder cancer?

A. Yes.

Q. And non-Hodgkin's lymphoma?

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A. Yes.

Q. And leukemia?

A. Yes.

Q. Is there any particular study of the seven that is not relevant for one cancer over the other?

MS. GREENWALD: Objection; form.

A. I couldn't possibly say.

Q. So you couldn't say whether one is more prostate kidney cancer versus bladder cancer, for example?

MS. GREENWALD: Objection; asked and answered.

A. I would have to sit there and evaluate -- I have -- I didn't do that kind of evaluation, so I can't tell you off the top of my head.

Q. Each of these seven studies involved comparisons for the measured outcomes between TCE-exposed and control or unexposed factory workers, right?

A. Yes.

Q. So, would you agree that for these seven studies to be relevant to Camp

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Lejeune, you have to adopt the assumption that the exposures to TCE for the workers in the studies were sufficiently similar to the TCE exposures at Camp Lejeune?

MS. GREENWALD: Objection; form.

A. No.

Q. You would not agree?

A. No, I would not agree.

Q. And why is that?

A. Are you asking me whether -- could you repeat that question again?

Q. Sure.

If your studies are relevant to Camp Lejeune, don't you have to adopt the assumption that the exposures to the workers in the seven studies you cited are sufficiently similar to the exposures to TCE at the population at Camp Lejeune?

MS. GREENWALD: Objection; form.

A. These particular studies, it would be very difficult to come up with some sort of dose response, partly because the estimates are in parts per million based on inhalation exposure, whereas the

1
2 exposure at Camp Lejeune was all
3 ingestion.

4 So, I -- I would not say that
5 you would have to compare -- that you
6 could directly compare these in terms of
7 concentrations.

8 Q. I think you anticipated my next
9 question, which is the factory workers in
10 the seven studies were exposed to TCE via
11 inhalation?

12 A. Correct.

13 And the body burdens from
14 inhalation, it's -- you have to calculate
15 that and -- and see how it relate to body
16 burdens from, you know, from ingestion.
17 And there's several different factors that
18 go into that?

19 Q. Was -- so, you won't know
20 whether the frequency, intensity and
21 duration of TCE exposure is the same
22 between the factory workers in your seven
23 studies and the Camp Lejeune subjects?

24 A. Correct.

25 MS. GREENWALD: Objection; form.

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A. Well, are you -- are you asking me whether I can tell whether the exposures are exactly the same, or?

Q. Yes.

A. I can't tell.

Q. Can you tell whether they're similar?

A. Once again, you would have to calculate this in a different way comparing inhalation versus ingestion, and you also have to take into the account that you can't really use this one limited -- these studies to come up with necessarily a dose response.

Q. Have you calculated it, the exposure for the seven studies?

A. How did I calculate it?

Q. Have you.

A. Oh, have I.

No.

Q. Okay. Let me ask you about a specific study.

You cited to Lee et al. It's reference number 96.

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And again, these were olfactory workers who had TCE exposure. Is that right?

A. Just a minute. I need to check on that reference.

Q. Sure.

A. You're talking about the Lee, did you say?

Q. Yes, L-E-E.

A. Yes. Yes, they were faculty -- factory workers.

Q. With TCE exposure?

A. Yes.

Q. Did any of the TCE-exposed or control workers in the Lee study have kidney cancer?

A. I cannot remember that.

Q. Did any of them have bladder cancer?

A. I would have to have the study in front of me. I can't really tell you that.

Q. Did any of them have non-Hodgkin's lymphoma?

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A. I don't know.

Q. Did you review the studies before your deposition?

A. All of these, no.

Q. If you -- so, in the Lee study, it looks like you honed in on serum immunoglobulin G and serum immunoglobulin M. And to avoid saying 'immunoglobulin' a hundred times, I'm going to say Ig, that's I lower-case G upper-case G, and IgM.

So, in the Lee study, you honed in on the serum IgG and serum IgM.

Is that right?

A. I don't remember if I homed [sic] in on it. That's what I reported.

Q. And those two serums were reported to be statistically significantly decreased for the TCE-exposed workers relative to the control subjects, right?

A. That's correct.

Q. And although the levels of serum IgG and serum IgM were statistically lower for TCE-exposed workers compared to control subjects, the levels were normal,

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correct?

MS. GREENWALD: Objection; form.

A. I don't remember. You -- are you asking me whether they fall in the normal range?

Q. I'm ask -- yes, I'm asking whether even though they were statistically decreased, whether they still had levels of serum IgG and IgM that were normal.

A. I don't remember what the normal range is for those serums.

Q. Do the statistically significant differences in the serum IgG and serum IgM show functional importance?

MS. GREENWALD: Objection; form.

A. I don't know how to answer that. I haven't studied whether -- how to correlate exact measurements of IgM or IgG to particular functions.

They certainly indicate an immune dysfunction.

Q. If they are still in the normal range, do they still indicate immune

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dysfunction?

A. Yes.

Q. Do they indicate immunosuppression?

A. Yes.

Q. So is it your opinion that subjects who were still in the normal level of serum IgG or serum IgM are immunosuppressed?

A. I'm saying that the subjects in this study demonstrate immunosuppression by virtue of the fact that their serum levels of IgM and IgG went down.

Q. Do you agree that low serum IgG can be a nonspecific finding?

MS. GREENWALD: Objection; form.

A. Do you mean -- what do you mean by nonspecific finding?

Q. A symptom, sign, or test result that doesn't point to a specific disease or cause; it could be associated with many causes.

A. It's true, different things can cause decreases in serum levels.

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Q. Would you agree that the TCE-exposed workers in Lee would not be characterized clinically as having low -- abnormally low serum IgG levels?

MS. GREENWALD: Objection; form.

A. Once again, I don't remember what the normal range is.

Q. If they had serum IgG levels within the normal range, would that have a clinical significance?

A. Difficult to say. It would probably be -- it could happen in conjunction with other kinds of immune dysfunctions.

Q. Meaning what other types of immune dysfunctions?

A. If you're getting decreased levels of serum IgM or IgG, maybe seeing decreases in the number of B-cells or the de -- a decrease in the ability of the humoral immune response to to respond to antigens, all of those things could definitely make you immunosuppressed and more susceptible to cancer and infections.

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Q. What about just one of those things?

A. Yeah, any one of those things could still be -- could be very detrimental -- detrimental.

Q. Did the Lee study do any follow-up to corroborate that the differences in the multiple markers were causally related to kidney toxicity?

A. As far as I know, this study was focused on immunotoxicity. I don't -- I can't remember if they looked at serum levels of kidney markers.

I'm using it here as an example of immunotoxicity.

Q. Let me ask you about a different study. This one is the Lan study, L-A-N. It's the first one.

A. Okay.

Q. In the Lan study, you looked at cell counts for lymphoid subpopulations in blood. Is that right?

A. Correct.

Q. And those were reported to be

1
2 significantly -- significantly, but
3 marginally decreased for the higher
4 TCE-exposed workers relative to the
5 control subjects, but not to the
6 lower-exposed TCE workers.

7 Is that right?

8 A. I would not characterize it that
9 way.

10 Q. How would you characterize it?

11 A. I would not say that a decrease
12 from 1,356 to 1,124 was marginal.

13 Q. And you're looking at which
14 number?

15 A. The total T-cells.

16 Q. And is that still within the
17 normal range?

18 A. I don't remember what the normal
19 range is.

20 I mean, the point of the study
21 was to see whether or not exposure to TCE
22 impacted the number of lymphoid cells in
23 the blood, and it did.

24 For example, in the NK cells
25 went from 467 to 282. Once again, that's

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not marginal.

Q. Is it within the normal range?

A. I don't know what the normal range is.

The point is that you are suppressing the immune function by doing this, and you're suppressing it across the board on several different types of immune cells?

Q. And this statistically significant result doesn't necessarily indicate a primarily important effect though --

MS. GREENWALD: Objection; form.

Q. -- does it?

MS. GREENWALD: Objection; form.

A. It would be very difficult to imagine how that kind of a decrease in multiple immune cells would not have a functional impact.

Q. Do you know from the Lan study whether they were measured for functional impact?

A. I don't remember.

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Q. Do you know whether there was any follow-up to determine whether any of the people in the Lan study developed kidney cancer?

A. Once again, the focus for these researchers was on dem -- or testing the immunotoxicity.

I don't remember that they were looking at other endpoints.

Q. So they wouldn't have followed to see whether there was an end result of cancer?

A. I can't say for sure.

Q. What's your definition of immunosuppression?

A. Well, there is -- I think I talked about this. There is a -- a definition, I think it was cell ratings company that laid out the criteria for calling something an immune toxicant, and there are a number of different parameters, but certainly decreasing cell numbers is one of them.

Q. We would have all different

1
2 levels though, right?

3 A. There's a -- yes, there's
4 heterogeneity, sure.

5 Q. So if mine are ten points lower
6 than yours, am I immunosuppressed?

7 MS. GREENWALD: Objection; form.

8 A. If you were exposed to -- if a
9 bunch of people were exposed to a chemical
10 and they got a uniform decrease in the
11 numbers of immune cells, you could say
12 that was immune toxicity.

13 Q. And would you say it's
14 immunosuppression?

15 A. Yes.

16 Q. In the Lan study, they looked at
17 recent infections between TCE-exposed
18 workers and the control subjects, right?

19 A. I don't remember that part.

20 Q. And you don't remember whether
21 they found that the TCE-exposed subjects
22 who lower percentage of recent infection
23 in comparison with the control group?

24 A. I don't. I was just presenting
25 the strictly immune lymphoid cell data.

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Q. And is it your opinion that the TCE-exposed subjects have normal or abnormal numbers of lymphoid subpopulations in their blood?

A. Once again, I don't know the range for normal cells, but there's clearly a decrease, and if you have a decrease in that many cell populations, you're demonstrating immunosuppression.

Q. Are you aware of any peer-reviewed scientific literature that would support the contention that someone with a low but normal lymphoid subpopulation is immunosuppressed?

A. The definition of an immune toxicant is decrease -- a demonstratable decrease in cell numbers. So if you demonstrate that, you are demonstrating immunotoxicity.

Q. And are you demonstrating immunosuppression?

A. In this case, yes.

Q. But going back to my question. Are you aware of any

1
2 peer-reviewed scientific literature that
3 would support the contention that someone
4 with normal lymphoid subpopulation counts
5 is immunosuppressed?

6 A. I think I just answered that.
7 You are immunosuppressed to a
8 degree because you are showing lower
9 levels of those cell populations.

10 Q. Was there any clinical
11 significance to the findings in Lan?

12 A. What do you mean? Did they --
13 did they show clinical significance; do I
14 think they're clinically significant?
15 What do you mean?

16 Q. Do they show any clinical
17 significance?

18 A. I don't remember what other
19 endpoints they looked at.

20 Q. Do you know whether they looked
21 at whether these subjects eventually had
22 cancer?

23 MS. GREENWALD: Objection; asked
24 and answered.

25 A. No, I -- this particular set of

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experiments, they did not do that.

Q. For the Lan study they did not do that?

A. Correct.

Q. Let me ask you about another study. This one is the Kamijina study. That's K-A-M-I-J-I-N-A.

A. Okay.

Q. And am I correct that the study population in Kamijina is different than the occupational cohorts that were involved in the Lee and Lan studies?

A. Yes.

Q. And can you expand on that by talking about what OMLDT is?

A. About what, about what is?

Q. OMLDT.

A. Where are you?

Q. Isn't that the disease?

A. Oh, there's -- there's different names for it.

You're talking about the TCE-induced hypersensitivity.

Q. Yes.

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A. Dermal disease.

Q. Yes.

A. It's fairly common in Asia, and people that get this, it usually occurs within a month when you starts working with it, and you can get -- you get terrible rashes. You get alterations in your immune cells. You get liver disease. And a lot of these people end up in the hospital.

Sometimes removing them from the -- their occupational setting negates the symptoms, and sometimes it doesn't, and sometimes people even die from this particular disease. And if you saw the pictures in the journals of people with this disease, you would realize how serious it is.

Q. And is it your opinion that people who have this disease that involves rashes is relevant to the diseases that people experienced at Camp Lejeune?

MS. GREENWALD: Objection; form.

A. It's relevant in that you're

1
2 seeing increases in pro-inflammatory
3 cytokines, and that is part of the whole
4 process by which immuno toxicants how it
5 can promote cancer.

6 So, these people saw an increase
7 in the serum levels of TNF-alpha, and that
8 is a pro-inflammatory cytokine that is
9 associated with different cancers.

10 Q. Do you know whether the Camp
11 Lejeune population has -- whether OMLDT is
12 one of the diseases being studied for the
13 Camp Lejeune population?

14 MS. GREENWALD: Objection; form.

15 A. As far as I know, it was not.

16 Q. And in this study, I think you
17 just said, the cytokines TNF-alpha and
18 IL-10 were elevated in the TCE-exposed
19 patients, right?

20 A. I don't see IL-10.

21 In the Kamijina?

22 Q. Yes.

23 A. I just see serum TNF-alpha.

24 Q. Okay.

25 And those were elevated, right?

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A. Yes.

Q. But in the Lan study, the lymphoid subpopulations in blood were decreased.

A. That's correct. As I explain in my report, immunotoxicity can take a form of inflammation, pro-inflammation, chronic inflammation is definitely a component for developing cancer, and you can also get, on the other hand, immune suppression.

Q. And how do you know which effect you're going to get again?

A. Which what you're going to get? What do you mean?

Q. Well, if it can have pro-inflammatory and suppressive effects, how do you know which -- how do you know that they don't balance each other out?

A. Because they are working at completely different aspects of the immune response.

So, the -- the pro-inflammation is these nonspecific cytokines that call in these other cells, and they cause

1
2 oxidative stress. They call a lot of
3 these cells in and all that, sort of,
4 initial inflammation certainly helps with
5 initiating the whole cancer effect.

6 Whereas, you also get
7 suppression of the -- this is the innate
8 immune system. You also get suppression
9 of the humoral -- or, the adaptive immune
10 system, which is what you're seeing here,
11 and you put those things together and it's
12 like a perfect storm for getting cancer.
13 You're causing inflammation early in the
14 response for these innate signals and
15 you're suppressing the B-cells and T-cells
16 that are supposed to come in later on and
17 deal with that cancer, and you put those
18 two things together, it's -- it's been
19 found to be -- that combination has been
20 found to be associated with numerous
21 cancers.

22 Q. And when you say "has found to
23 be," are you referring to specific
24 studies?

25 A. Yes.

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Q. Which studies?

A. Well, some of them I cite in here, but there -- it's also associated with other cancers. It's not just limited to kidney, NHL, and bladder. There -- almost any cancer you look at these days you will find a component associated with the pro-inflammation and the immune suppression.

Q. But those people haven't necessarily been exposed to TCE?

A. True. But if you have a compound that causes those two things, it's certainly more likely that that's going to impact the cancer process.

Q. Do you agree that none of the immune markers identified in the seven studies are considered to be validated markers of cancer risk?

A. No, I don't agree.

Q. And why not?

A. Well, as I said, if you see an increased level of pro-inflammatory cytokines in a chronic fashion, that is

1
2 considered a likelihood of getting cancer.

3 Q. A likely cause?

4 A. Depends what you mean by
5 "cause."

6 Did it initiate the
7 genotoxicity, or did it promote the events
8 that came after that? In any case,
9 it's -- it's a causative effect.

10 And the same thing with the
11 immunosuppressed adaptive immune response,
12 there's numerous papers showing that if
13 you have immunosuppression because of a
14 genetic reason or because you're taking
15 immunosuppressive drugs due to a kidney
16 transplant, for example, that makes you
17 much more likely to develop cancer.

18 Q. Specific cancers, right?

19 A. I'm sorry?

20 Q. Specific cancers?

21 A. I think it's -- there's a wide
22 range of cancers. I don't know if it's --
23 how limited that number is.

24 Q. So, you're saying that it could
25 lead to all cancers?

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MS. GREENWALD: Objection; form.

A. I can't say all cancers, but there's certainly a number of them out there where that would be true.

Q. So, if someone is taking immunosuppressive drugs, they've had a stem cell transplants or something, are those people more likely to get a certain kind of cancer?

A. Well, like I said, I don't know the specifics -- ones, but I know that if you look at many of the papers, they will tell you that generalized immunosuppression is one of the main criteria for developing cancer. And so, it certainly implies, I don't know if every single cancer has been studied in this regard, but just about every one that has been studied, that's what they find.

Q. So, would you agree or disagree that the concept of immunosuppression fostering carcinogenesis is not applicable to all cancers?

MS. GREENWALD: Objection; form;

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asked and answered.

A. I think he was just trying to answer that.

I mean, as I said numerous reviews will tell you that one of the most important indicator of likelihood of getting cancer is immunosuppression.

Now, does that mean that every single cancer falls into that category? I don't know that they've been studied, but it certainly is true for a vast number of them.

MS. McKEEVER: Let's take a short break, please.

THE WITNESS: Okay.

THE VIDEOGRAPHER: The time right now is 2:41 p.m.

We're off the record.

(Recess taken.)

THE VIDEOGRAPHER: The time right now is 3:07 p.m.

We're back on the record.

BY MS. McKEEVER:

Q. I just want to quickly go back

1
2 to a couple of things I referenced
3 earlier.

4 If you could look at Exhibit 6,
5 please.

6 A. Exhibit what?

7 Q. Six.

8 A. Six.

9 (Pause.)

10 Q. For the NHL/leukemia, the long
11 list of materials, I had said earlier that
12 the National Research Council report of
13 2009 appeared in your materials
14 considered, and it's in this supplemental
15 material. I had said it was attached to
16 the report.

17 But if you look at the long
18 list.

19 A. This?

20 Q. That, yes. It should be number
21 113.

22 A. What do you mean 113?

23 Q. Reference number 113.

24 A. Oh. Okay.

25 Q. So, that's the National Research

1
2 Council report that we discussed earlier
3 in Exhibit 13, right?

4 A. Yes.

5 Q. That's the reference to that.
6 Okay, I just wanted to clarify.

7 And then on -- attached to your
8 kidney cancer report, after -- at the very
9 back after the fee schedule.

10 A. Where?

11 Q. I'm on the kidney cancer report.

12 A. Oh, right, right, right.

13 Q. Which is Exhibit Number 5.

14 A. Mm-hm.

15 Q. So I'm not in the supplements
16 anymore. I'm sorry, I'm moving back to
17 the actual report, Exhibit Number 5.

18 A. Sorry.

19 Okay.

20 Q. At the very back of the report
21 there's a list of Dr. Gilbert's Reliance
22 Files.

23 A. Okay.

24 Q. And on page 2, number 7 it
25 references Bove, F.J. Evaluation of cancer

1
2 incidence among Marines and Navy personnel
3 and civilian workers exposed to
4 contaminated drinking water at USMC Base
5 Camp Lejeune: a cohort study, unpublished
6 2024 January 29, and then it references
7 the Bates number
8 CLJC_ATSDR_BOVE-000060101.

9 That is the preprint that we
10 discussed earlier. Is that right?

11 A. Yes.

12 Q. Okay.

13 So you have seen the preprint
14 before today?

15 A. I guess I have.

16 Q. Okay. Let me ask you some more
17 questions about immunotoxicity.

18 What data supports that DNA
19 reactive metabolized in the kidney are
20 transferred to the bladder via urine?

21 A. TCA, trichloroacetic acid, is
22 one of the metabolites of both PCE and
23 TCE, and one of those studies, they showed
24 that if they gave mice just the TCA, they
25 ended up finding a -- effects in the

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bladder. They found increases in c-Myc expression associated with decreases in the DNA methylation of c-Myc.

Q. Okay. That was a technical -- this was a very technical answer.

How does it show that the metabolites are transferred to the bladder via urine and that therefore caused bladder cancer?

A. Because TCA is eliminated via urine. So it goes into the bladder and then out of the bladder.

Q. Do all of the metabolites go from the kidney into the bladder?

A. No.

Q. And some are eliminated in other ways?

A. Yes.

Q. And which ways are those?

A. The -- the -- so, the oxidative metabolites, that's what we're talking about here, the TCA's, mostly end up going through the bladder.

The glutathione pathway, some of

1
2 those are broken down before they get into
3 the bladder.

4 Q. And go where?

5 A. They are eventually eliminated
6 through the urine, but they don't show up
7 as the toxic metabolites in the -- in the
8 bladder.

9 Q. Has any regulatory agency
10 concluded that TCE causes any particular
11 lymphoma through immunomodulation?

12 A. That's a long question. Could
13 you break it down?

14 Q. Has any regulatory agency
15 concluded that TCE causes any particular
16 lymphoma through immunomodulation?

17 MS. GREENWALD: Objection; form.

18 A. I'm -- I'm still not clear what
19 you're saying.

20 Are you saying what -- I'm still
21 not understanding what you mean -- what
22 you want me to ask about.

23 Q. So, regulatory agencies, like
24 the EPA for example, have any regulatory
25 agencies concluded that TCE causes a

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particular cancer, like bladder cancer or lymphoma?

A. Well, yes. That's what we're -- been talking about.

Q. They have concluded that TCE causes --

A. Causes kidney cancer, yes.

Q. Through immunomodulation?

A. Oh, so, I'm glad you brought that up.

The -- it's been recently in the last few years, I guess more like 20 years, decided that the mechanism of action component of causation is gaining more and more importance. And in fact, some agencies are talking about using the mechanism of action endpoints to identify carcinogens before you find anything at the epi -- at the epi level.

So, it's huge right now to talk about the mechanism of action, and if you look at the key characteristics of carcinogens, they describe a lot of different characteristics. There's ten

1
2 different characteristics, and I think TCE
3 hits at least eight of those endpoints.
4 So now you have TCE causing these
5 endpoints, which they say are important
6 for a carcinogen, and then they say what
7 the -- what you need to do next is put
8 those together in some sort of
9 biologically plausible process, which is
10 what I did in my -- in my studies.

11 So you don't have to show
12 that -- you -- you got these different
13 connecting lines. You don't have to show
14 that this one specifically ends up at this
15 one to conclude that the final endpoint is
16 going to be kidney cancer and that the
17 immune dysfunction plays a role in it.
18 That's -- it's becoming more and more
19 accepted as a way of looking at the
20 mechanistic components of carcinogens.

21 Q. What does that mean "becoming
22 more and more accepted"?

23 A. Well, because if you -- if you
24 look at some of the literature in terms of
25 just finding carcinogens, they -- they

1
2 are -- they are mentioning and saying that
3 mechanism of action is -- is really
4 important and that they want to be able to
5 use it. If they can define endpoints that
6 are biologically plausible that lead to
7 cancer, if they can define those
8 endpoints, they can screen chemicals for
9 hitting those endpoints without having to
10 wait and see on an epi study that they
11 actually cause cancer. And that way they
12 can say that they're likely to cause
13 cancer, and we can start dealing with it
14 before we actually have the epi studies.

15 Q. It isn't established science
16 yet?

17 MS. GREENWALD: Objection; form.

18 A. I don't know what you mean by
19 "established science."

20 It is becoming more and more a
21 way of looking at how to define
22 carcinogenesis.

23 Q. You said earlier, and I think
24 you were referring to the regulatory
25 agencies, they are talking about doing it,

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and I think you mean using the MOAs to determine whether TCE can cause cancer.

What does it mean -- did I say that wrong?

A. That they're -- they're not -- say that again. I don't think they're waiting to see whether the MOAs can link TCE to cancer.

Q. I'm talking about in the context of immunomodulation, and I thought you said they are talking about doing it, and I don't know what that means.

MS. GREENWALD: Objection; form.

A. I don't remember saying that exactly.

All I know is that if you look at the key characteristics which have now been defined, one of them is inflammation and one of them is immune suppression.

Q. But one of them is not kidney cancer?

A. They are talking about cancer in general. They haven't limited it to a particular cancer.

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Q. And have they concluded that the weight of evidence links these mechanisms' causability to a particular cancer?

MS. GREENWALD: Objection; form; asked and answered.

A. Well, sure. I mean, like I said, there's plenty of studies showing that if you're immunosuppressed you're more likely prone to get kidney cancer.

Q. And those are the studies cited in your kidney cancer report?

A. Well, not just there. They're currently widely known.

As I said, immunosuppression is considered one of the key characteristics that is going to make you much more likely to develop cancer.

Q. Let me ask you about inflammation. I think you mentioned that earlier.

A. Okay.

Q. Inflammation -- would you agree that inflammation can be caused by many extrinsic factors and intrinsic processes?

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A. Inflammation can be -- has many possible initiators, yes.

Q. And chronic inflammation is a characteristic of NHL, for example?

A. I believe it is, yes.

Q. And chronic inflammation in NHL would occur without TCE exposure, right?

A. Can't answer that.

Q. If chronic inflammation is characteristic of cancer, not all people who have cancer were exposed to TCE, right?

A. True.

Q. So how do we separate out inflammation that may have been caused by TCE versus inflammation that is inherent with the disease?

MS. GREENWALD: Objection; form.

A. When you say "inherent with the disease," I mean, it's usually a precursor of the disease.

Q. Okay. And --

A. So something's causing it.

Q. Would you agree though, and I

1
2 thought you said this a minute ago, that
3 chronic inflammation is a characteristic
4 of cancer?

5 A. Yes.

6 Q. But not all cancers have prior
7 TCE exposure?

8 A. Yes.

9 Q. How do inflammatory mechanisms
10 that are triggered by TCE differ from
11 background inflammation that is you said
12 it was a precursor to the disease?

13 A. Well, when you say "background
14 inflammation," so there's -- certainly
15 there is a normal amount of inflammation
16 that goes on in any kind of daily response
17 to any kind of antigen, but we're talking
18 about increased levels, unusual
19 inflammation.

20 Q. And where is the data that
21 supports that TCE tips the balance towards
22 harmful inflammation?

23 MS. GREENWALD: Objection; form.

24 A. Well, as I mention in my report,
25 there is that information that, for

1
2 example, TNF alpha is increased in -- in
3 people that are exposed to TCE.

4 Q. And that's what you're relying
5 on, the seven studies in your reports?

6 MS. GREENWALD: Objection; form.

7 A. I wouldn't say I was only
8 relying on those. I've been looking at
9 this for a very long time. So I'm sure
10 there are other studies that probably also
11 fit into that.

12 Q. But those are the studies you've
13 used in your report?

14 A. Those are the studies I've used
15 in my report.

16 Q. Why did you choose those seven
17 studies?

18 A. They're, I guess, fairly well
19 representative and they had a number of
20 different parameters that they looked at
21 in each study, for the most part.

22 Q. Did you consider and reject
23 studies that you don't have in your
24 reports?

25 MS. GREENWALD: Objection; form.

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BY MS. McKEEVER:

Q. Did you reject any studies that -- for -- excuse me. It's been a long day.

Did you reject any studies?

A. Did I reject any studies in what -- in what context?

Q. That could have been included in I keep calling them the seven studies because the tables are different -- have different numbers in your reports, but the -- in the immunotox section, you have seven.

Seven studies.

Did you consider and reject other studies that could have gone into this table?

MS. GREENWALD: Objection; form.

A. I may have -- there aren't -- I may have rejected one or two, but it's more likely that I just left a couple out.

Q. And do you recall that you did --

A. Yes.

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Q. -- or just that you may have?

A. I did.

Q. And do you recall which ones you left out?

A. I left out the Iavicoli and I left out Beyers.

Q. And why did you leave out Iavicoli?

A. Just an oversight.

Q. And why did you leave out Beyers?

A. Same.

Q. On your CV you listed a book that you co-edited "Trichloroethylene: Toxicity and Health Risks."

Right?

A. Yes.

Q. And you wrote two of the chapters in that book?

A. Correct.

Q. You wrote one chapter which was "Trichloroethylene and Cancer."

Right?

A. I co-authored that.

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Q. Co-authored.

That chapter does not discuss any ways in which TCE might cause cancer through changes in the immune system, does it?

A. That chapter was written many years ago. So we know a lot more about the mechanism than we did then.

Q. It was written in 2014, right?

A. (Nodding.)

Q. And -- is that right?

A. I honestly don't remember. That sounds about right.

MS. McKEEVER: What number are we on?

THE STENOGRAPHER: 15.

MS. McKEEVER: I'm handing you Exhibit 15, which is a excerpted copy of the book "Trichloroethylene: Toxicity and Health Risks" edited by Kathleen M. Gilbert, Sarah J. Blossom, and I've included the copyright page and chapters 2 and 9.

(Gilbert Exhibit 15, excerpt of

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Trichloroethylene: Toxicity and Health Risks, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. So, was this book written before the seven studies that you cited in your report?

A. I -- I can't remember the -- the sequence of the studies.

Q. But you agreed with me that the chapter in 2014 trichloroethylene and cancer does not discuss any ways in which TCE might cause cancer through changes in the immune system?

A. This chapter was written primarily as just a -- as another endpoint discussion. It wasn't intended to be an in-depth look at TCE and cancer. It was to -- to have a chapter in there that showed that TCE could induce cancer.

Q. But the answer to my question is "no," correct?

A. I have no look --

MS. GREENWALD: Objection; form.

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BY MS. McKEEVER:

Q. The chapter does not discuss ways in which TCE might cause cancer through changes in the immune system?

A. I will have to look through it. Just a second. It's been a while.

MS. GREENWALD: Objection to form also.

Go ahead, looking.

A. (Witness reads document.)

It does not. But we know at this point in time a lot more about how the immune system impacts cancer.

Q. On page 181 --

A. Of what?

Q. Of the book, yes.

A. Of the book.

Q. Chapter 9, Exhibit 15, page 181.

A. Okay.

Q. The last couple of sentences are: A number of cancers have shown a relationship to TCE exposure, some stronger than others. They include non-Hodgkin lymphoma and other

1
2 hematopoietic cancers, cancer of the liver
3 and biliary tract, breast cancer, bladder
4 cancer, and lung cancer. These may be the
5 next chapters in the history of
6 TCE-related cancer.

7 What does that mean "these may
8 be the next chapters in the history of
9 TCE-related cancer"?

10 A. As more studies come out, the
11 more information we'll have and the better
12 we'll be able to define those
13 relationships.

14 Q. Is there anything in this
15 chapter that you wrote about how chronic
16 inflammation promotes the growth of kidney
17 cancer?

18 A. I don't believe so.

19 Q. Is there anything in this
20 chapter about how TCE-induced oxidative
21 stress is another mechanism by which TCE
22 can cause kidney cancer?

23 A. So, Dr. Wartenberg is very well
24 known for looking at the effects of TCE on
25 cancer. And as I said, this chapter was

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primarily to talk about the fact that TCE could induce cancer. It was not intended to be an in-depth look of how TCE caused cancer.

Q. But it doesn't mention oxidative stress causing cancer. Is that right?

MS. GREENWALD: Objection; form.

A. Yes, you are correct, it does not mention those specific things.

Q. Have you opined outside of this case that TCE-induced tox -- immunotoxicity causes cancer?

A. Opined in what regard?

Q. In a book or a journal or an expert opinion in another case?

A. So, my work was primarily -- or, largely looking at autoimmunity. So, and it's only become, as I said in my report, the last few years, primarily since I retired, that the role of the immune system has been so well-documented in terms of causing cancer.

Q. And when you say "recent years," can you be more specific?

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A. Since I retired in 2017. So last eight years.

MS. McKEEVER: Okay.

I am handing you an expert report that you wrote. It's called "Expert Opinion of Kathleen M. Gilbert, Ph.D. In the matter of: Opal Millman," and it's dated June 15th, 2021.

THE WITNESS: Okay.

(Gilbert Exhibit 16, Expert Opinion of Kathleen M. Gilbert, Ph.D. In the matter of: Opal Millman, June 15, 2021, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. All right. On page 10 of Exhibit 16 you discuss TCE and cancer.

A. Mm-hm, yeah.

Q. Do you mention immunotoxicity?

A. Well, as that chapter -- I mean, that paragraph is just talking about whether TCE causes cancer, not how it causes cancer.

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Q. But there's a section on TCE and cancer that does not mention immunotoxicity, correct?

MS. GREENWALD: Objection; form.

A. It doesn't mention a lot of things.

Q. And in the next section -- I'm sorry, the third -- the section on page 11 it says: TCE and non-cancer effects. And then it lists immunotoxicity, and this is where immunotoxicity is discussed, right?

A. Yes.

Q. So in this report you did not opine that immunotoxicity can lead to cancer?

A. I don't think I did.

I can't remember what the emphasis was as far as Ms. Millman.

Q. On page 12, the last paragraph: Epidemiological studies examining the cause of disease often use a case-control study design. In this kind of study, people with a specific disease would be evaluated in parallel to a suitable

1
2 control of people without the disease.
3 The investigators could then compare the
4 two groups in terms of environmental
5 exposure. Unfortunately, such studies to
6 examine the link between TCE -- between
7 human TCE exposure and the development of
8 individual autoimmune diseases have not
9 been conducted.

10 Did I read that correctly?

11 A. Yes.

12 Q. If we don't know which of the
13 80-plus autoimmune diseases may be linked
14 to TCE-induced immunotoxicity, how do we
15 know which of hundreds of different
16 cancers might be linked to TCE
17 immunotoxicity?

18 MS. GREENWALD: Objection; form.

19 A. I'm trying to think this -- how
20 to word this.

21 All it's saying that the
22 specific studies have not been done, for
23 example myasthenia gravis. So there's
24 been a study showing is that TCE causes
25 myasthenia gravis. Not that I know of.

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It doesn't mean that we can't talk about the immunotoxicity being applicable to many different kinds of autoimmune disease.

Q. But the specific studies to link TCE-induced immunotoxicity to a particular autoimmune disease have not been done?

A. The incidence of any particular -- in general, there's a fair number of people with autoimmune diseases, but the incidence of any individual one, some of them are like one in a hundred thousand. So it is very difficult to do the kinds of studies that you're talking about.

Q. And is the same true for cancer?

A. No, there's -- cancer incidence is higher for individual cancers. There's so many different autoimmune diseases, like, that I -- like I said, that any one particular one is -- is very difficult to study the connection.

Q. Aren't there more cancers than autoimmune diseases?

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A. Yes.

Q. On page 25.

A. Yes, now I remember. She had trigeminal neuralgia.

Q. I am looking at the first paragraph underneath "Relative contribution of toxicant exposure and genetics to disease etiology." And the last sentence in that paragraph says: Disease etiology of complex diseases is thought to contain both genetic and environmental components. Although their relative contributions to disease etiology can be difficult to tease out.

You would agree --

MS. GJONAJ: What page are you on? I didn't mean to interrupt your question.

MS. McKEEVER: 25.

BY MS. McKEEVER:

Q. Do you need me to read it again?

A. No.

Q. Do you agree with this statement?

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A. Yes.

Q. And that would be true for autoimmune diseases?

A. It depends what you mean. It's all relative.

I mean, there are a number of twin studies for autoimmune diseases where they have a -- based on that, they have a pretty good idea of what the genetic component is to that. So they assume that the remaining percentage of the disease is some sort of environmental. Now, that can be different -- that means different things, but at least they can -- they talk about we know some of that, and whether or not that environmental component is one single trigger or more than one trigger is what we're trying to figure out.

Q. And for diseases without a known genetic component, it is different, as you said, to separate out the relative contributions of genetic and environmental components.

Is that right?

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MS. GREENWALD: Objection; form.

A. But most diseases at this point we have some idea of what the genetic contribution is. For -- that's true for different cancers. It's true for different autoimmune diseases.

(Pause.)

Q. Let me ask you a question about your kidney cancer report, which is Exhibit 5 on page 26.

A. Page 26, you said?

Q. Yes.

A. Okay.

Q. I am looking at the second paragraph under B TCE-Induced Oxidative Stress and there is a sentence in the middle of that paragraph that says: It is still not clear how TCE increases oxidative stress.

You would agree with that statement, right?

A. Yes.

Q. And: However, signs of oxidative stress have been found in liver,

1
2 placenta, gut, embryonic heart and kidney
3 following TCE exposure.

4 Right?

5 A. Yes.

6 Q. What about the bladder?

7 A. As far as I know, they haven't
8 looked at it in the bladder.

9 Q. So has oxidative stress been
10 shown in the bladder?

11 A. As I said, I don't think there's
12 any studies that have looked at it.

13 Q. But you acknowledge that
14 oxidative stress can either be causal or
15 secondary to inflammation, right?

16 A. Yes.

17 Q. And is the oxidative stress
18 purportedly caused by TCE causal or
19 secondary to inflammation?

20 A. I don't know that that has been
21 definitively defined.

22 We know that the oxidative
23 stress is -- has functional significance
24 because in some of the immune studies, not
25 by my group, but another group, if you

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block the oxidative stress, you block some of the immunotoxicity.

And the same thing is true for, as it says in here, for the neurotoxicity.

So, the oxidative stress is having a functional effect, at least in those two systems.

Q. Which two systems?

A. The immune as I say -- the autoimmune system and the neurotoxicity.

Q. Did you say autoimmune and neurotoxicity?

A. Yes. 'Cause they've done studies in mice where if they blocked the oxidative stress component by itself, then they negate to a certain extent the effects of the TCE exposure.

Q. So that statement is based on mouse studies?

A. Yes.

Q. Would you agree that oxidative stress is a characteristic of chronic diseases?

A. It's -- it is one -- it's

1
2 probably found in most chronic diseases.
3 I don't know that it's found in every
4 chronic disease.

5 Q. If it's found in most chronic
6 diseases, how do you separate out
7 background oxidative stress that's present
8 in the disease with or without TCE
9 exposure from oxidative stress caused by
10 TCE exposure?

11 MS. GREENWALD: Objection; form.

12 A. There's a certain amount of
13 normal oxidative stress that's involved in
14 the body's response to pathogens. If
15 you -- but -- and the body can -- can deal
16 with that. It has mechanisms to prevent
17 damage from oxidative stress as long as
18 the levels at a certain point. If the
19 levels get higher, then you start to see
20 damage caused by oxidative stress.

21 So, if you treat a mouse with
22 TCE and you see oxidative stress goes up,
23 but then you block that oxidative stress
24 with a chemical inhibitor and the
25 neurotoxicity associated with the TCE is

1
2 diminished, then you're saying that the
3 oxidative stress component, which is
4 induced by TCE, has a causal role.

5 Q. And you're talking about a mouse
6 study?

7 A. Yes, we're talking about mouse
8 studies.

9 Q. Okay. And I believe you agreed
10 that oxidative stress is a characteristic
11 of most chronic diseases.

12 A. Yes.

13 Q. And if you have oxidative stress
14 in most chronic diseases, but most of
15 those people were not exposed to TCE, how
16 do you separate out the chronic -- I'm
17 sorry, the oxidative stress caused by TCE
18 versus the oxidative stress that's a
19 characteristic of the disease?

20 MS. GREENWALD: Objection to
21 form.

22 A. I'm still not understanding your
23 question.

24 You're saying -- what are you
25 saying? I'm sorry, could you rephrase

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that?

Q. Sure.

You said oxidative stress is a characteristic of most chronic diseases, right?

A. Yes.

Q. And then TCE you say can cause oxidative stress.

A. Right.

Q. But if there's already oxidative stress associated with the disease, how do you know that it was caused by TCE, or how do you know what part of it was caused by TCE?

MS. GREENWALD: Objection; form.

A. I mean, if TCE is causing the oxidative stress in that disease, isn't --

Q. My question is there are people with chronic diseases who have oxidative stress as a characteristic of that disease, right?

A. Yes.

Q. Those people have not been exposed to TCE.

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A. As far as you --

MS. GREENWALD: Objection; form.
Go ahead.

A. As far as we know, yes.

Q. In this hypothetical, those
people --

A. All right.

Q. -- have not been exposed to TCE.

A. All right.

Q. How do you determine what TCE
exposure did to contribute to the
oxidative stress?

A. I thought you said --

MS. GREENWALD: Objection; form.

A. -- they hadn't been exposed to
TCE.

Q. In people who have -- in --
excuse me.

In diseases where you're saying
TCE leads to oxidative stress, how do you
separate out the TCE exposure from the
oxidative stress that's characteristic of
a chronic disease?

MS. GREENWALD: Objection; form.

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A. Well, partly in animal studies you can do it by looking at sequential events. So if you treat them with TCE and the oxidative stress goes up and then you get the disease, then that suggests a link.

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Now, oxidative stress is a little trickier, and they mention that in the key characteristics of carcinogens. If you only have oxidative stress, that's harder to put that into a mechanism of action. But TCE, as I say, induces or triggers at least eight of the different key characteristics of carcinogens, only one of which of those eight is oxidative stress.

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But if you have oxidative stress and you have all these other triggers indicative of cancer-causing elements, then it is perfectly reasonable to incorporate oxidative stress into your mechanism of action.

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Q. You mentioned the mouse models.

Are there mouse models where TCE

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exposure induces bladder cancer?

A. As far as I know, I have not seen that.

Q. Are there mouse models where TCE exposure induces non-Hodgkin's lymphoma?

A. For different reasons, mice may not be particularly conducive as models for some -- some diseases.

Q. Including NHL?

A. Including I can't remember if there's -- if there's a mouse model for NHL. I don't think so. I would have put it in my report.

But I know it's hard to induce bladder cancer in mice.

Q. Can TCE exposure in mice induce leukemia?

A. I don't think that these particular diseases have been studied with -- with regard to TCE and mouse models.

Q. And have they been studied in human models?

MS. GREENWALD: Objection; form.

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A. Well, we've been talking about the epi results indicating that TCE exposure causes NHL and leukemia.

Q. And those are the epi studies, we're now talking about?

A. Yes.

Q. Let me ask you about your opinion 4 we can look at the bladder cancer report.

MS. KONSTANTOPOULOS: Exhibit 3.

BY MS. McKEEVER:

Q. Exhibit 3.

Are you there?

A. I'm on.

Q. Okay.

On page 29 in the Summary you say: It is more likely than not that levels of TCE, PCE, and benzene exposure at Camp Lejeune were sufficient to promote the development of bladder cancer.

Right?

A. Yes.

Q. And there's a similar statement on page 35 of the NHL/leukemia report.

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A. Okay.
Hold on while I look that up.

Q. That is Exhibit?

MS. KONSTANTOPOULOS: 4.

BY MS. McKEEVER:

Q. 4.

MS. McKEEVER: Thank you.

A. What page did you say?

Q. 35.

A. Okay.

Q. And it says in the Summary section: Taking to -- into account disease latency, exposure levels, and exposure duration, it is more likely than is --

A. Oops.

Q. It should say "more likely than not."

A. Right.

Q. It says: More likely than the TCE exposure at Camp Lejeune was hazardous to human health and sufficient to promote the development of NHL and leukemia.

Right?

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A. Yes.

Q. I believe there's a similar statement on page 31 of the kidney cancer report which is Exhibit 5.

Are you there?

A. Yes.

Q. And under the Summary section it says: Taking into account disease latency, exposure levels, and exposure duration, it is more likely than not the TCE exposure at Camp Lejeune was sufficient to promote the development of kidney cancer and was hazardous to humans.

Right?

A. Yes.

Q. So, the -- the opinions -- opinion 4 in all three reports is very similar, right?

A. I think this one I just talk about TCE, and I'm not sure that that's true for the other studies.

Q. I think you're right.

Kidney cancer and NHL/leukemia talk about TCE, and I believe the bladder

1
2 cancer report talks about TCE, PCE, and
3 benzene.

4 Does that sound right?

5 A. Yes, I think that's right.

6 Q. In all of them you use the term
7 "promote the development of cancer."

8 What does "promote the
9 development" mean?

10 A. So, I believe that every agent,
11 any kind of component that promotes,
12 enhances, encourages, increases any step
13 in the process ever tumorigenesis is
14 considered to be promoting the disease and
15 to contributing to causation.

16 Q. Contributing to causation?

17 A. Well, is causative.

18 Q. When you're talking about the
19 exposure at Camp Lejeune, did you
20 consider -- did the levels you're
21 referring to consider the route of
22 exposure?

23 A. Well, they were exposed via
24 ingestion, and then I talk about how they
25 may also be exposed via inhalation and

1
2 dermal.

3 Is that what you're talking
4 about?

5 Q. Yes.

6 I'm asking whether the levels
7 you referred to in your opinion took into
8 consideration the route of exposure.

9 A. I think they -- for the most
10 part they did.

11 Q. And did they take into account
12 the duration of exposure?

13 A. Some of them did. Some -- there
14 were some -- a lot of that information
15 just not available, so we have to work
16 with what we have. And some of them
17 discussed duration. Some of them were
18 better than others at discussing
19 concentrations.

20 It's -- it is not a clear-cut
21 picture, but in epi studies, you have to
22 work with what you have.

23 Q. On page -- well, okay. On page
24 27 of your bladder cancer report which is
25 Exhibit --

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MS. KONSTANTOPOULOS: 3.

Q. -- 3.

MS. McKEEVER: Thank you.

Q. I'm on page 27. Under the section labeled "TCE Exposure Level" the second paragraph says: In addition to reviewing the ATSDR modeling reports that are publicly available, I have reviewed the summary tables of plaintiffs' expert Morris Maslia. These levels are hazardous to humans generally and have been shown to cause bladder cancer.

Did I read that correctly?

A. Yes.

Q. Who has shown that these levels cause bladder cancer?

A. That is a summary of the response, and I would have to go back and look at all the individual studies in that table.

Q. And you're referring to Morris Maslia's report?

A. I'm referring partially to him and partially to the reports that I

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mention in the epi studies.

Q. I'm confused about which reports in the epi studies.

A. Well, I think I mention some of the different levels at the different -- from the different epi studies. I don't remember the details at this point over what they were in -- and what duration and what exposure levels they were in individual studies.

Q. And are you talking about epi studies specific to Camp Lejeune?

A. No. I'm talking about epi studies specifically in terms of levels that can cause bladder cancer.

Q. The next paragraph you say: There is no one methodology that clearly defines what concentration of TCE, PCE, or benzene causes bladder cancer.

Right?

A. Yes.

Q. Doesn't that conflict with your statement in the previous paragraph?

MS. GREENWALD: Objection to

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form.

A. What I was trying to say is that there is -- that we can't define a threshold value, even though we can show from individual studies exposure levels that have been caused -- shown to cause bladder cancer.

Q. How do you find that the levels of haz -- that the levels of exposure are hazardous if there's no method for doing so?

MS. GREENWALD: Objection; form.

A. Well, if you see a points of departure in the studies where people are getting increased levels of bladder cancer, then there -- those levels are hazardous.

Q. And do those levels cause bladder cancer, for example?

A. Well, that's what we're talking about.

Q. They do cause bladder cancer?

A. Well, according to the epi studies, they -- as far as they can

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estimate the exposure levels in individual studies, they're saying people exposed to this concentration could have an increased incidence or mortality from bladder cancer.

So you're saying that, in essence, that particular concentration is hazardous and is causing bladder cancer.

Q. On page 28 of the bladder cancer report in the last paragraph you reference using the level of benzene at 720 micrograms per liter, right?

A. Yes.

Q. What literature shows that exposure to this level of benzene can cause bladder cancer in human beings?

A. I think that the benzene oral cancer scope factor is not specific for bladder cancer.

Q. And does that mean the literature does not show that the exposure to this level of benzene can cause bladder cancer?

MS. GREENWALD: Objection; form.

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A. I don't remember. I can't answer that. I don't remember the specifics of the benzene studies.

Q. Looking at page 27 on the bladder cancer report under "TCE Exposure Level" you mention a measured value of 1400 micrograms per liter.

Right?

A. Yes.

Q. And that was the maximum measured value, right?

A. It had at that point, yes.

Q. And what literature shows that 1400 micrograms per liter of TCE exposure can cause bladder cancer in human beings?

A. I don't know that any study has looked specifically at that value.

There are epi studies that have looked at other exposure levels.

Q. And what alert shows that 1400 micrograms per liter can cause NHL in human beings?

A. Once again, you would have to look at the exposure levels described in

1
2 the individual reports and take it into --
3 take all these other factors into account.

4 Q. You would need individual
5 exposure risks, is that what you're
6 saying?

7 MS. GREENWALD: Objection; form.

8 A. Like I said, there's never been
9 a study that's shown that specifically
10 that 1400 micrograms per liter causes a
11 specific cancer.

12 Q. And then you also mention in
13 that same paragraph that: Levels of PCE
14 at the Tarawa Terrace water treatment
15 plant reached a maximum monthly level of
16 215 micrograms per liter.

17 Right?

18 A. Yes.

19 Q. And what literature shows that
20 215 micrograms of liter of PCE exposure
21 can cause bladder cancer in humans?

22 A. Once again you'd have to look at
23 the -- obviously there's never been a
24 study that looked specifically at that
25 concentration.

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You would have to look at the concentrations from the other epi studies.

Q. Is it your opinion that any exposure above federal guidelines increases one's health risks, any TCE exposure?

A. What federal guidelines are you talking about?

Q. Let's see. How about the EPA federal guidelines?

A. Well, there's still -- that encompasses a number of --

Q. Too many to say?

A. Yeah, yeah.

Q. Is it your opinion that there's no safe level of exposure to TCE?

A. No studies have been done that allow you to say what that number would be. So it's impossible to speculate how to answer that.

All we know, but on the other hand, since TCE has been labeled genotoxic carcinogen, theoretically, even one molecule is supposedly able to cause

1
2 cancer. But in terms of actually defining
3 what a threshold would be, there's just
4 not enough information to do that.

5 Q. All right.

6 And I think I understood you
7 correctly, but can you identify the
8 minimum -- you're saying there -- you
9 cannot identify the minimum threshold
10 exposure level from TCE to cause cancer?

11 A. Based on the information we have
12 now, no.

13 Q. Okay.

14 Can you identify the minimum
15 length of TCE exposure at any level to --
16 to cause cancer?

17 MS. GREENWALD: Objection; form.

18 A. Once again, the -- the data on
19 that is very -- it's -- it's not as robust
20 we would like, but I know there have been
21 a couple of guidelines, including the --
22 the stuff from Bove and a couple of other
23 researchers that say there's no reason to
24 think that a one-month minimum is not
25 correct in terms of increasing your risk.

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Q. A one-month minimum at what level?

A. That can't -- can't say that.

Q. Is everyone exposed to TCE at some level on a daily basis?

A. Can't say that either. I mean, a lot of water systems have some level of TCE, but most of the -- the water treatment plants filter out that. But, so exposure is difficult to estimate.

But I was just reading a study the other day that was looking at a international group of epi studies that looked at kidney cancer worldwide, and they were saying that based on their modeling, the incidence of TCE-induced kidney cancer is increasing and will continue to increase until at least 2050, which is the -- the length -- the endpoint of their modeling. So people are still being exposed to it.

Q. And people are exposed to benzene on a daily basis, right?

A. I don't know what the -- the

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likelihood of benzene exposure is.

Q. What about PCE, are people exposed to PCE on a daily basis?

A. Once again, it -- the vary -- the levels vary and the -- the water systems vary. It's -- I couldn't answer that.

Q. Okay. Let me ask you about your opinion 5.

Yeah, it's Exhibits 3, 4 and 5 in all three reports.

I'm trying to make it a little quicker by asking about all the reports together, but would you agree that your opinion 5 in all three reports the likelihood that the contaminants in the drinking water at Camp Lejeune caused bladder cancer in the bladder cancer report, was increased by aggregate exposure via inhalation and dermal routes and by cumulative co-exposure? That's the bladder cancer opinion, right?

A. Yes.

Q. And then there's a similar

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opinion for NHL and leukemia, right?

A. I'm not sure whether benzene gets thrown into those co-exposures.

Q. Let's look then.

I'm looking at the NHL/leukemia report, Exhibit 4.

I should have these memorized by now.

Page 35.

Opinion 5: TCE-induced NHL and leukemia in the plaintiffs was augmented by aggregate exposure via inhalation and dermal routes and by cumulative co-exposure to other contaminants in the drinking water.

Right?

A. Right. But you will see ben -- that there's come conversation about vinyl chloride in that one.

Q. Right.

A. So, the -- the two -- the sections are not identical.

Q. And I thought vinyl chloride was in all five -- no, all three reports,

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actually. I could be wrong about that.

A. No.

Q. Okay. So, vinyl chloride then is in the leukemia/NHL report, but not in the bladder cancer report?

A. Yes.

Q. Okay. And let me check the kidney cancer report.

Okay. So, in the kidney cancer report your opinion includes TCE, vinyl chloride, benzene -- benzene (different pronunciation) and PCE.

Is that right?

A. I don't see benzene in here.

Q. I'm looking at the Summary on page 33.

A. Okay.

Okay, I do mention benzene.

Q. So benzene --

A. Okay.

Q. For NHL/leukemia, it's again all four chemicals: TCE, PCE, benzene, vinyl chloride.

Is that right?

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A. I can go back and look.

Yes.

Q. And in the bladder cancer report, opinion 5 is based on TCE, PCE, and benzene, but not vinyl chloride.

Is that right?

A. I mention vinyl chloride, but, yes, it doesn't seem to be emphasized in that.

Q. Not in the bladder cancer?

A. In the bladder cancer.

Q. Continuing with the bladder cancer report, on page 30.

A. Page what?

Q. 30.

A. 30.

Q. In the middle paragraph you cite a study by -- a 2024 study by Rosenfeld and then say: This suggests that at the very least one should double the ingestion exposure of TCE to estimate the total TCE exposure at Camp Lejeune.

Is that right?

A. Well, it wasn't based just on

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the Rosenfeld.

Q. Okay. That was my question.
What is that statement based on?

A. There's two other studies there,
references.

Q. And you're citing to the Canada
reference at the bottom of the page?

MS. GREENWALD: Objection; form.

A. I'm citing to reference 129 and
130.

Q. Okay. And those are two
articles, one by Weisel Environmental
Health Perspective January 1996, and one
by Haddad, Journal of Toxicology and
Environmental Health December 2006.

So, those three studies are what
you're relying on to -- in your statement
where you say: One should double the
ingestion exposure of TCE to estimate the
total TCE exposure at Camp Lejeune?

A. Yes.

Q. Is the route of exposure
important?

MS. GREENWALD: Objection; form.

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A. What do you mean is it important?

Q. Well, is it important how the VOCs are metabolized?

A. Once -- okay.
So, whether you're exposed to TCE via ingestion, via inhalation, or via dermal exposure, once it hits the blood, the percentage that hits the blood is then metabolized very similarly.

The difference is mostly between the ingestion versus the oral -- I mean, the inhalation exposure.

So, inhalation, a larger proportion of the exposure is exhaled without being metabolized. Whereas in the ingestion, at least at the concentrations that you're seeing at Camp Lejeune, most of it's going to be metabolized and not exhaled.

Q. And why do you think it's important to double the ingestion exposure dose?

A. Well, not just the ingestion.

1
2 I'm saying -- so, you have the -- you know
3 how much they were exposed to via
4 ingestion, and there's other studies
5 showing you -- showing is that when TCE is
6 in your drinking water, your actual
7 exposure, your actual body burden is not
8 dependent only on what you're taking in
9 ingestion-wise, that the amount that you
10 have in contact with in showering and
11 washing dishes and just inhaling it is --
12 the body burden from that is equal to that
13 which you are calculating based on
14 ingestion.

15 Q. And so, doubling the ingestion
16 exposure --

17 A. Gives you a rough estimate.
18 I'm sorry, didn't mean to
19 interrupt.

20 Q. Thank you.

21 On page 31 of the bladder cancer
22 report in the fourth full paragraph you
23 say: The chemicals in a mixture can
24 interact in a manner that is additive,
25 less than additive, e.g. antagonistic, or

1
2 greater than additive, e.g. synergistic.

3 Right?

4 A. Yes.

5 Q. So if they're less than
6 additive, that means -- if they're
7 antagonistic, that means they can
8 counteract each other. Is that right?

9 A. That's what that means.

10 Q. Do you acknowledge that mixture
11 science is uncertain?

12 MS. GREENWALD: Objection; form.

13 A. Uncertain is a very vague term.

14 Q. On page -- well, let me ask it
15 this way.

16 Do we fully understand how these
17 chemicals interact with each other?

18 MS. GREENWALD: Objection; form.

19 A. Fully -- fully understand?

20 Q. Yes.

21 A. Interact at what level? At
22 metabolism, at molecular level? At
23 what -- what exactly are you asking?

24 Q. Well, we're talking about
25 mixture science, so --

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A. Right.

Q. -- interact with one another.

A. Right, but -- could you rephrase your question, please?

Q. When you say that the effects of chemicals at Camp Lejeune could be additive or synergistic, can you identify any studies that support this opinion for bladder cancer?

MS. GREENWALD: Objection to form.

A. The studies have not been done.

Q. Can you identify any studies that support this opinion for NHL caused by benzene, TCE, and PCE?

A. Once again, at this point in the research associated with chemical co-exposures, we can talk about additive effects of carcinogens in general. I don't think that they've pinpointed the additive or synergistic effects on any particular cancer. That may be -- that may be wrong, but I don't know of any with regard to these.

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MS. McKEEVER: Okay. I'm going to take a short break and try to wrap up fairly soon.

THE WITNESS: Okay.

THE VIDEOGRAPHER: The time right now is 4:23 p.m.

We're off the record.

(Recess taken.)

THE VIDEOGRAPHER: The time right now is 4:49 p.m.

We're back on the record.

BY MS. McKEEVER:

Q. Welcome back, Dr. Gilbert.

Before the start of the deposition, the plaintiffs' attorneys provided six invoices billed by you related to work on the Camp Lejeune case from October 31st, 2023 to December 10th, 2024.

Is that your understanding?

A. Yes.

Q. And those were provided in response to the subpoena we sent.

Is that right?

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A. Correct.

MS. McKEEVER: I'm going to go ahead and mark these as Exhibit 17.

(Gilbert Exhibit 17, invoices of Kathleen Gilbert, was marked for identification, as of this date.)

BY MS. McKEEVER:

Q. And your billing rate for this case has been \$400 an hour. Is that right?

A. Correct.

Q. And it looks like for a brief period of time in December 2024 you charged 1.5 times the rate of your normal work to work on the bladder cancer report over the holiday.

A. Right.

Q. Understood.
Did you perform any work related to the Camp Lejeune cases prior to October 31st, 2023?

A. As I said, I did some general consulting on the subject, but I didn't write any reports before then.

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Q. And this first invoice dated 10/31/23, that is from Telan, Meltz, Wallace and Eide.

And then the others -- and then switching in March 28th, 2024, it's Bell Legal Group.

Do the first two invoices represent the consulting work you were discussing earlier?

A. Right.

Q. And before this, was there additional consulting work before October 31st, 2023?

A. I believe there was.

Q. And that was related to Camp Lejeune?

A. It was related to general toxicity of some of the chemicals.

Q. And did you charge for that time?

A. Yes.

MS. McKEEVER: Okay.

If those invoices still exist, I'd like to request a copy of those as

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well.

MS. GREENWALD: Counsel, we object as consulting work that has nothing to do with what's going on in this litigation right now. So we would object to that.

We can take that up outside the deposition, but...

BY MS. McKEEVER:

Q. Okay. So it looks like, according to my math, between October 31st, 2023 and December 10th, 2024, you billed approximately \$184,000.

Does that sound about right?

A. I would have to have a calculator to determine that, but...

Q. But you don't dispute the amounts listed on the invoices?

A. No, I do not.

Q. And it looks like you billed about 440 hours.

Does that sound about right?

A. I did not add up all the hours.

Q. But you don't dispute the number

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of hours listed on the invoices?

A. No, I do not dispute that.

Q. Have you performed work in the Camp Lejeune case since December 10th, 2024?

A. Yes.

MS. McKEEVER: So, we request any invoices since December 10th, 2024.

THE WITNESS: I haven't cited any invoices.

MS. McKEEVER: I understand. When they -- when they're created, we request a copy of those.

THE WITNESS: Oh, okay.

BY MS. McKEEVER:

Q. How many more hours do you think you have billed since December 2024?

A. I haven't added them up, but it would be hard for me to guesstimate, so. I would need to look back at my records.

Q. All right. Well, we'll just wait for the invoices.

A. Okay.

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MS. McKEEVER: I think that's about all I have.

After your deposition, you'll be provided with a transcript. We ask that you carefully read, correct, and sign the deposition transcript.

Do you understand?

THE WITNESS: Yes.

MS. McKEEVER: And thank you for your patience today in answering my questions.

And I'll pass the witness.

MS. GREENWALD: So, we need a little bit of time. Give us, like, 10, 15 minutes.

THE VIDEOGRAPHER: The time right now is 4:54 p.m.

We're off the record.

(Recess taken.)

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THE VIDEOGRAPHER: The time
right now is 5:19 p.m.
We're back on the record.

EXAMINATION BY
MS. GREENWALD:

Q. Dr. Gilbert, can you pull up the
bills that we look at? I didn't put an
exhibit number on mine. It's the last
thing you looked at.

MS. GREENWALD: Did you mark the
bills?

MS. McKEEVER: Exhibit 17.

BY MS. GREENWALD:

Q. Exhibit 17.

A. Okay.

Q. I want you to just look at the
first two invoices, 10/31/2023 and
3/28/2024, okay?

A. Okay.

Q. You testified right before the
break that you -- that this was consulting

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work.

Do you remember that?

A. Yes.

Q. Now that you've looked at them, do you have a different opinion --

A. Yeah.

Q. -- what these bills are for?

A. Yes. I mean, I met with the leadership for the Camp Lejeune Bell Legal Group, and some of the information I wrote up at that point went towards that association.

Q. So, like for example, on the first page of number 17 it says: Attended New York City meeting at Weitz and Luxenberg.

Do you see that?

A. Yes.

Q. And that's --

A. Yes.

Q. In fact, I can we were in this room.

A. Yes.

Q. Was that the first time you ever

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met me?

A. Yes, that was the first time I met you.

Q. Okay, thank you.

You can put that aside.

So, you talked a lot today about TCE and the immune system at the cellular level.

Is there information about how those cellular changes are regulated at the molecular and/or the genetic level?

MS. McKEEVER: Objection; form.

A. Yes.

Q. Can you explain that, please?

A. Yes. And I think this lends a lot of support to the importance of the MOA as far as the causation.

So, we know that TCE is metabolized into TCAH, which is metabolized into trichloroacetic acid. That can have a molecular effect on T-cells via a mechanism I'm not going to get into, but it's called a shift base. And we also talked about how that TCA can

1
2 impact some of the other tissues, like the
3 kidney and the bladder, in a molecular way
4 by increasing the expression of c-Myc,
5 which is a oncogene that is found upgraded
6 in a lot of different cancers, and it does
7 that, TCA does that by decreasing the DNA
8 methylation of c-Myc. It's a process
9 that's mutually exclusive.

10 So, most oncogens are kept
11 suppressed because you don't, obviously,
12 want them, and it's kept suppressed by
13 methylation. And we know that TCA,
14 according to some of those reports, can
15 demethylate that particular gene and other
16 genes and cause them now to be expressed
17 when they're not supposed to be expressed,
18 and that is a way to promote cancer.

19 And we also know that TCE has
20 molecular and genetic effects on T-cells.
21 Some of that work was done in my lab, and
22 we showed that TCE can effect cytokines,
23 which is the molecules that the immune
24 system use to communicate with each other.
25 We know that it can commune -- it can

1
2 impact that at the genetic level and that
3 some of that is obviously mediated by,
4 once again, epigenetic changes.

5 So we're saying that some of the
6 long-term effects of TCE can be due to
7 these epigenetic changes which can exist
8 for a long time.

9 And other people have also shown
10 that other epigenetic effects like the
11 increase in what they call microRNAs,
12 which are another way of regulating the
13 immune system, is increased by TCE
14 exposure, and there are a number of
15 reports saying that multiple cancers,
16 including the leukemias, are associated
17 with increased levels of microRNAs.

18 Now, the combination -- the
19 clear-cut association is still being
20 worked out, but it seems very important
21 molecularly and genetically that TCE does
22 have these mechanisms that are generally
23 considered epigenetic that can have
24 long-term impacts on the immune system and
25 on some of the target tissues.

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Q. Thank you.

Okay. I want to ask you just a couple questions about TCE generally, okay.

Is TCE naturally occurring?

A. No.

Q. Okay. So it's a manufactured chemical?

A. Yes.

Q. Are most people in the United States exposed to TCE in their everyday lives?

A. No.

Q. Would you agree that people who live in areas not near industrial sources or contaminated sites are unlikely to be exposed to measurable levels of TCE?

MS. McKEEVER: Objection to form.

A. Yes.

Q. Do you agree that ambient levels in most residential areas are extremely low or undetectable?

A. Yes.

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Q. Are municipal water supplies in the United States tested for TCE?

A. Yes, they are.

Q. And do you think that the vast majority of the population in the United States --

MS. GREENWALD: Well, let me strike that.

Q. Would you agree that the vast majority of the population is not exposed to TCE-contaminated water?

A. Yes.

Q. In your view, is the general population exposed to TCE?

A. No.

Q. I think you said something earlier that -- maybe you did and maybe you didn't, but I want to make sure that the transcript is clear in case I heard it right. I believe you said, or I thought you said that you would have a difficulty of saying whether TCE alone causes bladder cancer.

Is that correct?

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A. I would say that it's more likely than not that it causes bladder cancer.

Q. Okay.

And that's what you actually say in your report as well, right?

A. Yes.

Q. Okay.

All right. The last thing I want to ask you about is Exhibit Number 8 that counsel for the Government showed you which is the IARC classification.

A. I haven't been able to keep these in order. I should have pulled it up before we started.

Here it is.

Q. You have it? Okay.

If you can turn to the second page of this exhibit. You see on the top it says "Cancer Site" on the left, the second -- the second box down?

A. Yes.

Q. And then to the right it says: Cancerogenic agents with sufficient

1
2 evidence in humans.

3 A. Yes.

4 Q. That's referring to
5 epidemiological studies, right?

6 A. Yes.

7 MS. McKEEVER: Objection to
8 form.

9 BY MS. GREENWALD:

10 Q. This wouldn't be the full
11 classification that IARC looks at when
12 it's determining the carcinogenicity of a
13 chemical, right?

14 MS. McKEEVER: Objection to
15 form.

16 A. Correct.

17 Q. Okay.

18 And then next to that it says:
19 Agents with limited evidence in humans.

20 A. Yes.

21 Q. Okay.

22 Is that also relating to
23 epidemiology?

24 A. Yes.

25 MS. GREENWALD: Okay.

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Let me mark as exhibit, are we
on 18 or 19?

THE STENOGRAPHER: 18.

MS. GREENWALD: Can I have a --
this is 18.

(Gilbert Exhibit 18, IARC Bias
Assessment in Case-Control and Cohort
Studies For Hazard Identification, was
marked for identification, as of this
date.)

BY MS. GREENWALD:

Q. So, Dr. Gilbert, if you could
turn to page 7 of Exhibit 18, please. And
on the right-hand column -- so, let me
just step back for a minute.

So, this is a IARC, it's called
"Bias Assessment in Case-Control and
Cohort Studies For Hazard Identification."

Do you see that on the cover
page?

A. I do.

Q. And this is an IARC publication,
right?

A. Yes.

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Q. Okay.

So, if you can go to page 7 now.

A. Okay.

Q. On the right-hand column, this is IARC defining what the various categories mean for epidemiological studies, correct?

A. Okay.

Q. Okay.

If you could read starting with the word "In order" which is --

A. I see it.

Q. -- six lines down which is the definition of "Sufficient."

If you could read that into the record, please.

A. (Reading) In order to reach a determination that there is sufficient evidence that an agent causes cancer in humans, the working group judges that a causal relationship has been established for one or more cancer sites in that a positive association has been observed in the body of evidence that the chance,

1
2 bias, and confounding can be ruled out
3 with reasonable confidence as an
4 explanation for these positive findings.

5 Q. Okay.

6 And that's the "Sufficient,"
7 which was the first column -- well, I
8 should say the middle column --

9 A. Yes.

10 Q. -- of the exhibit we looked at
11 before, right?

12 A. Yes.

13 Q. Okay. So then the next line,
14 the next sentence defines "Limited."

15 Is that right?

16 A. Yes.

17 Q. And can you read what IARC says
18 is limited evidence in humans, please?

19 A. (Reading) When a determination
20 is made that the evidence is limited, this
21 implies that a causal interpretation is
22 credible in that a positive association
23 between exposure and cancer has been
24 observed in the body of evidence, but that
25 chance, bias, or confounding, or some

1
2 combination thereof could not be ruled out
3 with reasonable confidence.

4 Q. Okay.

5 Then they go on to say what's
6 inadequate evidence, right?

7 A. Yes.

8 Q. Can you read that sentence too?

9 A. (Reading) When it is determined
10 that the evidence is inadequate, this
11 implies that the ensemble of research does
12 not permit a conclusion about a causal
13 association.

14 Q. Okay.

15 Can you read the next sentence
16 too? They have two sentences for this
17 one.

18 A. (Reading) This usually reflects
19 one of the following reasons: no data or
20 sparse data were available, or a positive
21 association was not observed in the body
22 of evidence, or findings were positive but
23 were judged to be entirely explained by
24 chance, bias, or confounding.

25 Q. Okay, thank you.

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Now, if you go to the next page, page 8, there's a figure in the middle which says, it says figure 1.1 "Overview of the IARC monograph's evidence, synthesis and evaluation process."

Have you ever seen this type of chart before?

A. I've seen similar charts.

Q. Okay.

Well, this is a relatively new publication, right? This is from 2024?

A. Mm-hm, yes.

Q. So have you seen their prior charts like this?

A. Well, this -- this association is used by many different organizations to talk about causal relationship.

Q. Okay.

I'd like to have you look at the -- in the three square boxes in the middle of that chart, I'd like you to look at "Mechanistic Evidence," please.

A. Yes.

Q. And there's three different

1
2 categories for Mechanistic Evidence. Is
3 that right?

4 A. Yes.

5 Q. All right.

6 And for under "Strong" is strong
7 the highest category for mechanistic
8 studies?

9 A. Yes.

10 Q. And they identify three
11 different ways you can have strong
12 evidence, right?

13 A. Yes.

14 Q. And can you -- or, would --
15 would -- you talked a lot today about key
16 characteristics. Is that right?

17 A. Yes.

18 Q. And is that one of the ways you
19 can establish mechanistic evidence being
20 strong is through key characteristics,
21 according to IARC's classification?

22 A. Yes --

23 MS. McKEEVER: Objection to
24 form.

25 A. Yes, it is.

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Q. And what's your opinion about TCE and mechanistic evidence under the IARC classification?

A. I think it's extremely strong. As I said, it hits many of the key characteristics that have been defined -- that have been described as defining carcinogens.

MS. GREENWALD: I don't have any other questions.

MS. McKEEVER: No questions.

THE WITNESS: I'm sorry?

MS. McKEEVER: You're finished. No questions.

THE VIDEOGRAPHER: The time right now is 5:32 p.m.

We are off the record.

(Deposition adjourned at approximately 5:32 p.m. EDT)

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INSTRUCTIONS TO WITNESS

Please read your deposition over carefully and make any 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. It 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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A C K N O W L E D G M E N T

STATE OF)
 : ss
COUNTY OF)

I, KATHLEEN M. GILBERT, PH.D.,
hereby certify that I have read the
transcript of my testimony taken under
oath in my deposition of April 11, 2025;
that the transcript is a true and complete
record of my testimony, and that the
answers on the record as given by me are
true and correct.

KATHLEEN M. GILBERT, PH.D.

Signed and subscribed to before me this
----- day of -----, 20__.

Notary Public, State of

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C E R T I F I C A T E

I, MARIE FOLEY, Registered Merit Reporter, Certified Realtime Reporter, and Notary Public for the State of New York, do hereby certify that prior to the commencement of the examination, KATHLEEN M. GILBERT, PH.D., was duly sworn by me to testify to the truth, the whole truth and nothing but the truth.

I DO FURTHER CERTIFY that the foregoing is a verbatim transcript of the testimony as taken stenographically by me at the time, place and on the date hereinbefore set forth, to the best of my ability.

I DO FURTHER CERTIFY that I am neither a relative nor employee nor attorney nor counsel of any of the parties to this action, and that I am neither a relative nor employee of such attorney or counsel, and that I am

Marie Foley, RMR CRP in the action.

COURT REPORTER
Registered Merit Reporter
Certified Realtime Reporter
Notary Public
Dated: April 16, 2025

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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate.

The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY.

THE ABOVE RULES ARE CURRENT AS OF APRIL 1, 2019. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

VERITEXT LEGAL SOLUTIONS

COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

Veritext Legal Solutions represents that the foregoing transcript is a true, correct and complete transcript of the colloquies, questions and answers as submitted by the court reporter. Veritext Legal Solutions further represents that the attached exhibits, if any, are true, correct and complete documents as submitted by the court reporter and/or attorneys in relation to this deposition and that the documents were processed in accordance with our litigation support and production standards.

Veritext Legal Solutions is committed to maintaining the confidentiality of client and witness information, in accordance with the regulations promulgated under the Health Insurance Portability and Accountability Act (HIPAA), as amended with respect to protected health information and the Gramm-Leach-Bliley Act, as amended, with respect to Personally Identifiable Information (PII). Physical transcripts and exhibits are managed under strict facility and personnel access controls. Electronic files of documents are stored in encrypted form and are transmitted in an encrypted

fashion to authenticated parties who are permitted to access the material. Our data is hosted in a Tier 4 SSAE 16 certified facility.

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