Exhibit 598

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

IN RE: Case No.
7:23-CV-00897

CAMP LEJEUNE WATER LITIGATION

This Document Relates to:
ALL CASES

- - -

June 30, 2025

- - -

VIDEOTAPED DEPOSITION of MATTHEW J. WEISS, M.D., MBA, held at 1111 Marcus Avenue, New Hyde Park, New York, commencing at 9:00 a.m. EDT, on the above date, before Marie Foley, a Registered Merit Reporter, Certified Realtime Reporter and Notary Public.

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VIDEOGRAPHER:
Jonathan Juarez

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FEDERAL STIPULATIONS

IT IS HEREBY STIPULATED AND AGREED by and between the parties hereto, through their respective counsel, that the certification, sealing and filing of the within examination will be and the same are hereby waived;

IT IS FURTHER STIPULATED AND AGREED that all objections, except as to the form of the question, will be reserved to the time of the trial;

IT IS FURTHER STIPULATED AND AGREED that the within examination may be signed before any Notary Public with the same force and effect as if signed and sworn to before this Court.

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STIPULATIONS

QUESTIONS MARKED

- - -

9:26 a.m. EDT

New Hyde Park, New York

- - -

THE VIDEOGRAPHER: We are now on the record. My name is Jonathan Juarez. I am a legal videographer for Golkow.

Today's date is June 30th, 2025, and the time is 9:26 a.m.

This deposition is taking place at 1111 Marcus Avenue, New Hyde Park, New York, in the matter of In Re Camp Lejeune Water Litigation.

The deponent is Dr. Matthew Weiss.

Counsel, please identify yourselves for the record.

MS. SULPIZIO: Gabrielle Sulpizio and Zach Mandell for the plaintiff.

MR. BU: Nathan Bu for the United States.

MS. SPRAYREGEN: Sharon

Sprayregen also 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.

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MATTHEW J. WEISS, M.D., MBA, 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:

EXAMINATION BY

MR. BU:

Q. Dr. Weiss, can you please state your name for the record?

A. Sure. Matthew John Weiss.

Q. And can you spell your last name?
A. W-E-I-S-S.
Q. You're a physician here at Northwell Health, right?
A. I am, correct.
Q. And our address -- or, Northwell's address is 1111 New Hyde Park, New York -- or, Marcus Avenue, Hyde Park, New York.
A. So, that's where we're located right now.
Q. Okay.
A. Obviously there's a lot of sites at Northwell Health.
Q. What's the address for the primary site where you practice?
A. 1111 Marcus Avenue.
Q. Okay.
My name is Nathan Bu. I'm a trial attorney with the Department of Justice. I represent the United States in this lawsuit.
The purpose of our time together

today for this deposition is to understand the opinions you are offering in this case and how you came to those opinions.

You understand that?

A. Yes.

Q. All right.

To do that, I'll ask you some questions, and all that I ask is that you answer them to the best of your ability.

Do you understand that?

A. I do.

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

A. No.

Q. And you're not taking any medication that might affect your ability to offer complete and accurate testimony?

A. No.

Q. Have you been deposed as an expert witness in other litigation?

A. I have.

Q. About how many times have you been deposed?

A. Probably about half a dozen, maybe -- maybe five or six times.

Q. Were all of those as expert witnesses?

A. They were.

Q. Okay.

Have you ever been deposed as a fact witness?

A. I'm not sure I understand what that means. What do you mean by a fact witness? Meaning?

Q. Okay. A fact witness meaning you're testifying to something you saw or did or saw someone else do.

A. No.

Q. Okay.

You understand that your deposition today is being recorded?

A. I do.

Q. So that means all your answers must be verbal.

You understand that?

A. Yes.

Q. And you're doing a very good job

so far.

You understand that you may request a break unless there is a question pending? If there's a question pending, I'm going to ask that you provide your response before we take a break.

A. Okay.

Q. And my general practice is to try to take a break about every hour, just so you know.

A. Sounds good.

Q. You understand that if a question is unclear, you should explain how it's unclear so I could try to rephrase the question?

A. Okay.

Q. If you answer a question, is it fair for me to assume that you understood the question being asked?

A. I mean, yes. If I'm -- if I don't understand the question, I'll ask you to rephrase it or clarify it for me.

Q. Okay. Perfect.

And if you need to correct an

answer, you will do so, right?
A. Sure.
Q. Okay.
Do you understand that you'll have the opportunity to review the transcript and correct any responses?
A. Yes.
Q. And do you understand that if you correct responses, the United States may reopen this deposition or question you at trial about those corrections?
A. Sure.
Q. Okay.
And you understand that your answers today are being given under oath under penalty of perjury?
A. Yes.
Q. You understand that your testimony today has the same force and effect as if you were testifying in a courtroom with a judge and a jury present?
A. Yes.
Q. All right.
Would you agree that physicians

who assist in legal proceedings, including as an expert witness, should accurately represent their qualifications?

A. Yes.

Q. Okay.

And you'd agree that physicians who assist in legal proceedings should testify honestly?

A. Yes.

Q. Would you agree that a physician who testifies as an expert witness should testify only in areas where they have appropriate training and substantive experience and knowledge?

A. I mean, yes. I guess so. I mean, it depends on what they're being asked to comment on, I mean.

Clarify that. Can you just say that one more time?

Q. Sure.

Would you agree that a physician who testifies as an expert witness should only testify in areas where they have appropriate training and substantive

experience and knowledge?

A. Yes.

Q. Okay.

And would you agree that a physician who serves as an expert witness should ensure that his testimony appropriately characterizes the theory on which testimony is based if that theory is not widely accepted in the profession?

MS. SULPIZIO: Object to the form.

A. Can you repeat it one more time? Sorry.

Q. Sure.

Would you agree that a physician who serves as an expert witness should ensure that his testimony appropriately characterizes the theory on which the testimony is based if the theory is not widely accepted in the profession?

MS. SULPIZIO: Object to the form.

A. I mean, truthfully, I'm not sure I understand what you're asking.

Q. Okay. If a theory --
A. Yeah.
Q. If the physician's testimony is based on a theory that's not widely accepted, should the physician acknowledge that the theory is not widely accepted when giving that testimony?
MS. SULPIZIO: Object to the form.
A. I mean, I -- I think that makes sense. I think then they should probably give both sides of the -- of the theory or, you know, what is believed to be true versus what is -- what is fact versus what is theory.
Q. Okay.
And do you agree to hold yourselves to -- yourself to those standards as best you can when giving your testimony today?
A. Yes.
MR. BU: Okay.
So I'm going to introduce our first exhibit. This will be tab --

or, Exhibit 1, tab 1.

(Weiss Exhibit 1, Specific Causation Expert Report for David Fancher, was marked for identification, as of this date.)

BY MR. BU:

Q. Do you recognize this document?

A. I do.

Q. Okay.

Can you tell me what it is?

A. Well, there's multiple documents here. The first document is the report that I put together on the Mr. Fancher case, and the second document is my curriculum vitae.

Q. Okay.

And this was the report you submitted earlier this year, right?

A. Correct.

Q. Okay.

And the curriculum vitae was also submitted earlier this year?

A. Yes.

Q. Okay.

Can you take a look at the curriculum vitae and let me know when you're ready?

A. Sure.

Q. Do you know if this is your most recent curriculum vitae?

A. I mean, this is -- this is pretty current. The date on this says August 15th of 2024. I may have had a few publications since this time. I publish probably anywhere from five to ten, you know -- actually, last year I published 30 papers. So I publish five or six papers, you know, a month.

Q. Okay.

A. Or three or four papers a month that would be.

So it may be a little outdated in terms of publications, but not grossly.

Q. Do you know if you have a more recent version of your curriculum vitae?

A. I have -- I have a version that I probably updated within the last two or three months.

Q. Okay.

MS. SULPIZIO: And, Nathan, happy to supplement that, if that's what you want.

BY MR. BU:

Q. Does this report contain all of the opinions you formed in Mr. Fancher's case to date?

A. Yes, I believe so.

Q. Okay.

To the best of your knowledge, are any of those opinions incomplete or incorrect?

A. No.

(Weiss Exhibit 2, Plaintiffs' Designation and Disclosure of Phase III Expert Witness With Respect to Kidney Cancer. Materials Considered List For Matthew J. Weiss' Report on Plaintiff David W. Fancher, was marked for identification, as of this date.)

BY MR. BU:

Q. Okay. All right.

I'm handing you what's been

marked Exhibit Number 2.

A. Okay.

Q. Do you recognize this document?

A. Yes, I do.

Q. What is it?

A. I think this is the -- this is the document that designates me as an expert on this case.

Q. Does it list all of the facts and data you considered in drafting your report?

A. This -- this document lists many of the -- like, the background research and the documents that I utilized in formulating my -- my opinion.

Q. Okay.

(Weiss Exhibit 3, Dr. Matthew Weiss - Supplemental Materials Considered List, was marked for identification, as of this date.)

(Weiss Exhibit 4, Dr. Matthew Weiss - Second Supplemental Materials Considered List, was marked for identification, as of this date.)

BY MR. BU:

Q. I'm handing you what's been marked Exhibit 3, and then I'm also going to hand you what's been marked Exhibit 4.

A. Okay.

Q. Do you recognize these documents?

A. I don't recall seeing this -- these actual document papers. I -- I recognize the -- you know, many of the records that are on these documents.

Q. Okay.

And were these records that were provided to you by counsel?

A. Yes.

Q. And --

A. I'll be honest -- I'll be honest, some of these I just -- I don't recall like what this -- this designation is here. Like on -- on Exhibit 3 it says FANCHER000, I don't recognize that.

I reviewed -- I reviewed Mr. Fancher's medical records, and I'm assuming it's -- that's what it

corresponds to, but I'll be honest in that I just don't know what some of these mean there.

Q. Okay.

Do you remember reviewing other witnesses' deposition testimony?

A. Yes.

Q. And do you remember reviewing other witnesses' expert reports?

A. Yes.

Q. Okay.

And would those include the deposition testimony and the expert reports that are on Exhibits 3 and 4?

A. Yes.

Q. Okay.

Are there any documents, deposition transcripts or reports, that you remember reviewing that are not on your materials considered list, Exhibit 3 or Exhibit 4?

You can take some time to look over them.

A. I don't think so. Although

the -- the documents that are listed under Fancher, it's a little unclear to me which these four are.

I reviewed depositions by Mr. Fancher. I reviewed deposition by Mr. Fancher's wife, and I believe I -- I reviewed a deposition by maybe one of his daughters.

I went -- I went through a lot of documents. So to be honest, I can't remember.

Q. Okay. That's fine.

To the best of your knowledge, did you consider any other materials that have not been listed on your materials considered list, Exhibit 2, or -- I'm sorry, Exhibit 3 or Exhibit 4?

A. I -- I don't believe so. No, I don't believe so.

Q. Okay.

Can you turn to page 3 of Exhibit 1, please?

A. Okay.

Q. All right.

Do you see that middle paragraph above the italicized section: In the case of Camp Lejeune exposures?

A. Yes.

Q. Can you read that first sentence out for me, please? Read it into the record.

A. (Reading) In the case of Camp Lejeune exposures, the standard for causation has been defined as sufficient to conclude that a causal relationship exists or sufficient to conclude that a causal relationship is at least as likely as not.

Q. Okay.

So for the lawyers, we would call this a disjunctive statement. There are two standards that are being described, right?

A. There are.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Is your opinion based on the

standard of causation being defined as sufficient to conclude that a causal relation exists?

A. I'm sorry, could you repeat that?

Q. Sure.

Which of these two standards are you applying for your report in Fancher?

MS. SULPIZIO: Object to the form.

A. I believe I'm applying the second: sufficient to conclude that a causal relationship is at least as likely as not.

And I think that in Mr. Fancher's case, it actually -- it actually goes further in that I think that it's -- it's more likely than not.

Q. Okay.

Is your understanding of that definition on the ATDSR assessment published in 2017?

A. It -- it's based upon the ATSDR assessment, but it's also related to

causation statements that I read by Drs. Hatten and Dr. Bird, as well as the exposure calculations that were presented by Dr. Reynolds, as well as existing literature, you know, on exposure levels of these known carcinogens and the relationship to kid -- the development of kidney cancers.

Q. Okay.

So, are you looking to Dr. Hatten and Dr. Bird's report to inform your understanding of what "sufficient to conclude that a causal relationship is at leastly -- at least as likely as not" means?

MS. SULPIZIO: Object to the form.

A. No.

Q. Are you -- and you -- are you relying on the exposure reports to inform your definition for what "sufficient to conclude that a causal relationship is at least as likely as not" means?

MS. SULPIZIO: Object to the

form.

A. I'm not looking for those reports to interpret this statement. I'm looking at those reports to make a medical opinion as to whether I think it is causal or not.

Q. Okay.

Are there other documents that you looked at to inform your definition for what "sufficient to conclude that a causal relationship is at least as likely as not" means?

A. No.

Q. Did you apply any other standard of causation in your Fancher report?

A. Not that I'm -- no, I don't think so.

Q. All right.

Did you consider applying any other standard of causation?

MS. SULPIZIO: Object to the form.

A. I -- I mean, I'll be honest, I -- I clearly am not a lawyer, and I'm

not aware of all the different ways you can interpret causation. I utilized the statements that were in the ATSDR to try to formulate my opinion.

Q. Did you do any independent research on the Camp Lejeune Justice Act in preparing your report?

A. Not on the Camp Lejeune Justice Act, no.

Q. Did you do any independent research on how "at least as likely as not" is used in other scientific texts?

A. I did not.

Q. Did you do independent -- independently research how "at least as likely as not" is used in other medical contexts?

A. I did not.

Q. Would you agree that a correlation between exposure and disease is not necessarily the same thing as exposure causing disease?

MS. SULPIZIO: Object to the form.

A. Say -- say that one more time. Sorry.

Q. Sure.

Would you agree that a correlation between exposure and disease is not necessarily the same as exposure causing disease?

MS. SULPIZIO: Note my objection.

A. I would agree that -- that -- in the medical field we talk about associations, and an association does not necessarily, you know, confirm that it's -- that it's causative, but -- but we use associations all the time to make determinations as to whether we think something is causal.

In the absence of a -- of a prospective randomized trial in medicine, you really can never show causation.

Q. Why can't you necessarily show causation without a prospective randomized trial?

MS. SULPIZIO: Object to the

form.

A. Prospective randomized trials are the -- are the gold standard for how we determine whether, you know, a certain intervention has a -- a certain effect.

In the absence of a prospective randomized trial, you have to utilize other methods to evaluate, and most studies in the medical literature are really retrospective studies, meaning looking back. And in a retrospective study, you could really only show associations between a certain intervention, or in this case a certain exposure, and a potential outcome.

Q. Okay.

Can you explain a little bit more, I guess, the advantages of a prospective randomized trial over retrospective studies?

A. Prospective randomized trials in theory get rid of all biases related to a -- to a study. They're randomized and -- and most of us in the cancer space

would say the only way that you can -- you know, with a hundred percent certainty, you know, show direct causal -- show a, like, inference, if we have an intervention and we want to show is there a benefit to this intervention versus not, the only way you can definitively show a benefit is through a prospective randomized trial. But the reality is is that in medicine, we frequently can't perform prospective randomized trials in order to see if there's an exact correlation.

So for instance, if you even think about Mr. Fancher's case, it's -- it's not like you're going to take a hundred individuals and expose half of them to a known carcinogen like TCE or PCE or vinyl chloride or benzene and then not expose the other half and see which ones develop cancer. That would be, you know, unethical.

Q. So let me put it this way.

Would you agree that prospective

randomized trial is better at controlling for bias in general than retrospective studies?

A. I agree.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Okay.

And would you agree that prospective randomized trials are generally better at controlling for confounding variables than retrospective studies?

MS. SULPIZIO: Object to the form.

A. I would agree with that.

Q. Okay.

Are -- are there other errors or issues that prospective randomized trials are better at controlling for than retrospective studies?

A. Limiting biases, confounders. I think the -- I think that pretty much covers it.

Q. Okay.
What about random error?
A. What about random error?
Q. Would a prospective randomized trial be better at controlling -- or accounting for random error than a retrospective study?
A. It would as long as the prospective randomized trial is -- is powered to show -- is powered properly to show a significant difference.
Q. Would you agree that determining whether an association is causal includes evaluating the quality of the studies reporting an association?
A. I would agree with that.
Q. Okay.
And part of that evaluation should look at whether the study is able to eliminate the role of bias with reasonable confidence?
A. I would agree with that.
Q. Okay.
Would you also agree that part

of that determination is determining whether the study is able to rule out confounding variables?

A. I would agree with that.

Q. And would you also agree that part of that determination includes determining whether a study can rule out random error?

A. Yeah, I would agree with that.

Q. Can you turn to the next page of Exhibit 1 for me, please?

A. Sure.

Q. Do you see the last sentence of both point 2 and point 3 ends "In which chance and biases can be ruled out with reasonable confidence"?

A. Sorry, which page are you on now?

Q. I'm sorry, page 4.

A. It goes on the back, sorry.

Q. Yeah.

A. And where am I looking again one more time?

Q. So at the top of the page for

points 2 and 3, both of those sections end "In which chance and biases can be ruled out with reasonable confidence."

Do you see that?

A. Sorry, point 2 and point 3?

Q. Yeah. If it's easier, let's just stick with point 2 for now.

A. All right.

Q. Can you read point 2 into the record for me?

A. (Reading) A meta-analysis does not provide -- does not provide convincing evidence where if the meta-analysis observes a non-monotonic exposure response relationship, but there is at least one epidemiological study considered to be of high utility occurring after the meta-analysis has been conducted in which an association between the exposure and increased risk of the disease of interest has been found and in which the chance and biases can be ruled out with reasonable confidence.

Q. Okay.

So we had just discussed the role of ruling out chance and biases, right?

A. Correct.

Q. Okay.

And so here, ATSDR is describing ruling out chance and biases with reasonable confidence.

Do you see that?

A. I do.

Q. What does reasonable confidence mean to you?

A. Reasonable confidence to me means that it's within reason, that the -- that -- that it's -- if you're ruling something out with reasonable confidence, what that means is that it's reasonable to have confidence based upon these data. I'm using the word again "reasonable." I'm looking for a thesaurus. But it -- it's not outlandish. It's -- it's within the realm of -- within the realm of reason.

Q. Okay.

Are you aware of any guidelines or other resources that describe the confidence needed to rule out chance or bias?

A. I'm not aware.

Q. Okay.

Would you agree that testing for a statistical significance is one way to rule out chance?

A. I would.

Q. And would you agree that controlling for confounding variables is one way to rule out bias?

A. Agree.

Q. All right.

And when you review medical literature as a physician, do you consider the study's ability to rule out bias?

A. I do.

Q. Okay.

And similarly, when you review medical literature as a physician, do you consider the study's ability to rule out chance?

A. I do.
Q. Do you ever serve as a peer reviewer for medical literature?
A. I do.
Q. How long have you been acting as a peer reviewer for medical literature?
A. Probably 15 years.
Q. When you serve as a peer reviewer for medical literature, do you consider a study's ability to rule out bias?
A. We do.
Q. And when you peer review medical literature, do you also consider the study's ability to rule out chance?
A. I do.
Q. Have you ever peer-reviewed literature that applies an "at least as likely as not" standard?
A. Have I ever peer-reviewed literature? No.
Q. Have you ever published literature that uses an "at least as likely as not" standard?

A. I have -- I have not. The question was have I -- have I ever seen?

Q. Have you ever published literature?

A. Have I ever published? No, I have not published.

Q. Okay.

A. I -- I have seen a paper that utilizes it, but I'll be honest, I think it was actually related to this case, and it was by an expert on the -- on the defense side that I think had published something on this.

Q. Okay.

A. And that's the only time I've ever seen it, to be honest with you, in the medical literature.

Q. When you came across that paper, was that as part of this litigation or as part of your other professional practice?

A. It was part of this litigation.

Q. Okay.

Do you remember the study author?

A. I don't remember, to be honest.
Q. Okay.
But you think they were a defense expert?
A. Yeah. I reviewed so many, you know, documents, you know, over the last -- and when I -- when I started this, I'll be honest, I just can't remember who it was, but I know I took note of it because I just hadn't seen it before.
Q. Other than this one study that was provided to you as part of this litigation, are you aware of any other literature that uses an "at least as likely as not" standard?
A. I am not.
Q. Okay.
Have you ever offered opinions in another case using an "at least as likely as not" standard?
A. I have not.
Q. I apologize because I think I may have asked this already, but other than the ATSDR Assessment of the Evidence,

are you aware of any published guidance in how to apply an "at least as likely as not" standard?

A. I am not.

Q. Are you aware that ATSDR also uses the term "equipoise and above"?

A. I did see that.

Q. Okay.

Have you seen "equipoise and above" used in other scientific contexts?

A. I mean, we don't -- I haven't seen the exact term "equipoise" used. We -- we do have a similar terminology used in some studies which we -- which we essentially would say are like a -- like a noninferiority study where -- where you have a -- a gold standard treatment that's, you know, tried and true and that's the way we treat a certain condition, and there may be a new treatment that comes out, maybe it's less expensive or maybe it, you know, is less toxic to the patient and they'll do a study where they don't necessarily want to

show that the new treatment is better than the old treatment, but they want to show that it's at least as good as the old treatment, and we call that a noninferiority trial.

Q. Okay.

So, for noninferiority, are you measuring, like, the costs and benefits of a new treatment?

A. I mean, sometimes. Sometimes it's -- you know, you're -- you're -- you're evaluating the outcome of the treatment or intervention. Sometimes you're also, you know, weighing not just the cost of the drug, but maybe side effects of the drug and so forth. Like is it -- do you have the same desired effect for the condition that you're treating but with maybe it's less expensive or maybe it's less toxic to the patient or better tolerated to the patient?

So -- so the whole idea of a noninferiority trial is just that you're trying to show from an interventional

standpoint that you're having an at-least-as-good-as-desired outcome.

Q. Is noninferiority looking at more than just the probability that a intervention works?

MS. SULPIZIO: Object to the form.

A. Say it one more time, sorry.

Q. Sure.

Is noninferiority looking at more than just the probability that an intervention works?

MS. SULPIZIO: Object to the form.

A. I think it is -- I think it's more than that. I think it's that you're -- you're trying to show that not only does it work, but it -- but it works as well as the other treatment, or the -- or the -- or the current gold standard treatment.

Q. Okay.

Other than noninferiority, have you seen equipoise or a similar standard

used in any other context?
A. I don't think so.
Q. Would you agree that the term "equipoise" denotes a lack of consensus across the medical community?
MS. SULPIZIO: Object to the form.
A. I don't think I would agree with that. I think equipoise means -- I equate it to they're equivalent. It doesn't mean that we don't either all agree that they're equivalent. So I don't think I -- I agree with that statement.
Q. Okay.
A. There may be consensus that there is equipoise.
Q. Are you familiar with the National Academies of Science?
A. Yes.
Q. What is -- what are the National Academies of Science, or are?
A. I mean, the National Academy of Sciences is essentially a -- a group that, you know, people are accepted to -- into

as members based upon their either academic reputation or advances in their field of expertise and it's a -- it's a relatively prestigious, essentially like a club, like a society.

Q. Have you ever reviewed publications by the National Academies of Science as part of your practice as a physician?

A. I mean, I'm sure I have. I mean, I -- I do literature searches and so forth on topics all the time, and some of those papers will come out of National Academy of Science, some won't, but I'm sure I -- I'm sure I have.

Q. Is the work by the National Academies of Science considered reliable in your field?

A. Yes. I mean, it's -- it's peer-reviewed and...

Q. Is the work by the National Academies of Science considered authoritative in your field?

A. I mean, I wouldn't say it's

authoritative, meaning it's, you know, it's the bible.

I guess I shouldn't say that.

But, I mean, it's -- it's -- it's a well-respected, you know, publication that should be taken into account, but like any publication, you know, has to be interpreted, you know, with a grain of salt and, you know, there's -- there's lots of publications in, you know, very strong journals that maybe are not as well done as we would -- as -- as we would like in the medical community and vice versa. So I wouldn't say that it's authoritative like you have to follow what's in the National Academy of Science, but it should be -- it should certainly be taken into account that it's a peer-reviewed publication and that, you know, it's a well-respected journal.

MR. BU: Okay.

Could we pull tab 30, please?

MS. SPRAYREGEN: 30?

MR. BU: 30, yes.

(Weiss Exhibit 5, National Academies Sciences Engineering Medicine Article Review of the Department of Veterans Affairs Presumption Decision Process, was marked for identification, as of this date.)

BY MR. BU:

Q. I'm handing you what's been marked Exhibit Number 5.

Have you seen this document before?

A. This is a particular document.

I'll be honest, I don't -- I don't -- I don't think I recall reviewing this.

Q. Okay.

A. I don't think I've seen this before.

Q. All right.

So, earlier we were talking about the term "equipoise" and whether or not it indicates a lack of consensus.

Do you recall that?

A. I do.
Q. Okay.
Are you aware that the National Academies of Science concluded that the term "equipoise" denotes a lack of consensus across the medical community?
MS. SULPIZIO: Object to the form.
If you're going to ask him questions about it, I'd like for him to review it.
MR. BU: I'm just asking if he's aware of that.
A. I am not aware of that.
Q. Okay.
And are you aware that the National Academies of Science concluded that the term "equipoise" is inconsistent with current scientific use?
MS. SULPIZIO: Object to the form.
A. I'm not aware of it.
Q. Okay.
Could you turn to page 10 for

me, please?

A. Okay.

MS. SULPIZIO: And if you're going to ask him questions about the document, again I'm going to request that he can look at the full document if he needs it.

BY MR. BU:

Q. Do you see that italicized section at the bottom of 10?

A. I do.

Q. Okay.

Can you read that sentence into the record for me, please?

A. (Reading) The committee concludes that the term "equipoise" denotes a lack of consensus across the medical community and that the term, as required by law to be used in the presumption decision process, is inconsistent with the current scientific use of it.

Q. Okay.

Do you have any reason to

disagree with that conclusion by the National Academies of Science?

MS. SULPIZIO: Object to the form.

A. I mean, I think it would -- I haven't reviewed the entire document. Just taking this one line out of the document, I -- I personally am not in agreement with it. But I don't -- I'd have to take it into -- in context.

Q. Okay.

And why are you not in agreement?

MS. SULPIZIO: Object to the form.

A. Because I -- I don't believe -- when I think of the term "equipoise," this denotes a lack of consensus. You can have a consensus that there is equipoise. So I don't think they're exclusive of one another. I think that if you have two interventions that have the exact same outcome, there is equipoise and you can have a consensus that both interventions

have the same outcome, that -- that -- that doesn't mean that the medical community wasn't able to come up with a consensus. So I think you can have equipoise without -- and have a consensus that there's equipoise.

But again, I -- I haven't seen the entire document, but reading that one line to me, I have -- I have some issues with.

Q. Okay.

Could you have it, sort of, the other way around where the community's thoughts about a certain intervention are just so divided the conclusion is that it's an equipoise?

MS. SULPIZIO: Object to the form.

A. I don't think so. I mean, if -- if you have, let's say, half the scientific community thinks that drug A works, but the other half of the scientific community believes that drug B works, that doesn't mean that they're

equivalent. I mean, one of the groups can be correct. I would find it hard to believe that both could be correct.

But actually, the more I think about it, they -- they could both be correct. Depends on their endpoint of the study. Like a blood pressure medication if your endpoint is just it lowers blood pressure into a normal range, then group A may say this drug works better. But if your endpoint of the study is how well do they tolerate it, do they get lightheaded, do they have to have dose adjustments, is it really expensive, then -- then drug B may be better, considered better by one group.

So I think it depends on the endpoint that you're looking for.

Q. Okay. You can set that aside.

Can you go back to Exhibit 1, your report, please?

A. Okay.

Q. Do you see at the bottom of section 1 the last sentence you state

that: All my opinions in this report are expressed to a reasonable degree of scientific and medical certainty?

A. I do.

Q. Okay.

How do you define "a reasonable degree of scientific and medical certainty"?

A. That -- that based upon my experience and based upon my knowledge, that -- that -- that most physicians would agree with -- with -- you know, or at least more than half of physicians, you know, are -- would agree with the opinions that I'm making, that they're based upon scientific and medical certainty and that that's to a reasonable degree.

Q. Okay.

Have you ever used the phrase "a reasonable degree of scientific and medical certainty" in your academic publications?

A. Not in my academic publications.

Q. Okay.

Have you ever used that phrase in your clinical practice?

A. I don't know if I've ever used that exact phrase, but I may in conversations with patients, you know, say, you know, if they, for instance, you know, ask, you know, what caused this or I may say well, you know, it's hard to say, but with a reasonable degree of medical certainty I think it was this.

Q. Okay.

Can you think of any other instances where you may have used the phrase "reasonable degree of scientific and medical certainty" outside of litigation?

A. No. It always seems to come up in litigation.

Q. Okay.

How, if at all, did the "at least as likely as not" standard influence your application of the term "reasonable degree of scientific and medical certainty"?

MS. SULPIZIO: Object to the form.

A. I think they're -- I think they're two separate issues. I'm commenting on do I think it's as least as likely as not, but I'm making that decision or -- based -- based on a reasonable degree of medical certainty. I think they're kind of separate -- separate issues, but I'm basing at least as likely as not, I'm basing that opinion on a reasonable degree of medical certainty.

Q. Okay.

So would it be fair to say you see these two different standards, "reasonable degree of scientific and medical certainty" and "as likely as not," or "at least as likely as not," as measuring two different things?

MS. SULPIZIO: Object to the form.

A. I mean, I -- I think they're -- I think they can be related, but I think they're, kind of, two separate entities.

Q. Okay.

One is -- would it be fair to say one, "at least as likely as not," is likelihood that the exposure caused disease and the other, "reasonable degree of medical" -- "reasonable degree of scientific and medical certainty," is your level of confidence in that likelihood?

MS. SULPIZIO: Object to the form.

A. Yeah. I mean, I think -- I think one is, like, the standard of what are you looking for, you know. Is it as -- at least as likely as not, you know. You could -- you could change that standard. Are you -- do you want to see that this intervention A is clearly better than intervention B, but you're basing the opinion on all of the data and to a, you know, reasonable -- you know, reasonable degree of scientific and medical certainty.

Q. Okay.

A. So you could -- you could base

lots of things on a reasonable degree of medical certainty.

Q. Okay.

We talked a little bit before about, you know, your publications and -- and your peer review.

Do you recall that?

A. Yes.

Q. Okay.

And you've published articles about cancer generally, right?

A. Correct.

Q. Okay.

And you've peer-reviewed articles about cancer generally, right?

A. Correct, for the most part.

Q. How would you describe cancer to, like, a layperson?

A. Yeah, I mean, cancer is -- is basically a -- an abnormal growth or a tumor that somehow develops the potential to spread and metastasize. We get into conversations all the time between what's the difference between a tumor and cancer.

Well, cancer is a -- is a tumor that has the ability to spread, metastasize and either, you know, harm you permanently harm you or -- or kill you.

The roots of cancer, you know, in -- in theory, all cancer to some extent is -- is genetic, but those genetics are -- you know, we -- we define as all cancers have -- have genetic mutations in them. That doesn't mean that genetics causes the cancer. All cancer cells because there's a disregulation of cell proliferation will frequently have genetic defects in it. And -- and cancer in -- in my opinion, is always related to either environmental exposures, some of which we know about, some of which we don't know about, or genetics. We know that certain patients have a genetic predisposition to developing cancers.

And then sometimes broadly we'll say that the third group is good old-fashioned bad luck, but as I tell patients, I believe that all of those

patients that have good old-fashioned bad luck are probably all environmental exposures or genetics that we just don't know about yet.

Q. Okay.

Are -- are personal health characteristics, like obesity, associated with cancer?

A. They are.

Q. Okay.

And where would you put personal health characteristics like obesity in this dichotomy with environmental genetics?

A. I think obesity is an environmental exposure.

Q. Okay.

And how -- how is obesity similar, or how is obesity an environmental exposure?

A. I think we don't necessarily know. Clearly obesity is associated with increased cancer risks. Lots of different cancers have -- have -- have an increased

risk when patients are obese. I'm not sure we know mechanistically why that is. Could it be the obesity itself or could it be the process by which patients get obese? I'm not sure we really know, to be honest with you.

Q. When we talk about genetics causing cancer, are you mostly referring to or only referring to, like, germline genetics, germline mutations?

A. I -- I am. So, I think, you know, all cancers have somatic mutations in the tumor itself, but the cancers that are truly caused by genetics usually we're talking about germline mutations, meaning every cell in the body has that mutation to begin with. So yes.

Q. Okay.

Would you agree that for all cancer, there's either some genetic -- or, germline or somatic mutation that starts the cancer process?

A. Say that again, I'm sorry.

Q. Sure.

Would you agree that all cancers are ultimately traceable back to some sort of mutation, whether it's somatic or germline?

MS. SULPIZIO: Object to the form.

A. I -- I wouldn't. I would say that all cancers have genetic mutations in them, but that doesn't mean that the genetic mutation is what caused the cancer.

Q. Is the mutation a necessary condition for cancer?

MS. SULPIZIO: Object to the form.

A. Explain that a little bit better to me.

Q. Sure.

A. I'm sorry.

Q. No, it's okay.

Can you have cancer without a mutation?

A. I mean, all -- all cancers have -- have genetic mutations in them but

you don't think that the mutation is always the cause of the cancer.

MS. SULPIZIO: Object to the form.

A. I think it becomes a little bit of semantics. You know, if -- if -- you know, I use ionizing radiation on a cell in order to disrupt the genetics of that cell and cause a cancer, was it caused by the genetic alteration, or was it caused by the ionizing radiation that was applied to the cell?

I think that all cancers have genetic mutations in them. It's just that not all of them are caused by that genetic mutation. Some can be caused by an environmental exposure that leads to a genetic mutation.

Q. Okay.

Does DNA repair play a role in cancer formation?

A. Some. Some cancers, yes.

Q. How so?

A. I mean, the way our cells work,

our cells are dividing all the time at different rates. When cell division occurs, sometimes there are mutations in the DNA as the cell divides and -- and our body has a -- has an inherent mechanism to identify genetic defects that occur when cell division takes place and has a way to kind of protect ourselves against developing cancers.

But for whatever reason, you know, cancers that go on to grow, metastasize, have -- have essentially developed mutations that allow them to -- to circumvent our own protective mechanisms.

Q. Does cell death play a role in cancer formation?

A. It can. The -- the -- the mode of cell death, we believe, may have, you know, some impact in particular on our -- on our immune system and our immune system's ability to recognize abnormal cells.

So for instance, the most

natural process by which cells die is apoptosis, but if they die in another way, for instance with a noxious stimuli like they're -- you know, they're exposed to a carcinogen, that's a different mode of cell death.

Q. Would you agree that the causes of cancer are often multifactorial?

MS. SULPIZIO: Object to the form.

A. Just say the beginning part again, would I agree that all cancers or most?

Q. The causes.

MS. SULPIZIO: Objection.

A. That the causes of cancer are multifactorial?

MS. SULPIZIO: Object to the form.

A. I mean, they can be. Sometimes -- sometimes they're not multifactorial. For instance, if there's germline mutation, that -- you know, that's not multifactorial. That's caused

by a genetic defect. If there's, you know, a -- a clear, clear exposure to a carcinogen, I don't -- I don't think that makes it multifactorial. I think you have one factor that leads to cancer.

So I don't think that they're all multifactorial.

Q. All right. I think earlier we had -- you had talked about, you know, without a randomized control trial you can't have a hundred percent certainty about causation.

Do you recall that?

MS. SULPIZIO: Object to the form.

A. I do recall that.

Q. In your practice as a physician, do you offer 100 percent guarantees to your patients?

MS. SULPIZIO: Object to the form.

A. I mean, there's no hundred percent guarantees in anything. The only thing that I can guarantee the patients,

and I say this to the patients and their families all the time, is that I'm going to -- I'm going to provide the best level of care that I possibly can, that I'm going to be, you know, committed to them, and that I'm going to optimize things for the best potential outcome possible.

That I can guarantee. Many other things I can't guarantee.

Q. Okay.

And why can't you guarantee those other things?

A. Because there are a lot things that I don't control in taking care of a patient. Patients sometimes, you know, present with their cancer with lots of medical comorbidities, maybe even unrelated to their cancer diagnosis, that may make treatments and may make surgery, which is what I perform, more challenging or more higher risk. I -- I do, you know -- I do operations that have a known complication rate. Even though my complication rates, you know, are

published and are at the, you know, I would argue are as good as anywhere else in the country or in the world, there's still a known complication rate.

So for instance, if I do an operation that has a 1.4 percent mortality rate, you know, there's no guarantees. 1.4 out of a hundred will have a mortality after that operation no matter what is done. So I can't guarantee those things.

Q. Okay.

Have you published literature addressing risk factors for postsurgical complications?

A. I have.

Q. Okay.

And have you published articles addressing, you know, the chances of mortality postsurgery?

A. I have.

Q. Okay.

What -- what are some of the risk factors for mortality following surgery, let's say for a liver resection?

A. Some of the -- comorbid conditions. For instance, obviously cardiovascular disease a big one is chronic renal insufficiency or renal failure. There -- those are -- represent very high postoperative, you know, complication rates and even mortality rates. You know, underlying liver disease. Basically medical conditions that make people poor operative candidates. You know, pulmonary status. If they're on oxygen at home before surgery, they frequently don't tolerate, you know, a big abdominal operation very well, and they have a risk for being on a ventilator after surgery and so forth. So the list is, like, pretty clear in terms of medical comorbidities.

We actually, you know, stratify these patients the American Anesthesia Association, the ASA score, and it -- and it puts people in a category for what operative risk they're going to be.

Q. Okay.

And I -- you would agree there's always at least some operative risk for mortality, right?

A. Every operation has at least some operative mortality rate.

Q. Even if the patient has none of the listed risk factors?

A. Correct.

Q. Okay.

Are there -- are there perioperative risk factors for mortality, things that happen during the surgery?

A. I mean, classically we report the risk factors as preoperatively available information 'cause -- 'cause we want to be able to predict. You know, when I sit with patients, I tell them all of my complication rates, you know, leak rates, you know, wound infection rates, mortality rates. So, and usually that assessment is based upon preoperatively available factors.

Q. Okay.

A. So, you know, there -- there

are -- you know, there are publications showing that, like for instance, increased blood loss during the operation has a little bit worse outcome, but -- but those aren't -- because they're occurring during surgery or after, they're not really modifiable. We're -- we're -- we're really looking at preoperative available factors.

Q. Is surgical margin width associated with complications or mortality?

A. On surgical mortality?

Q. Yes.

A. Not in the diseases that, you know, that I regularly take care of. I'm not aware of any data shows that margins would increase surgical mortality.

Q. Okay.

A. It may impact long-term oncologic outcome, meaning recurrence rates, et cetera, but not -- surgical mortality, surgical complications we usually lump together within 90 days of

surgery, and I'm unaware of the margin affecting that.

Q. Okay.

But you are aware of margin affecting the likelihood of recurrence?

A. Correct.

Q. And are wider margins associated with a greater risk of recurrence or narrower margins?

A. Depends on the disease. Some -- some cancer's margins are not that important. Some cancer margins don't really impact local recurrence rates or overall survival. Other cancer's margin is -- the larger margin is associated with a lower local recurrence rate.

But truthfully, it really depends on the cancer that we're talking about.

Q. Okay.

All right. Would you agree that these risk factors, even if they're present in a patient, are not necessarily the cause of a postsurgical complication

or recurrence?

A. Yeah, I would agree with that.

Q. Okay.

For example, if a patient has a cancer recurrence and they had narrow surgical margins, they wouldn't necessarily -- the recurrence would not necessarily be because of the margin width?

MS. SULPIZIO: Object to the form.

A. I mean, I -- I think that's reasonable to -- to say that.

MR. BU: Okay. We've been going about an hour. I think this is a good place to stop.

Do you want to take, like, a five-minute break?

THE WITNESS: All right. Sounds good.

THE VIDEOGRAPHER: The time right now is 10:26 a.m., and we're off the record.

(Recess taken.)

THE VIDEOGRAPHER: The time right now is 10:40 a.m., and we're back on the record.

BY MR. BU:

Q. Dr. Weiss, during the break, did you speak to anyone about your testimony?

A. I did not.

Q. Is there anything that you've testified to today so far that you'd like to clarify or correct?

A. I don't think so.

Q. Okay.

Do you recall earlier we were discussing margin widths and the risk of recurrence for different types of cancer?

A. Yes.

Q. Okay.

And the risk of recurrence will vary depending on the type of cancer being resected, right?

MS. SULPIZIO: Object to the form.

A. The risk of recurrence does depend on the type of cancer that's being

resected, correct.

Q. And would you agree that the risk factors for different types of cancer vary?

MS. SULPIZIO: Object to the form.

A. The risk factors for -- for the development of the cancer or for the recurrence of the cancer or --

Q. For the development of the cancer.

A. Yes.

Q. So for example, the risk factors for skin cancer may be different than the risk factors for lung cancer from the risk factors for liver cancer?

A. Yes, correct.

Q. Okay.

Is most of your clinical practice treating liver and pancreatic cancer?

A. Yes. Yes, most of my clinical practice is liver and pancreas cancer, but I'm a general surgical oncologist as well,

so I treat lots of different cancers.

Q. What are the other cancers that you treat?

A. Anywhere in the digestive tract: colon cancer, gastric cancer, small bowel cancer.

I -- I do treat kidney cancers, although I'm not a urologist. I get involved in kidney cancers when they're related -- in particular when they're related to structures that I operate on all the time. So I -- I do take care of patients that have urologic, you know, malignancies.

One of the places that -- that renal cell cancers can recur is actually in the pancreas. So I've taken care of a lot of patients that have had kidney cancers that have had recurrences in the pancreas as well.

Q. Have you seen any kidney cancer patients who only had a local kidney cancer?

A. Yes.

Q. About what percent of your clinical practice is treating kidney cancers?

A. Probably less -- less than ten percent.

Q. And what percent is treating liver and pancreatic cancer?

A. Probably greater than 60 percent.

Q. Are there difference types of liver cancers?

A. There are.

Q. Okay.

And do they vary depending on, like, where in the liver the cancer arises?

A. Not necessarily. The different type of liver -- liver cancers -- the most common type of liver cancer isn't a liver cancer. It's cancer from someone -- somewhere else that metastasizes to the liver. So that's the most common, kind of, misnomer that it's not a liver cancer; it's a cancer from somewhere else that

recurs in the liver. That's the most common liver tumor.

But then you -- the different type of liver cancers are related to the cells that they come from. So you have hepatocellular carcinoma which come from hepatocytes. You have cholangiole carcinoma which comes from anywhere along the biliary tree or the bile ducts of the liver.

So it's not necessarily location in terms of right/left. It's what cells they come from.

Q. Okay.

I guess physically these different cells are located in different parts of the liver?

A. I mean, they are, but they're -- they're located throughout the entire liver. So you have bile ducts in every part of the liver. You have hepatocytes in every part of the liver. So -- so they're not geographically oriented. They're -- like a hepatocellular carcinoma

can occur in the same part of the liver that a cholangiole carcinoma occurs even though they're coming from different cells.

Q. Okay.

Are there also different types of histologies?

A. There are.

Q. What are some of the histologies that are relevant to liver cancer?

A. So, as I just discussed, cholangiole carcinoma, hepatocellular carcinoma. Sometimes you can have mixed tumors that have a component of both. Those are the big ones. And then you get into some of the more rare types of primary liver tumors, sarcomas.

Q. Do the different types of liver cancers have different prognostic significance?

A. Yes.

Q. And do they have different clinical characteristics?

A. Yes. I mean, they -- they --

they frequently present differently. They frequently look different on -- on radiographic imaging. They -- they behave biologically differently.

Q. Okay.

And do these different types of liver cancers have different risk factors?

A. Yes, they do. Although in the liver it's -- it -- it's -- it's to some extent somewhat simpler in that we know that chronic inflammation, no matter what the cause of that chronic inflammation, predisposes patients, or is a high risk factor for developing liver cancer. So any condition that causes a lot of inflammation in the liver we think then increases the risk of developing a liver cancer.

Q. What's the theory behind inflammation being a cause of liver cancer?

A. I think we don't necessarily understand. You know, theoretically when there's chronic inflammation, the liver --

the liver's entire purpose -- well, not its entire purpose. But one of the main jobs of the liver is to essentially detoxify our bodies, and when it detoxifies our body, it does so by kind of breaking down whatever toxin is -- is there, creating inflammation. And then the liver is one of the few organs in our body that regenerates; kind of grows back. It doesn't grow back. What happens is it kind of swells and increases in volume.

So, we think that because there's inflammation, essentially it's like a noxious stimuli that can alter, you know, cell proliferation and cell division that can then lead to cancer. I'm sure it's completely known though.

Q. Okay.

Are you aware of inflammation being associated with other types of cancer?

A. Yes.

Q. What other types of cancer?

A. I mean, many cancers. Gastric

cancers we know can be related to, you know, infections by bacteria called H pylori that causes chronic gastritis. We know that even something as simple as skin cancers related to some exposure and repeated inflammation related to that.

We -- we believe that pancreatic cancer can have -- you know, can be related to chronic inflammation. We know that patients that have chronic pancreatitis, or inflammation of their pancreas, have a higher risk of developing pancreas cancer. So -- so many, many cancers, we believe, are related to chronic inflammation.

Q. Okay.

All right. So similar to liver cancer, there -- would you agree that there are different types of renal cell cancer?

A. Yes.

Q. And the different types of renal cell cancer have different risk factors?

A. Correct.

Q. And the different types of renal cell cancer also have different prognostic significance?

A. Yes.

Q. And different clinical characteristics?

A. They can.

Q. And would you agree that clear cells are the most common form of renal cell cancer?

A. Clear cell is the most common form.

Q. Can you turn to page 11 of your report for me, please? This is Exhibit 1.

A. Sure.

Q. So, for your report you considered risk factors for renal cancer, correct?

A. Yeah. I mean, for my report basically what I tried to do is develop a differential for potential causes of Mr. Fancher's kidney cancer.

Q. Okay.

And what are those potential

causes?

A. One is, you know, is there a genetic predisposition, meaning is there a -- you know, was this a -- a germline mutation inherited predisposition to developing kidney cancer, and based upon all of the -- the data that I evaluated, there -- there -- there did not appear to be any genetic predisposition to this. He had no family history of it. It was unilateral. It was unilocular. It was -- you know, there was lots of reasons that made it look like it was not genetically caused. He had no family history of Von Hippel-Lindau or anything like that.

Then I looked at other potential risk factors for the development of kidney cancer, like smoking. He is a nonsmoker, a -- an always nonsmoker, a lifelong nonsmoker.

We looked at, you know, did he have some of the other risk factors, like chronic kidney disease, being on dialysis. He had none of those risk factors.

We then looked at -- let's see. Then we looked at, you know, obviously environmental exposures and we -- and we know from -- you know, from -- from these materials that he had had a significant exposure to, you know, multiple volatile organic compounds that are -- that are known and documented to increase a risk of developing a kidney cancer.

Q. Okay.

Were there any other risk factors that you considered?

A. I'm sure there was. I mean, I think I did a -- I would need to look through this, but I did a complete, you know, differential, all the known potential causes of kidney cancer, you know, other environmental exposures, like had he been exposed to heavy metals like cadmium. I don't see anything in the record that showed that he had other exposures. You know, had he been exposed to things like asbestos, I -- I didn't see any evidence that he had, you know, had

been -- had any of those types of exposures.

And I obviously did a thorough review of his -- of his medical history looking for potential, you know, chronic conditions that we know can predispose to kidney cancer, and didn't identify any.

Q. Okay.

Earlier you said Mr. Fancher's kidney cancer was unilocular. That means that there was only one tumor, right?

A. One tumor.

Q. Okay.

And when you were explaining --

A. Can I can I just correct myself?

I should have said unifocal.

Q. And when you were explaining identifying these risk factors, you said "we."

Who is "we"?

MS. SULPIZIO: Object to the form.

A. Yeah, I don't know why I would have said "we." I think I probably meant

like we as a medical community when we put together a differential, we basically look at all known risk factors and look for potential causes of a patient's cancer.

Q. Did you have any staff assist you in drafting your report?

MS. SULPIZIO: Object to the form.

A. I drafted the -- the report on my own.

Q. Okay. All right.

How did you develop this list for your differential?

A. Combination of just experience and knowing what the risk factors for kidney cancer are, as well as, you know, doing another PubMed search for potential causes of kidney cancer, and then reviewing Mr. Fancher's medical records and -- and comparing the two and looking for, you know, what the total -- you know, this is what we do in medicine, we -- when a patient presents with cancer or presents with a condition, we develop a

differential, what are the possible causes of this condition. So I did it based on experiential knowledge. I did it based upon my review of the, you know, literature, existing literature related to clear cell carcinomas of the kidney and renal cell cancers and comparing the two.

Q. Okay.

Were there any guidelines or checklists that you consulted?

MS. SULPIZIO: Object to the form.

A. Not that I'm aware of.

Q. Okay.

A. No, I don't think so.

Q. All right.

Are you offering any opinions about what percentage of kidney cancers are attributable to these different risks?

A. I mean, I -- say that again. Am I --

Q. Sure.

Do you have any opinions about what percentage of kidney cancers are

attributable to the risks that you identify in your differential?

MS. SULPIZIO: Object to the form.

A. Like in general, like, you know, for instance, 5 percent is attributable to smoke and 10 percent -- I -- no, I'm not going to submit exact percentages in terms of, like, each cause and what percent that represents of all renal cell cancers.

Q. Okay.

Do you have a general sense of what those percentages are?

A. I do have a general sense.

MS. SULPIZIO: Object to the form.

A. I do have a general sense.

Q. Okay.

And you may want to pause to allow Gabby time to lodge her objection.

A. Sorry.

Q. That's fine.

MS. SULPIZIO: I know our court reporter got it. She's great.

BY MR. BU:

Q. What percentage of kidney cancers have smoking as a risk factor?

MS. SULPIZIO: Object to the form.

A. I mean, it's a difficult question. My -- you know, my -- again, I'm not going to give you exact percentages, but I would guess of all kidney cancers probably, you know, at least 10 percent or so could probably be attributable to smoking.

Now, what becomes difficult is frequently in patients' histories you're not -- you're not a hundred percent sure of what their true smoking history is, and so it becomes difficult. When you read the literature, you know, what did they quantify someone's smoking history? Did they give pack years; did they not; did they smoke once in college, or did they smoke, you know, for four years while they were in college; have they been smoking for 30 years and just quit?

So in a lot of these exposures, it's just -- it's difficult to -- it's difficult to quantify it as "yes, smoking," "no, smoking."

Q. Okay.

Do you know if obesity is a risk factor for kidney cancer?

A. Obesity can be a risk factor for -- for kidney cancer. I think it's -- it's -- it's a less -- it's a smaller percentage than something like smoking. It's becoming a bit of a challenge to differentiate the obesity risk factors for cancer these days because obesity just in general is on the rise, and so you're clearly seeing, you know, more patients that are presenting with cancers that have obesity, but it's sometimes difficult to differentiate between is the obesity what caused the cancer or is it just there's a cancer occurring in -- in this patient that happens to be obese.

Q. Right.

Do you have a sense of what

percentage of kidney cancer cases have no identifiable risk factors?

MS. SULPIZIO: Object to the form.

A. I would -- I would guess that -- or I would think that -- that as many as half of -- of kidney cancers may not have a -- a clear identiful -- identifiable cause or source.

But that being said, like when I put this differential together, obviously I was in the differential I was looking for identifiable sources and the identifiable source that I -- I identified was the exposure to these, you know, organic compounds.

Q. Okay.

But you agree generally there are not identifiable or unidentified causes of kidney cancer, correct?

A. There are.

Q. Okay.

Would you also agree that the presence of a risk factor doesn't mean

that the patient will necessarily develop kidney cancer?

MS. SULPIZIO: Object to the form.

A. I would agree with that.

Q. And would you agree that the same risk factor may affect different individuals differently?

MS. SULPIZIO: Object to the form.

A. I would agree with that.

Q. So for example, smoking for one pack year may increase patient A's cancer risk differently than a one pack year smoking history for patient B?

MS. SULPIZIO: Object to the form.

A. I agree. I think there -- there -- in particular, there are environmental exposures, there's -- there's clearly some patients that may be extremely sensitive to even a low level of exposure and develop a cancer, and then there may be other patients that can have

a high level of exposure and never develop a cancer. So -- so even in -- with exposure to the same carcinogen, like -- like tobacco, you can have people that, you know, smoke their entire lives and don't die of lung cancer, they die of aortic aneurysms and peripheral vascular disease, and heart attack's probably related to the smoking, but not cancer, and then you can have -- you can have individuals that have a relatively low exposure to the carcinogen. Maybe they just, you know -- you know, had secondhand smoke while they were in -- you know, for years and they developed a cancer.

So I think there is -- there -- there are clearly patients that have increased sensitivity to certain exposures and the development of cancer.

Q. I guess to follow on that example, if the patient only had exposures to secondhand smoke or environmental tobacco smoke, would we be confident that the secondhand smoke is necessarily the

cause of the cancer?

MS. SULPIZIO: Object to the form.

A. I think we would struggle with it and then we would end up categorizing it as an unknown cause.

I think a lot of the quote -- in medicine we use the -- the term, like, idiopathic causes, which really is, we like to joke, it's idiot proof. It's basically when we can't figure out what the cause is, we call it idiopathic. I believe that a lot of the idiopathic causes are actually exposures that we just haven't identified.

Q. When you say "exposures we haven't identified," do you mean, you know, we know what the carcinogens are, we just don't know if people have been exposed, or we don't even know what the carcinogens are?

A. I think both.

Q. Okay.

I guess what's -- what's the

evidence that there are carcinogens that we have not yet identified?

MS. SULPIZIO: Object to the form.

A. I mean, I don't know how you would develop evidence for that, to be honest with you. We're exposed to lots of things every day. We have, you know, increased incidences of cancers in younger and younger patients that we don't have a good explanation for. I think many of us in this field believe that it is likely related to exposure to carcinogenic, you know, materials, food products, et cetera, that we -- that we just -- that we just don't know about yet.

Q. Are you familiar with the phrase "the dose makes the poison"?

MS. SULPIZIO: Object to the form.

A. I mean, I -- I understand what you're saying. I wouldn't say it's a term that I use or that I've heard a lot, but I -- I think I understand the -- the

phrase that you're saying.

Q. Have you used the phrase "dose-response relationship"?

A. Sure.

Q. Is there another phrase that you use to convey a similar idea?

A. No. Dose-response.

Q. Okay.

And how would you explain what a dose-response is?

A. That -- that you have a -- that you have a direct correlation between the amount of dose of something you're giving and -- and the -- the outcome, or the response, and that dose-response, you know, can be linear, it can be -- you know, there's mathematical equations to see what that relationship is.

But, yeah, dose-response is you give a dose, you get a response, and -- and the mathematical model of what dose leads to what response.

Q. Have you looked at any of those mathematical models as part of your

practice?

A. Mathematical related to, like, dose-response curves?

Q. Yeah.

A. I mean, I'll occasionally see dose-response curves, for instance if a new drug comes on to the market, you'll frequently see, you know, the early phase trials are usually more dose-response 'cause it's -- they're less -- they're looking at what levels of drug, you know, they get the maximal response and then when going to a higher level, they don't -- they don't get as much of a response. So -- so it does come up in reviewing literature on new drugs in particular that are coming on to the market.

Q. Okay.

Have you reviewed any mathematical models for dose-response in cancer risk?

A. I don't think so.

Q. Okay.

Have you reviewed mathematical models about the cancer slope factors?

A. I don't -- I don't think so.

Q. Okay.

Earlier you were describing the term "idiopathic."

Can you define what "idiopathic" means to you for me one more time?

A. Yeah, so, idiopathic to me is like a diagnosis of exclusion, meaning you look at all the known causes of this condition and you rule all of them out, and if you can't -- if -- if you rule everything else out, then we call it idiopathic, meaning we don't know what caused it.

Q. In your experience treating kidney cancer patients, are unexplained causes common?

MS. SULPIZIO: Object to the form.

A. I think it's common to have a kidney cancer that -- that is -- that is classified as idiopath -- idiopathic and

unknown cause.

You know, again it -- like as it pertains to Mr. Fancher's case, we -- when I reviewed his medical records and I reviewed, like, he -- he has a known carcinogenic exposure. So I don't think idiopathic would -- would relate to his case.

But -- but in general, there is a -- there is a -- you know, a group of patients, a good group of patients, 50 percent or so, that -- that don't have an identifiable cause of their kidney cancer.

I personally believe that -- that many of these you -- you could find it eventually, maybe it's a toxin that we're unaware of. Maybe it's an exposure that they had that we're unaware of. But idiopathic doesn't mean there is no cause. Idiopathic means we can't figure out what the cause is.

Q. Okay.

So, I guess would it be fair to say we don't know the universe of all

potential causes of kidney cancer?

MS. SULPIZIO: Object to the form.

A. Correct.

Q. And so when you're going through your differential, you're not looking at the universe of all potential causes; you're looking at the identifiable causes?

MS. SULPIZIO: Object to --

BY MR. BU:

Q. Is that fair to say?

MS. SULPIZIO: Sorry.

Object to the form.

A. I'm looking at all the known causes of kidney cancer, and then if there's -- if you can't identify a known cause, then it get lumps -- it gets lumps into that category of "unknown" which we call idiopathic.

Q. Okay.

Would it be fair to say that kidney cancer is fairly common compared to other types of cancer?

MS. SULPIZIO: Object to the

form.

A. I mean, I wouldn't say it's fairly common compared to others.

Q. Okay.

A. I think there's, I don't know, six or 8,000 cases a year, 8,000 cases a year. Incidence rate is, I don't know, 1.8 percent. So like everyone has about a 1.8 percent lifetime risk of developing kidney cancer. But, you know, obviously there's many more common cancers. Breast cancer's more common. Lung cancer's more common.

Q. Okay.

A. So I don't know how to answer that, to be honest with you. I mean, I wouldn't say it's as common as a lot of other cancers. But, yeah it's -- it's more common than others. In the middle.

Q. You said about 8,000 cases a year?

A. I think.

Q. Okay.

A. Approximately.

Q. All right.

MR. BU: Sharon, can we pull out tab 17, and this will be Exhibit 6.

(Weiss Exhibit 6, National Cancer Institute article Cancer Stat Facts: Kidney and Renal Pelvis Cancer, was marked for identification, as of this date.)

BY MR. BU:

Q. Are you familiar with the National Cancer Institute's SEER data?

A. I am.

Q. Okay.

And what is SEER?

A. SEER is essentially a nationwide database that tracks, you know, cancer, cancer incidences, cancer outcomes. It's utilized a lot for publishing papers on outcomes.

Q. Okay.

Is SEER data considered reliable in your field?

A. It is.

Q. All right.

Can you turn to page 6 for me, please? And I apologize that they're not actually numbered.

A. Okay.

Q. But it will list the common types of cancer at the top.

A. Okay.

Q. Do you see that?

A. Common types -- hold on a second. I think I'm looking at the correct page.

Common types of cancer, yes.

Q. All right.

A. Yep.

Q. And about how many new cases in 2025 is SEER report?

A. About 80,000.

Q. Okay.

A. Yeah.

Q. Does that number sound about right now?

A. It does. Now -- now that you say this, when I said -- when I was quoting 8,000 cases a year, I'll be honest

with you, I was thinking of deaths.

Q. Okay.

A. I was thinking of mortality related to kidney cancer because unfortunately that's the way I think. So I should correct myself. It was -- I was saying about 8,000 deaths related to kidney cancer. Incidence just -- a lot of kidney cancers are -- go on to -- you know, are cured these days.

Q. Right.

Is kidney cancer considered to be more treatable than other types of cancer?

MS. SULPIZIO: Object to the form.

A. I mean, I don't know how to -- I don't know how to, like, relate it to other cancers, to be honest with you. I mean, kidney cancer if it's localized is -- is treated with surgery. But I don't know how to compare it to other cancers, to be honest with you.

We could, you know, look at

five-year survival rates and so forth and compare it, I guess.

Q. And I think earlier you had said that there's about a 1.8 percent lifetime risk of developing renal cancer.

Do you recall that?

MS. SULPIZIO: Object to the for.

A. I believe so. I think it's in that ballpark.

Q. Yeah.

And can you turn to page 2 of that exhibit for me, please.

A. Okay.

Q. And SEER also reports the lifetime risk of developing cancer, right?

A. I believe so.

Q. Okay.

And SEER also reports the lifetime risk of kidney and renal pelvis cancer being 1.8 percent?

A. I'm sorry, say that again. I'm just --

Q. Sure.

A. I was looking at the sheet when you were talking. I apologize.
Q. No, that's fine.
Do you need more time to review that page?
A. No, it's okay.
Q. Okay.
SEER also reports that approximately 1.8 percent of men and women will be diagnosed with kidney and renal pelvis cancer at some point during their lifetime, correct?
MS. SULPIZIO: Object to the form.
A. They do.
Q. Okay.
Do you have any reason to disagree with that figure?
A. No.
Q. So this means approximately for every 100,000 people, about 1,800 will develop kidney cancer at some point during their lifetime?
MS. SULPIZIO: Object to the

form.

A. Yes, 1.8 percent.

Q. Okay.

And the 1.8 percent lifetime risk includes all potential causes of a kidney cancer, whether known or unknown, right?

A. That's correct.

Q. Would you agree that a reliable methodology for determining the cause of disease should take into account the background risk?

MS. SULPIZIO: Object to the form.

A. Yeah, I think so.

Q. Okay.

And here the background risk would be approximately 1.8 percent?

MS. SULPIZIO: Object to the form.

A. Are you saying in -- in relation to this case 1.8 percent? Just rephrase that for me, sorry.

Q. Sure.

Would you agree the background risk for renal cancers is about 1.8 percent?

MS. SULPIZIO: Object to the form.

A. I would agree that -- that -- that for men and women, their lifetime risk of developing a kidney cancer is 1.8 percent.

Now -- now, that being said, the -- the vast -- that's lifetime risk and -- and most kidney cancers are diagnosed later in life, people in their 60s, people in their 70s.

So the background of all-comers developing it in their entire lifespan is 1.8 percent, yes.

Q. Do you have any opinions about the likelihood of someone developing cancer before the age of 40 -- sorry, developing renal cancer before the age of 40?

A. I think it's highly unusual. I said this in any statement that I

produced. If you look at all -- all patients develop -- developing kidney cancer -- being diagnosed with kidney cancer before the age of 40 is pretty rare, like single-digit percentage, less than 5 percent chance. So it -- it is a bit of an outlier. Many of the patients develop kidney cancers, you know, in their sixth and seventh decades of life. That's much more common. But to develop it before the age of 40 is -- is pretty rare.

Q. I guess setting -- do you have any opinions about what the ordinary background risk would be for someone -- or, I guess how -- how rare would it be to develop cancer before the age of 40?

MS. SULPIZIO: Object to the form.

A. I mean, if you look at -- if you look at the number that -- that the general population has a 1.8 percent lifetime risk, and over 95 percent of those are going to be diagnosed after the age of 40, then I have to do the math real

quick, but -- but you're talking about a very, very, very small percent chance that it's going to occur in the first four decades of life.

Q. So roughly, like, 5 percent times 1.8 percent?

MS. SULPIZIO: Object to the form.

A. Probably even lower because --

Q. Okay.

A. -- a lot of the early kidney cancers that are diagnosed are also in genetically predisposed patients, patients with tubular sclerosis, Von Hippel-Lindau disease, again which -- which in the case of Mr. Fancher was -- was not present. So you're getting into a very, very, very small percent of developing a cancer without any other, you know, genetic predisposition before the age of 40.

Q. The likelihood of someone developing renal cancer before the age of 40, absent a genetic predisposition, is not zero percent though, correct?

MS. SULPIZIO: Object to form.
A. Correct.
Q. Okay. So let me put it this way.
Do you have an opinion about how much more than zero percent that likelihood is?
MS. SULPIZIO: Object to the form.
A. Rephrase that for me. I'm sorry.
Q. Sure.
A. Yeah.
Q. So, the likelihood of someone developing renal cancer before the age of 40 absent a genetic predisposition is greater than zero, right?
A. I agree.
Q. Okay.
And it's probably less than five if we think that all cancers -- for all cancers, only 5 percent are diagnosed before the age of 40, right?
MS. SULPIZIO: Object to the

form.

A. I think it's less than five.

Q. Right.

A. Correct.

Q. So do you have an opinion about where between zero and five that likelihood is?

MS. SULPIZIO: Object to the form.

A. Yeah, I mean, I -- I -- I'll be honest, now I'm getting confused between just like the general population and Mr. Fancher's case, you know, I'll be honest.

Like you -- you basically have a very young individual from a kidney cancer standpoint, less than, you know -- under the age of 40 who develops a kidney cancer that has absolutely no genetic predisposition to developing a kidney cancer. The chances of that happening have to be, like, far under one percent, and in his case, again, you -- you look for potential causes and he has had exposure to -- to known carcinogenic

compounds related to kidney cancer. So I think, like, for another patient who's under the age of 40 with no genetic predisposition, their chances of -- and -- and no environmental exposures, if you have that patient, the chance of them developing a kidney cancer are extraordinarily small.

Q. Okay.

But you're not quantifying the risk for that other kidney cancer patient other than extraordinarily small?

MS. SULPIZIO: Object to the form.

A. I mean, it's not zero, but it's far, far, far under one percent. It's -- it's a very small number.

Q. Okay.

Earlier you mentioned you reviewed some other expert's reports.

Do you recall that?

A. I did.

Q. Okay.

And you reviewed the general

causation report for Dr. Benjamin Hatten; is that right?

A. I did.

Q. And Dr. Steven Bird?

A. I did.

Q. Okay.

Did you review the report of Dr. Timothy Mallon?

A. I don't recall Dr. Timothy Mallon.

Q. Okay.

A. I'll be honest, I reviewed a lot of documents, and I -- and I'm struggling to remember the names and correlating it with what I read, but you'd have to refresh my memory.

Q. Sure.

A. I don't remember that name.

Q. So, Dr. Hatten and Dr. Bird both issued general causation reports, correct?

A. Correct.

Q. Okay.

And what -- what's your understanding of the opinions that Dr.

Hatten and Dr. Bird were offering in their reports?

A. I mean, in their reports, they -- they -- and I utilized those reports for a couple of things. They -- they first, kind of, explain, you know, some of the background of, you know, TCE, PCE, you know, benz -- benzene, vinyl chloride, and the mechanism of believed carcinogenesis, how they're metabolized, what the byproducts are that can lead to cancer, and then -- and then they went through and actually, you know, looked at the exposure levels for Mr. Fancher and correlated it with, you know, the -- the development of a kidney cancer and -- and also utilized obviously some of the other papers that are out there, like -- like the paper by Bove that actually looks at patients at -- or looks at personnel and family members at Camp Lejeune and showed that the -- that the exposure that Mr. Fancher was, you know -- had been exposed to was sufficient enough and high enough

that it -- that it increases his risk of developing a -- a kidney cancer.

Q. Okay.

Were there other experts who issued reports that you read describing exposure levels or background mechanisms or the Bove studies?

A. I mean, I -- I reviewed Dr. Reynolds' work, but that was more related to the calculations of Mr. Fancher's exposure.

I -- I don't recall any others, but that doesn't mean that I didn't. To be honest, I just...

Q. Do you recall reviewing a report by Dr. Michael Freeman?

A. I recognize the dame -- the name, but I don't remember the exact -- I don't remember the report.

Q. Okay.

Do you recall reviewing a report by Dr. Kathleen Gilbert?

A. I definitely had a report by Dr. Gilbert, but I don't remember the

specifics of it.

Q. Is there any particular reason you focused on the reports of Dr. Hatten and Dr. Bird?

MS. SULPIZIO: Object to the form.

A. No. I mean, they -- you know, the way they described the, you know, carcinogenesis and everything from a background perspective just was, you know, honestly, seemed appropriate, easy to read, and -- and I agreed with what they were saying. So those are the ones that I -- that I remember the most, but I wouldn't say that I focused in on just those two. I -- you know, I read through, you know, several reports and certain ones, kind of, stand out in my mind now and -- and others don't, to be honest with you.

It's like when I, you know -- it's like when studying for a test. If you take in all the material and some of it sticks with you and you formulate your

opinion based upon the material, but you can't necessarily cite back and say oh, yeah I got this idea from this, you know, source or...

Q. And you talked about using Dr. Hatten and Dr. Bird's reports regarding I think you said exposure levels? Is that what you had said earlier?

A. Mostly their causation dealt with, you know, mechanisms. The -- the actual levels I really relied on Dr. Reynolds' report for, in particular for Mr. Fancher. But I really relied on -- on hers the most and then compared those levels, you know, to some of the peer-reviewed publications like the Bove papers.

Q. Okay.

When you were talking about the levels in the Reynolds report, you're saying -- you're referring to the levels that Mr. Fancher was exposed to, correct?

A. Correct.

Q. Okay.

And are you aware of levels of exposure that are associated with kidney cancer risk?

A. I am because -- because I reviewed the Bove papers that talk about, you know, levels of exposure and dividing the exposure levels into, kind of, a low -- lower exposure level or a moderate exposure level and then correlating that with -- in that -- in that paper it was mortality rates related to renal cell cancer.

Q. Okay.

Other than the Bove studies, are there other resources that you considered in determining what levels of exposure can cause kidney cancer?

A. I mean, it -- it was tough because, you know, a lot of the early reports on some of these compounds were more, kind of, anecdotal, more, sort of like, individual cases. And in a lot of those cases, they didn't really -- they didn't have access to actual levels to

compare it to.

But specifically for Mr. Fancher, I relied very heavily on, you know, Dr. Reynolds' calculations and -- and the causative statements, you know, by Drs. Hatten and Bird and, you know, I read those, I read those with a -- with a critical scientific mind in place and -- and made the determination that I was in agreement with those reports based upon my experience and -- and to a high degree of scientific and medical certainty.

Q. When you say "causative statements," are you referring to Dr. Hatten and Dr. Bird's opinions regarding all four VOCs?

A. Yes.

Q. And you agree with those opinions regarding all four VOCs?

A. I do.

Q. Okay.

Did you do any of your own research or analysis as to whether those VOCs can cause kidney cancer?

A. So, I did, you know, usual PubMed search which is what we utilize the most in medicine, and I, you know, searched for those compounds, combined those terms with renal cell cancer, clear cell cancer of the kidney. You know, yeah, did my own research, but pretty much relied on PubMed for that.

Q. When you were reviewing Dr. Hatten's report, was there anything you recall disagreeing with or doubting?

A. I don't really recall, to be honest. I -- I mean, I read his report. I seemed to be in agreement, you know, with what he was saying. I don't remember any specifics where I said -- I thought I disagree with this.

Q. Okay.

Do you recall disagreeing with anything in Dr. Bird's report?

A. I don't.

Q. Okay.

Is the level of our understanding about the relationship

between TCE and kidney cancer the same as it is for our understanding of PCE, vinyl chloride, and benzene and kidney cancer?

MS. SULPIZIO: Object to the form.

A. I think the -- I think the data's, you know, a little more mature for TCE than for PCE, vinyl chloride or benzene. I think, you know, there is data that suggests that those other organic compounds can increase the risk of multiple malignancies, including kidney cancer, but I think TCE, you know, for whatever reason, seems to be the most mature and the most developed.

Q. When you say "most mature and most developed," are you referring just to the number of studies or quality of those studies? Can you explain what you mean by "most mature"?

A. Mostly the number of studies and -- and the time period that those studies, you know, have been going on for.

Q. And why is the time period

relevant?

A. Just because, as you can imagine, a -- you know, a potential compound that causes a cancer, like in the case of Mr. Fancher, sometimes there's a latency period. And so -- so if you're going to -- if you're going to -- you know, if you're going to follow a substance and look for a car -- to think that there's a carcinogenic exposure, there needs to be time to follow those patients.

And so I think it -- you know, I'll be honest, I don't know when they first, you know, identified some of these substances like, you know, benzene and vinyl chloride as a carcinogenic, but for whenever reason, I think the number of papers in the time period they were able to follow patients and identify it as a potential cause adds to the literature, adds to the, you know, the argument that this is a truly carcinogenic substance.

Q. Do you know if the literature is

more consistent for TCE than for the other chemicals?

A. I don't know.

Q. Are you familiar with IARC?

A. I am not.

Q. Okay.

You didn't review an -- an IARC monograph on TCE or PCE for your report?

MS. SULPIZIO: Object to the form.

A. I'll be honest with you, I would need to look at it and see if I reviewed it and, you know, there's a lot of -- in the -- to be honest, when you review these literature that we're looking at, there's a lot of acronyms, ATSDR, TCE, PCE, and IARC, I-A-R-C. My guess is I reviewed something that's related to IARC, but I just -- but I don't know it by that terminology and it's -- it's hard to recall. But I would -- you know, I'm happy to look at it if you want to.

Q. Okay. No, that's fine. We can come back to that.

Did you review the ATSDR Assessment of the Evidence?

A. I did.

Q. Okay.

MR. BU: Sharon, can we pull tab 6, please?

(Weiss Exhibit 7, ATSDR Assessment of the Evidence For the Drinking Water Contaminants At Camp Lejeune and Specific Cancers and Other Diseases January 13, 2017, was marked for identification, as of this date.)

BY MR. BU:

Q. So, I'm handing you what's been marked Exhibit 7.

A. Okay.

Q. Do you recognize this document?

A. I do.

Q. Okay.

And what is it?

A. It's the ATSDR Assessment of the Evidence for Drinking Water Contaminants At Camp Lejeune and Specific Cancers and Other Diseases.

Q. All right.

And why did you review this article when you were preparing your report?

MS. SULPIZIO: Object to the form.

A. Because it was a -- it -- it was a, you know, a government study that dealt specifically with levels of contaminants that were known at Camp Lejeune and specific cancers and other diseases. To me, the ATSDR seemed like -- like the most appropriate document to review for such a case.

Q. Okay. So your understanding -- sorry.

So your understanding is for the assessment of the evidence, ATSDR was looking at these contaminants at the levels at Camp Lejeune; is that right?

MS. SULPIZIO: Object to the form.

A. I mean, my -- I was -- the ATSDR reported on the contaminants in the

drinking water at Camp Lejeune, and that's what I was utilizing it for. And then to me it seemed like the most appropriate source to -- to look if there was environmental exposures at Camp Lejeune.

Q. Okay.

ATSDR issued several publications about Camp Lejeune, right?

A. Mm-hm.

Q. Okay.

Is your understanding that the assessment of the evidence is related to the levels of contaminants at Camp Lejeune?

MS. SULPIZIO: Object to the form.

A. Sorry, say that again.

Q. Sure.

Let's table that. We can come back to it.

Can you turn to page 13 for me, please?

A. Okay.

Q. So, this is a table with the

overall summary of the evidence for ATSDR; is that right?

MS. SULPIZIO: Object to the form.

A. Yes.

Q. Okay.

And what was ATSDR's conclusion as to PCE in its assessment of the evidence?

MS. SULPIZIO: Object to the form.

A. I mean, truthfully, I would -- I -- I'm looking at one page of this, and this is a huge document. So I want to make sure I don't -- you know, I can tell you what it says on this page 13, but, you know, I -- I think I just need to make sure that I -- that it's in context of the entire document.

So what -- what's the question again? Say it one more time.

Q. Sure.

What was ATSDR's conclusion as to PCE in its assessment of the evidence?

A. For PCE --
Q. Yeah.
MS. SULPIZIO: Object to the form.
A. For PCE, for PCE it says below equipoise evidence for causation.
Q. Okay.
And then for TCE, what was the conclusion?
MS. SULPIZIO: Object to the form.
A. It says for TCE it says sufficient evidence for causation.
Q. All right.
And is your understanding that these conclusions are related to the levels of contaminants at Camp Lejeune?
MS. SULPIZIO: Object to the form.
A. I think these are based upon the -- like again I'm looking at just this one figure out of context of the entire document, but I think these are -- these are trying to summarize the existing

literature on what the evidence is, not specifically looking at the Camp Lejeune data. It's -- it's talking about what is the evidence in the -- in the published literature and summarizing that.

Q. Okay.

And so that summary literature may include studies where the levels of contamination were different than the levels of contamination at Camp Lejeune, right?

A. They could be.

Q. Okay.

You mentioned you conducted a PubMed search when preparing your report; is that right?

A. I did.

Q. All right.

And did you do a PubMed search for PCE and renal cancer?

A. I'm sure I did, yes. I mean, I used -- I did a PubMed search for all of the, you know, volatile organic compounds that are listed and kidney cancer.

Q. Okay.

Do you recall whether your search identified any studies that found no association between PCE and kidney cancer?

MS. SULPIZIO: Object to the form.

A. I'll be honest with you, I don't recall every study that I found doing the PubMed search, but -- but I would think that if I found one that I thought was a well done study that I would remember it.

You know, obviously when you do a PubMed search, lots of studies get pulled, and then that's kind of what we do is we -- you know, we look at the study, we look at shortcomings of the study, we look at holes in the study, and we -- you know, we utilize that to decide, you know, is it a good study or is it a bad study. That's why, you know, we -- we generally stick to peer-reviewed publications. We generally stick to, you know, peer-reviewed publications that are in

high-impact journals because in theory the peer review process is a little more stringent.

But I don't recall seeing a, you know, a specific paper that showed that there was no association, but -- but to be honest with you, you know, I -- I don't remember every paper that I pulled.

Q. Okay.

A. I think every paper that -- that comes out needs to be judged separately on its -- on its merit.

Q. Okay. Can you -- sorry, we're still on 7.

A. Okay.

Q. Can you turn to page 22 for me, please? I think this is the very last page of the kidney cancer section.

A. Okay.

Q. The last page of the exhibit.

A. Okay.

Q. Okay.

Do you recall reviewing the ATSDR's analysis of the PCE literature?

A. I don't specifically recall reviewing it. I'm sure I did 'cause I read this document, but I can't say I specifically remember reviewing it.

Q. Okay.

And this paragraph about PCE identifies several different studies; is that right?

A. It does.

Q. Okay.

And do you see in the middle of that section there's a sentence that reads: No increased risks were observed in the Lipworth et al. 2011, Vlaanderen et al. 2013 and Silver et al. 2014 studies?

A. I see that.

Q. Okay.

Did you review those articles?

A. I don't specifically remember reviewing them.

Q. Okay.

Do you recall whether they came up in your PubMed search?

A. I don't recall. It doesn't mean

they didn't, but I just don't remember.

Q. Okay.

All right. You can set that aside.

Can you go back to your report on page 11 -- or, no, I'm sorry. Page 9.

A. Okay.

Q. All right.

And in the first full paragraph do you see that sentence: Mr. Fancher's exposure is similar to many of the above listed levels that have been found to be correlated with kidney cancer?

A. I'm looking for it. Sorry, it's in --

Q. Sorry, the first full paragraph at the top of the page.

A. Here we go, yep.

Q. And there are 23 levels that you list in your report, right?

A. Correct.

Q. Okay.

And you're getting these levels from I think it's Dr. Bird's report; is

that right?

A. Not all of them were -- you mean the -- some of these levels are -- are from the -- and they're cited next to it. You know, like cumulative exposure, number 8 "Cumulative Exposure PCE," that's from the Bove study. So these levels are -- I'm citing where these levels have been published as being...

Q. Okay. Let me ask this.

Did you identify the Bove article yourself, or how did you -- how did you come across the Bove article?

MS. SULPIZIO: Object to the form.

A. The Bove article was shared with me by the plaintiffs' attorneys.

Q. Okay.

And how did you develop this list of 23 levels?

MS. SULPIZIO: Object to the form.

A. By using the literature and using the Bove article, as well as other

papers, and just basically putting together a list of, you know, exposure levels from the literature that looked to be, you know, consistent with carcinogenesis.

Q. Okay.

So, the first level comes from a study by Aschengrau. Is that right?

A. Correct. That was a study from Massachusetts, I believe.

Q. Okay.

Did you review the Aschengrau article?

A. I did. I remember reviewing it. It's been a little while since I reviewed it, to be honest, but I did review it.

MR. BU: Okay.

Actually, before we do that, let's take another short break.

THE VIDEOGRAPHER: The time right now is 11:43 a.m., and we're off the record.

(Recess taken.)

THE VIDEOGRAPHER: The time

right now is 11:55 a.m., and we're back on the record.

BY MR. BU:

Q. Dr. Weiss, did you discuss your deposition testimony with anyone during the break?

A. I did not.

Q. Is there anything that you've testified to today that you'd like to clarify or correct?

A. I don't think so.

(Weiss Exhibit 8, Aschengrau study, was marked for identification, as of this date.)

BY MR. BU:

Q. All right. I'm handing you what's been marked Exhibit 8.

Do you recognize this document?

A. I do.

Q. And what is it?

A. This is a paper by Aschengrau on cancer risk and TCE-contaminated drinking water in Massachusetts.

Q. Okay.

And this is the same article that is cited for that first level in your report; is that right?

A. Yes.

Q. And you may want to have both of these 'cause we're going to do some jumping back and forth between Exhibit 1 and Exhibit 8.

A. Okay.

Q. All right.

So, on page 8 of your report, the first level that you list is 27 to 44 milligrams of PCE; is that right?

A. Correct.

Q. Do you recall where that level comes from in Aschengrau, or what it reflects?

MS. SULPIZIO: Object to the form.

A. I mean, I don't recall where exactly in the paper I found it, but it reflects, you know, a -- a total exposure of 27 to 44 milligrams of -- of PCE.

Q. Okay.

Can you turn to page 289 of Exhibit 8?

A. Okay.

Q. All right.

And in the left-hand column the first full paragraph beginning "A total of 5.7 percent."

Do you see that?

A. I do.

Q. Okay.

Does that paragraph describe a 27 to 44 milligram range?

MS. SULPIZIO: Object to the form.

A. I'm sorry, I need to read it.

Q. That's fine.

A. (Witness reads document.)

Yeah, so -- so, yes. What this is, the 27 to 44 is -- is the 90th percentile of exposed, you know, controls.

Q. Okay.

And can you turn to the next page of that article, page 290, and do you see the section titled "Discussion"?

A. I do.
Q. All right.
And do you see that last sentence for this -- the first paragraph of "Discussion"?
A. I do.
Q. All right.
And so, Aschengrau and the co-authors concluded that no kidney cancer cases were considered exposed when latency was taken into account; is that right?
MS. SULPIZIO: Object to the form.
A. In their discussion, they say: No kidney cancer cases were considered exposed when latency was taken into account.
Q. Okay.
And they also say: No meaningful increases in the risk of kidney cancer were detected without latency.
Is that right?
MS. SULPIZIO: Object to the form.

A. Yes, that's what they say in their "Discussion."

Q. Okay.

And what is your understanding of taking latency into account for a health study?

A. I mean, it's taking into account the fact that when someone is exposed to a carcinogenic substance, they don't necessarily develop the cancer right away, you know, on exposure, but -- but the latency is how long it could potentially take for the -- you know, for the cancer to develop.

Q. Okay.

So is considering latency one of the ways a study might account for confounding bias or random error?

MS. SULPIZIO: Object to the form.

A. Say it one more time.

Q. Sure.

A. Sorry.

Q. Is considering latency one of

the ways a study might account for the possibility of a confounding variable or bias?

A. It is one way that you could.

Q. Do you have any reason to disagree with the statement we just read that, in Aschengrau, that no kidney cancer cases were considered exposed when latency was taken into account?

A. Say -- say that again. Do I have any reason to?

Q. To disagree with that statement we just read that, in Aschengrau, no kidney cancer cases were considered exposed when latency was taken into account.

MS. SULPIZIO: Object to the form.

A. I don't have a reason to -- to -- to disagree with that statement.

MR. BU: All right.

Can we pull up tab 12, please?

And you can set Aschengrau aside.

(Weiss Exhibit 9, Moore study 2010, was marked for identification, as of this date.)

BY MR. BU:

Q. I'm handing you what's been marked Exhibit 9.

Have you seen this document before?

A. I -- I'll be honest, I -- I don't remember. I may have. I reviewed so many articles, in particular in the beginning, that I'm sure I've -- I'm sure I've seen this, but it's just not jumping out at me right now, to be honest with you.

Q. Okay.

This is an article by Moore and other authors published in 2010 --

A. Yeah.

Q. -- is that right?

A. Yeah. I have seen this, yeah. Yeah, I've -- I've reviewed this.

Q. Okay.

A. For sure.

Q. And is this the same article that's cited for levels 2 and 3 on page 8 of your Fancher report?

A. Yeah, I believe -- yes.

Q. Okay.

And for level number 2 you cite a exposure to a TCE concentration of greater than 76 ppb.

Is that right?

A. Correct.

Q. And a ppb is a part per billion?

A. Yes.

Q. A part per billion is describing a concentration; is that correct?

A. It is. I mean, part per billion, I guess, you know, it is a concentration.

You know, with all of these studies, it becomes a challenge because I think a lot of times people almost use part per billion and -- and other metrics almost interchangeably, but parts per billion is a -- is a -- is a concentration.

Q. Okay.
A. Correct.
Q. So this is measuring something a little bit different than Aschengrau which was a cumulative exposure?
A. I mean, this is getting back to the days of, you know, conversions and calculations and...
Yeah, I mean, they're -- they're just representing it as parts per billion as opposed to Aschengrau which represented it as -- as like a total mass of, like, 27 to 44 milligrams, total mass ingestion or exposure versus you know, a exposure to a -- to a cumulative concentration.
Q. Okay.
Would you agree that determining the duration of an exposure to a given concentration is relevant to whether that exposure can cause cancer?
MS. SULPIZIO: Object to the form.
A. I mean, I think it probably depends. Sort of like we talked about the

dose-response curve earlier, it may be that, you know, certain high doses at a short duration may have a similar effect to a lower dose over a long duration, and I think it's -- I -- I think they're -- I think they're probably interrelated.

I'm -- you know, truly this isn't -- this isn't -- this isn't necessarily, you know, measuring exposure and -- and milligrams and -- this isn't necessarily -- this is left to more the epidemiologists and the, you know, environmental health experts, and, you know, I have to read their -- their papers and their data and rely on their expertise in terms of how they calculate things and so forth. But, it's not something that I, for instance, would use in my clinical practice on a daily basis.

Q. Okay.

I guess based on your understanding, if the concentration stays the same though, the health risks are different between a one-year exposure and

a 20-year exposure and a 50-year exposure?
A. I mean, I would think so.
Q. And that's because of the dose-response relationship?
MS. SULPIZIO: Object to the form.
A. I mean, it just -- I would believe that if you had the same level of ex -- I mean the same amount of exposure for a longer period of time that it probably -- you know, a longer exposure to the same carcinogen you would think would have a higher impact.
Q. Do you know what the median duration of exposure was for the cases in Moore 2010?
A. I don't recall.
Q. Okay.
Can you turn to the table -- to Table 1 on page 6,531. I don't know why they're numbered that way, but they are.
A. They're numbered that way 'cause cancer research literally publishes all their papers in order for the year. So

this came out in August, there was that many papers before this.

If you looked in my desk in the other room you'd see a similar journal that's about, you know, eight inches thick.

Q. Okay.

A. That's a different journal.

Q. Do you see the footnotes to Table 1?

A. I do.

Q. All right. And the footnotes describe some different interquartile ranges and medians.

Do you see that?

A. I do.

Q. Okay.

What's an interquartile range?

A. It's basically like the -- like the -- you divide -- you divide things into quartiles. So if you're looking at the -- for instance, the interquartile among controls between the 25th and the 75th, you're then looking at the middle 50

percent. So you divide the entire group into -- into fours, and then you can look at any one of those quartiles.

Q. For the cases in the study, do they report the median exposure in the interquartile range for duration of exposure?

MS. SULPIZIO: Object to the form.

A. In -- in this paper that we're reviewing by Moore?

Q. Yeah.

A. They do. They -- they measure it in -- in hours.

Q. Okay.

Above that do they also measure exposure duration in years?

A. They do.

Q. Okay.

A. Sorry, yes.

Q. And for the cases, what was the median exposure in years?

A. The median was 19.5 years.

Q. All right.

And what was the interquartile range?

A. 5.8 years to 31 years.

Q. Okay.

The 76 parts per billion, does that refer to a concentration in air or in water, if you know?

A. I don't know, to be honest.

Q. Okay.

Do you know if parts per billion in air would be different than parts per billion in water --

MS. SULPIZIO: Object to the form.

Q. -- or in another median?

MS. SULPIZIO: Sorry.

Object to the form.

A. I mean, I -- I -- I think my general chemistry professor in college would be very disappointed in me, but I -- I -- I don't recall. I -- I don't -- I don't remember.

Q. Okay.

A. I would assume, I mean, water

and air have different, you know, have different densities. So -- so I have to presume -- and I'm -- and I'm -- and I'm admittedly kind of guessing here, but I have to believe it's different in air than in water just because of the density of the media.

Q. In your report for Fancher on page 8 you also cite Moore for a cumulative exposure of greater than 1,580 ppb years.

Do you see that? Line 3.

A. Okay. Yep.

Q. Okay.

And I'm -- when you say 1,580 ppb years, you're referring to exposures to TCE; is that right?

A. Yes.

Q. And a ppb year is describing a cumulative exposure; is that correct?

A. Yes.

Q. And similar to the concentration, you're not sure whether the ppb years is referring to cumulative

exposure to contaminated air or contaminated water; is that right?

MS. SULPIZIO: Object to the form.

A. I'm not sure.

MR. BU: Can we please pull tab 13?

(Weiss Exhibit 10, Andrew study 2022, was marked for identification, as of this date.)

BY MR. BU:

Q. I'm handing you what's been marked Exhibit 10.

A. Okay.

Q. Have you seen this document before?

A. I have. I've definitely seen it before, yes.

Q. What is this document?

A. "Kidney cancer risk associated with historic groundwater TCE contamination."

Q. Is this the article that you cite for the fourth level included on

page 8 of your Fancher report?

MS. SULPIZIO: Object to the form.

A. This paper is -- is cited as -- as point number 4, yes.

Q. Okay.

And the level you cite is sustained exposure to zero to 25 ppb of TCE; is that right?

A. Correct.

Q. And similar to the concentrations in Moore, our understanding of that risk would also depend on the duration of the exposure to these concentrations of TCE.

Is that fair to say?

MS. SULPIZIO: Object to the form.

A. I mean, I -- I would think so, but to be honest, this isn't my expertise in terms of dose-response of carcinogenic agents and the development of cancer. I'm just going to be honest.

You know, whether you evaluate

it as just total exposure or total exposure over time, honestly, I think I would have to rely on -- on people that evaluate, you know, environmental exposures and the development of cancer, you know, to -- to -- to found an opinion, which is what I've done.

Q. Do you know how sustained exposure is being defined or described in this article?

MS. SULPIZIO: Object to the form.

A. I would have to -- I would have to look through it again and -- and remind myself.

Q. Okay.

Can you turn to page 5, Table 2?

A. Okay.

Q. In this study, do the authors look at 5-, 10-, and 15-year epics for exposure?

A. Yes.

Q. And so would it be fair to say sustained exposure would be a 5-, 10-, or

15-year exposure?

A. I mean for -- for this study they divided it into 5-, 10-, and 15-year.

Q. Okay.

So for item 4 in your report where you say "sustained exposure to zero to 25 ppb of TCE," is sustained exposure referring to 5-, 10-, or 15-year exposures.

MS. SULPIZIO: Object to the form.

A. Yes.

Q. Okay.

And on Table 2 do you see the odds ratios reported on the right-hand side?

A. I'm sorry, in Table?

Q. In Table 2 that we were just looking at?

A. Okay. Yeah, and the odds ratios.

Q. Okay.

A. Yes. Yes, I do.

Q. And what's your understanding of

what an odds ratio's approximating?

A. An odds ratio is essentially, you know, determining, you know, whether there's a -- whether there's a correlation. So if you have an odds ratio of, let's say, 1.5 it's 1.5 more times likely -- you know, more likely to have happened.

Q. Okay.

And Table 2 also reported confidence intervals; is that right?

A. It does.

Q. Okay.

And a confidence interval is one form of testing for statistical significance. Is that fair to say?

A. Correct.

Q. All right.

For this article, the only statistically significant odds ratio is for a 15-year exposure; is that right?

MS. SULPIZIO: Object to the form.

A. The p-value for a 15-year median

was -- was -- wait, hold on a second.

Say that again. Ask me a question one more time?

Q. Sure.

The only odds ratio that is statistically significant is for a 15-year exposure; is that right?

A. I mean, I think all the odds ratio here are -- are -- are significant.

If you mean by statistically significant meaning falling with -- within less than a p-value of .05, then yes.

Q. Zero to 25 parts per billion also reflects a range of concentrations; is that right?

A. Yes.

Q. Would you agree that the risk of kidney cancer is affected by whether the concentrations is at the low end of that range versus the high end of that range, all else being equal?

MS. SULPIZIO: Object to the form.

A. I mean, probably. Although as

we talked about before, probably different individuals have different susceptibility to some of these carcinogenic, you know, agents. So what -- you know, some patients with a very, very low level of exposure may be more prone to develop a kidney cancer, whereas other patients may require a somewhat higher level.

But, I mean, intuitively, a lower -- a lower exposure should have a little bit lower risk and if you have a higher exposure, it should be a higher risk.

Q. And the range being reported here also includes zero parts per billion; is that right?

A. It does.

Q. All right.

Is your opinion that an exposure to zero parts per billion is associated with kidney cancer?

MS. SULPIZIO: Object to the form.

A. No.

Q. Okay.

Is your opinion that any exposure greater than zero parts per billion is capable of causing kidney cancer?

A. I believe it's capable.

MR. BU: Can we pull tab 14, please?

And you can set that one aside.

(Weiss Exhibit 11, Woburn Center Incidence and Environmental Hazards 1969-1978 January 23, 1981, was marked for identification, as of this date.)

BY MR. BU:

Q. I'm handing you what's been marked Exhibit 11.

A. Okay.

Q. Do you recognize this document?

A. I do.

Q. What is it?

A. "The Cancer Incidence and Environmental Hazards 1969-1978 in Woburn."

Q. Okay.

And you cite this article for items 5 and 6 on page 8 of your Fantry -- Fancher report; is that right?

MS. SULPIZIO: Object to the form.

A. Yes.

Q. And both 5 and 6 are also concentrations; is that right?

A. Correct.

Q. This study is looking at residential exposures in Woburn; is that right?

A. Yes.

Q. Okay.

Do you recall how long those residential exposures were for?

MS. SULPIZIO: Object to the form.

A. I think it was -- I mean, I think this -- well, it says the study was -- was a nine-year study, but I don't remember how long the in -- I mean, I don't -- I don't recall, to be honest with you, the individual exposure levels from

this paper were.

MR. BU: Okay. All right. You can set that one aside.

Can we pull this will be, I think, tab 8?

(Weiss Exhibit 12, Bove study 2014, was marked for identification, as of this date.)

THE WITNESS: Okay.

BY MR. BU:

Q. I've handed you what's been marked as Exhibit 12.

Do you recognize this document?

A. I do.

Q. And what is it?

A. This is one of the Bove studies from 2014 "Evaluation of mortality among Marines and Navy personnel exposed to contaminated drinking water at US Marine Corps Base Camp Lejeune: a retrospective cohort study."

Q. Okay.

And you cite this article, I guess, several times on page 8; is that

right?

A. I do.

Q. All right.

And this article also distinguishes different levels of exposure; is that right?

A. It does.

Q. Like low, medium, high exposures?

A. Low, medium, and high, correct.

Q. Okay.

MR. BU: I'm sorry, can we also pull 29?

MS. SPRAYREGEN: Do you want to mark this as part of the same exhibit?

MR. BU: No, we can give it a different exhibit number. That's fine.

(Weiss Exhibit 13, Additional File 2: Table 11: Categorical Cumulative Exposures and Underlying Cause of Death, was marked for identification, as of this date.)

BY MR. BU:

Q. All right. I'm handing you what's been marked Exhibit 13.

A. Okay.

Q. Do you recognize these tables?

A. I do. I -- I do recognize these. I'm not sure where from, to be honest.

Q. Okay.

A. It's not labeled, but I recognize it.

Q. Do you recall whether Bove's 2014 mortality study included supplemental tables?

A. Yeah. Yeah, it did.

Q. Okay.

And do you recall reviewing those supplemental tables?

A. I do.

Q. All right.

So, on your Fancher report, I guess the levels -- well, we can skip around.

So, level 9 is cumulative

exposure to 1 to 4600 micrograms per liter month of exposure to all compounds at Camp Lejeune. Is that right?

A. That's correct.

Q. Okay.

And so this would be exposure to TCE, PCE, benzene, and vinyl chloride; is that right?

A. That's correct.

Q. And then item 12 is, I guess, medium exposure in Bove, is that right, to total VOC?

A. I believe so. I'm just going to check the chart.

Q. Okay.

A. Yes.

Q. All right.

And then item 15 cumulative exposure to greater than 12,250 is the high exposure to TVOC in Bove 2014; is that right?

A. Correct.

Q. And the hazard ratios for TVOC exposures for each of these categories on

the first page of Table S1 Exhibit 13; is that right?

A. Correct.

Q. And do you see the line for kidney cancer?

A. I do.

Q. Okay.

None of these hazard ratios are statistically significant, are they?

MS. SULPIZIO: Object to the form.

A. I mean, they're -- they're increased hazard ratios. I think -- I think one of the difficult things with this paper is that it relates purely to mortality as opposed to the development of the cancer, which can sometimes be -- you know, make it difficult to interpret. But the hazard ratios are -- are well above one. It's just that when you have only 42 cases of kidney cancer in the entire group, it makes it -- just due to small numbers, it makes it hard to make it statistically significant. Just because

something's not statistically significant doesn't mean it's not clinically significant. So the hazard ratios are -- are clearly elevated, but they do cross one.

Q. Okay.

And because they cross one, would it be fair to say we cannot reliably rule out chance or random error in explaining the association?

MS. SULPIZIO: Object to the form.

A. I -- I -- I would say that because they cross one, it makes it -- it makes it a challenge to completely rule out chance.

Q. Okay.

Can you turn to page 7? And this is still Exhibit 13.

And on page 7 of the supplemental tables, the hazard ratios for cumulative exposure to TCE are reported; is that right?

A. That's correct.

Q. Okay.

And similarly, each of these hazard ratios is also statistically insignificant; is that right?

A. I mean, each of these hazard ratios is -- is significantly elevated for kidney cancer, but again, I think because of just the -- the small number of -- of patients total, even though this is a, you know, a large series of a lot, the incidence is low, so the N is only 42. It's not uncommon for a confidence interval to cross one, but still be clinically meaningful. I mean, the hazard ratio is, you know, one-and-a-half, 1.5. So maybe it's clinically significant, but it may statistically cross one.

Q. Okay.

And because it crosses one, it's statistically insignificant?

A. No, I dis --

MS. SULPIZIO: Object to the form.

A. I disagree. You know, just

because a -- just because it crosses one, it means it's -- it's usually a law of numbers. It may -- it still can -- you know, still insinuates that there's an increased risk here, but it's harder to completely rule out chance.

Q. Okay.

And these hazard ratios in the supplement are what you refer to for items 7, 10, and 13 in your Fancher report; is that right?

A. Correct.

Q. Okay.

For the supplemental tables, can you turn to page 9?

This table is describing the hazard ratios for cumulative exposure to PCE; is that right?

A. That's correct.

Q. And these are the hazard ratios that relate to items 8, 11, and 14 in your Fancher report; is that right?

A. That is correct.

MS. SULPIZIO: Object to the

form.

BY MR. BU:

Q. Okay.

Similarly, the hazard ratios for cumulative exposure to PCE and kidney cancer are also all statistically insignificant, right?

A. I mean, the hazard ratios are all elevated. So I wouldn't stay that -- I wouldn't say that they're statistically insignificant. The hazard ratios are elevated.

Q. Okay.

Would you agree that the hazard -- sorry. That the confidence intervals cross one which makes it harder to rule out chance?

MS. SULPIZIO: Object to the form.

A. I would agree with that. And again, it -- the confidence intervals are wide because the -- the N is so small. That's what you -- that's a -- it's a statistical, you know, difficulty, but --

but I agree with that statement.

Q. Okay.

When you say "the N is small," you mean the number of cases in the study?

A. The number of -- number one, the number of actual mortalities from kidney cancer which is what they were looking at.

Q. In your Fancher report for item 16 you cite 18 months of residence on base from 1975 to 1985.

Do you see that?

MS. SULPIZIO: Object to the form.

A. I do.

Q. Okay.

And this 18-month residence is also coming from Bove 2014?

A. Yes.

Q. Okay.

Do you recall if this level is based on the overall hazard ratio for kidney cancer in Bove 2014?

A. I'm sorry, which? The 18 months of residence?

Q. Yeah.
A. Repeat that question, 'cause I'm not sure I understood it. Sorry.
Q. Let me ask it this way.
Why do you associate 18 months of residence on base from 1975 to 1985 with an increased incidence of kidney cancer?
MS. SULPIZIO: Object to the form.
A. I'd have to look. To be honest with you, it's been a -- it's been a little while since I re-reviewed this, but I think -- I think that's -- I shouldn't say 'cause I'm guessing.
I think -- I think it's coming from the Bove report as somehow they zeroed in on that length of time. I don't know if that was the average length of time or -- I think -- I think it was the average length of time that a resident was felt to be on base at Camp Lejeune, if I recall correctly.
Q. Okay.

A. So, when they were looking -- when they were looking at exposures of everyone that was there, they had to come up with some measure of, like, well, how long on average was someone there, and I think that's -- I think that's what they zeroed in on was 18 months.

Q. Okay.

For Exhibit 12, can you turn to page 8, Table 5?

A. Okay.

Q. And do you see the findings related to kidney cancer?

A. I do.

Q. Okay.

So, Table 5 is describing hazard ratios for all levels of exposure. It's not divided into these low, medium, high exposure groups like the supplemental tables. Is that right?

A. Correct.

Q. Okay.

The confidence intervals for kidney cancer also cross one; is that

right?

A. Yes.

Q. And the p-value is .19?

A. Yes.

Q. Okay.

Because the confidence interval crosses one, this makes it harder to rule out random error.

Is that fair to say?

A. Yes.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Okay.

And because the p-value is .19, that also makes it harder to rule out random error.

Is that fair to say?

A. Yes.

MR. BU: Can we please pull tab 9?

(Weiss Exhibit 14, Bove study 2014, was marked for identification, as of this date.)

BY MR. BU:

Q. I'm handing you what's been marked as Exhibit 14.

A. Okay.

Q. Do you recognize this document?

A. I do.

Q. And what is it?

A. It's the Bove article again from 2014 that does a similar cohort analysis to the personnel that were on base, but this is for the civilian employees that were on base or exposed to contaminated drinking water at Camp Lejeune.

Q. And this is the article that you cite for item 17 in your Fancher report; is that right?

MS. SULPIZIO: Object to the form.

A. Correct.

Q. Okay.

Can you turn to page 8, Table 4 for me, please?

A. Okay.

Q. And this is reporting hazard

ratios for different diseases, including kidney cancer; is that right?

A. It does.

Q. Okay.

And the hazard ratio for kidney cancer also crosses one, correct?

A. So, the hazard ratio is 1.92, but the confidence interval crosses one.

Q. Yeah.

And the p-value is greater than .05; is that right?

A. It is.

Q. Okay.

And because the confidence interval crosses one, this makes it harder to rule out random error. Is that fair to say?

A. That's fair to say.

Q. And because the p-value is .28, this makes it harder to rule out random error. Is that fair to say?

A. That's fair to say.

Q. Would you agree that because of the width of the confidence intervals,

these effect estimates have limited statistical precision?

MS. SULPIZIO: Object to the form.

A. I mean, I wouldn't necessarily agree with that. I think you can still, you know, get some information.

I think the problem is statistically when you have a small number of -- of N, your confidence intervals are frequently quite wide. And so you get some indication as to whether there is, you know, an increased risk based upon the hazard ratio, but, you know, you're -- you're somewhat limited just based purely on by numbers on whether you can get that -- that -- that confidence interval above one. You know, it's -- it just makes it a challenge, you know, from a statistical standpoint.

Q. Okay.

Would you agree that generally if the effect size is large, you don't need -- you can find statistical

significance with a smaller N?

A. If it's a very, very high impact, you know, yes.

Q. Okay.

And conversely, if the effect size is small, you'll likely need a larger N to find statistically significant results?

A. Correct.

Like for instance, in my practice, I frequently run prospective randomized trials, and when you're setting up a prospective randomized trial and you're doing your power calculation, to show it -- you know, to show -- you take into account a couple of things. You have to take into account what you think the impact's going to be, what's going to be the delta or the change, and then you take into account how many patients you're going to need to enroll in order to statistically prove that change. And it can be -- it could be a real challenge. Obviously if you have -- if you're trying

to -- if you have, let's say, a wound infection rate of 30 percent, it's going to be a lot easier to show a 15 percent reduction in a smaller number of patients than if you only have a wound infection rate of one percent. You're then only -- even if you only want to show a 50 percent reduction, you're going to have to enroll thousands upon thousands of patients. So the higher the impact, the lower the number of patients you need -- you usually need to use to -- to -- to identify it statistically.

It doesn't mean that you don't still have an impact. Even if that wound infection rate was only one percent, your intervention still may reduce it. It's just harder to show statistically that it reduced it.

MR. BU: Can we pull up tab 19, please?

(Weiss Exhibit 15, ATSDR study April, 2018, was marked for identification, as of this date.)

BY MR. BU:

Q. I've handed you what's been marked Exhibit 15.

Do you recognize this document?

A. I do.

Q. And what is it?

A. It's the report from the ATSDR "Morbidity study of former Marines, employees, and dependents potentially exposed to contaminated drinking water at US Marine Corps Base Camp Lejeune."

Q. Okay.

And this is the article you cite for items 18, 19, 20, and 21 in your Fancher report; is that right?

A. Correct.

Q. Okay.

And items 18 and 20 relate to TCE exposures?

A. Correct.

Q. And 19 and 20 relate to PCE exposures?

A. Correct.

Q. Okay.

Can you turn to page 76, Table 7?

A. Okay.

Q. And Table 7 describes odds ratios for cumulative exposure to TCE; is that right?

A. Yes.

Q. Okay.

And Table 7 is divided into low, medium, and high exposure categories; is that right?

A. It is.

Q. Okay.

And items 18 and 20 in your Fancher report refer to the medium and high exposure categories, is -- for kidney cancer; is that right?

A. I'm sorry, say that again.

Q. Sure.

A. Sorry.

Q. Item 18 in your Fancher report you cite a cumulative exposure to 110 to 11,030 ppb months of TCE?

MS. SULPIZIO: Object to the

form.

BY MR. BU:

Q. Is that right?

A. Yes.

Q. And this would be the medium exposure category in ATSDR 2018; is that right?

A. It would.

Q. Okay.

And item 20 this in your Fancher report is a cumulative exposure greater than 11,030 ppb months of TCE; is that right?

A. Yes.

Q. And this is the high exposure category on the Table 7; is that right?

A. Yes.

Q. And the confidence interval for kidney cancer for medium exposures to TCE also crosses one; is that right?

A. It does.

Q. And the high exposure for TCE in kidney cancer also crosses one?

A. It does.

Q. Okay.
All right. Can you turn to the next page of ATSDR 2018?
I'm sorry, two more pages to Table 8.
A. Is -- I'm sorry, on page 79?
Q. Page 78.
A. Page 78, okay.
Q. All right.
And this table describes odds ratios for cumulative exposure to PCE; is that right?
A. Yes.
Q. Okay.
And do you see kidney cancer in Table 8?
A. I do.
Q. Okay.
And similar to before, item 19 in your report exposure to 36 to 711 ppb months of PCE corresponds to the medium exposure in Table 8; is that right?
A. Yes.
Q. And item 21 cumulative exposure

to greater than 711 ppb months of PCE corresponds to the high exposure group; is that right?

A. Correct.

Q. Okay.

And the confidence interval for the medium exposures to PCE and kidney cancer crosses one; is that right?

A. Yes, the 95 percent confidence interval crosses one.

Q. And for high exposures, the confidence interval is greater than one, or the low end of the confidence interval is greater than one; is that right?

A. That is correct.

MR. BU: Can we pull tab 10, please?

(Weiss Exhibit 16, Bove study October 2024, was marked for identification, as of this date.)

BY MR. BU:

Q. I have handed you what's been marked Exhibit 16.

Do you recognize this document?

A. I do. I do.
Q. Okay.
A. Yes.
Q. And what is it?
A. It's another paper by Bove that came out in October 2024 "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."
Q. This is not a mortality study; is that right?
A. Correct. This is a cancer incidence study.
Q. Can you explain what the difference is between a mortality study and a cancer incidence study?
A. Sure. So, I mean, a mortality study is the, you know, the patients -- or, the -- the patients are -- you know, die of their disease. An incidence is -- is just that they developed the disease.
So it becomes very important in -- in cancers that have a, you know,

have a large number of patients that survive their cancer. Frequently we look at incidences as opposed to survival. So they're just -- they're just, you know, different measurements. One is you die of the disease and the other is you developed the disease. You may be cured of the disease. You may not be cured of the disease. But if -- incidence is the disease developed.

Q. Okay.

For kidney cancer, do you consider one type of study to have more utility than the other?

MS. SULPIZIO: Object to the form.

A. I mean, I don't think so. I think it depends on what you're looking at. No, I don't -- I don't think one is -- I think they're just -- they're different metrics.

Q. Okay.

Can you turn to page 7, Table 3 for me, please?

A. I'm sorry, Table?
Q. Page 7, Table 3.
A. Okay. Got it.
Q. It says "Comparison of cancer outcomes at Camp Lejeune versus Camp Pendleton among with the Marines/Navy subgroup" at the top.
A. Yes.
Q. And Table 3 reports some hazard ratios for kidney cancer; is that right?
A. Yes. I'm just trying to find it here.
Yes, there it is.
Q. Okay.
And then it also reports hazard ratios for different subtypes of kidney cancer?
A. Yes.
Q. The confidence intervals for all of these hazard ratios also crosses one; is that right?
A. Yes.
Q. Okay.
And the hazard ratios for clear

cell only are less than one; is that right?

A. Yes.

Q. And this would mean for this study there were fewer incidences of clear cell following exposure to water at Camp Lejeune than were expected; is that right?

MS. SULPIZIO: Object to the form.

A. Just repeat that, I'm sorry.

Q. Sure. Let me ask it this way.

How would you interpret a hazard ratio of less than one?

A. It can be very difficult to interpret. There may be no difference. You know, I find it hard to believe that it's protective, but it's basically that -- there's not statistical significance. You can't really say it helped or it didn't help. There's no data, in my mind.

Q. Okay.

Would it be fair to say if the hazard ratio is less than one there's no

increased incidence of that type of disease?

A. I -- it would be fair to say that there's not been identified a statistically significant increased incidence.

Q. Okay.

And can you turn to page 9, Table 4 for me, please?

A. Okay.

Q. And Table 4 is reporting hazard ratios for civilian workers at Camp Lejeune; is that right?

A. Yes.

Q. Okay.

And this is comparing civilian workers at Camp Lejeune to civilian workers at Camp Pendleton; is that right?

A. That's correct.

Q. Okay.

And I should have asked before, Table 3 is also making comparison between Marines at Camp Lejeune to Marines at Camp Pendleton?

A. That's correct.
Q. Okay.
The confidence intervals for kidney cancer on the Table 4 are -- they also all cross one; is that right?
A. Correct.
Q. Okay.
And is Table 4 what you referred to for item 23 in your Fancher report, more than 21 quarters spent on base as a non-civilian worker?
MS. SULPIZIO: Object to the form?
A. Yes.
Q. Is Table 3 what you refer to for item 22, one to six quarters stationed on base as a service member from 1975 to 1985?
MS. SULPIZIO: Object to the form.
A. Yes.
Q. Okay.
For the levels described in items 1 through 23, were there any other

articles that you relied on?
MS. SULPIZIO: Object to the form.
A. I don't think so.
Q. Okay.
And in all the Bove and ATSDR studies, the only statistically significant association was the high exposure group for PCE in the 2018 morbidity study; is that right?
MS. SULPIZIO: Object to the form.
A. I mean, they -- they all had increased hazard ratios, but -- but the confidence intervals, 95 percent confidence intervals crossed one.
Q. Okay.
So there was only one association that we looked at where the confidence interval did not cross one.
Is that fair to say?
MS. SULPIZIO: Object to the form.
A. There was one where we can say

with 95 percent confidence. The remainder we -- you know, had increased hazard ratios.

Q. Would you agree that if you measured 20 associations, you can expect to find one statistically significant association at a 95 percent confidence level even if there's no true association?

MS. SULPIZIO: Object to the form.

A. I mean, it's possible.

Q. Okay.

But that's what we would expect, right?

MS. SULPIZIO: Object to the form.

A. Repeat the question for me. Maybe I misunderstood the question.

Q. Sure.

A. Sorry.

Q. If we are conducting significance testing at a 95 percent level and we measure 20 associations that have no true association, we can expect one of

them to be statistically significant just by pure chance, right? One out of 20?

MS. SULPIZIO: Object to the form.

A. Yes.

Q. Okay.

A. One out of five by chance, it's the 5 percent.

Q. One out of 20.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. 5 percent.

MS. SULPIZIO: Object to the form.

A. I don't think I understand.

Q. Okay.

A. So let's back up.

Q. Yeah, sorry, I thought you said one out of five, but I think it's one out of 20, or 5 percent.

MS. SULPIZIO: Object to --

Q. Not one out of five 20 percent. Sorry.

MS. SULPIZIO: Sorry.

Object to the form.

A. Can you repeat the question and start fresh --

Q. Sure.

A. -- 'cause now I'm definitely getting confused.

Q. No, it's okay.

So, the convention is to conduct statistical significance at a 95 percent level or P of .05; is that right?

A. Correct.

Q. And if you set your confidence level at 95 percent, you should expect a statistically significant result 5 percent of the time even if there is no true association.

Is that fair to say?

MS. SULPIZIO: Object to the form.

A. That's correct.

Q. Okay.

So if you tested 20 associations with no true association, you can expect

that one of them will come back as statistically significant just by chance?

MS. SULPIZIO: Object to the form.

A. That's correct.

Q. Okay.

Other than the studies that we've discussed this morning and the studies listed in 1 through 23 of your Fancher report, are you aware of any statistically significant associations found between exposure to Camp Lejeune water and kidney cancer?

MS. SULPIZIO: Object to the form.

A. Specifically Camp Lejeune water and kidney cancer?

Not -- no.

Q. Okay.

Are you aware of any other levels of exposure that are associated with risk of kidney cancer?

MS. SULPIZIO: Object to the form.

A. Say that one more time. Sorry.
Q. Sure.
So, 1 through 23 you list several levels of exposure that have been found to be correlated with kidney cancer; is that right?
MS. SULPIZIO: Object to the form.
A. Correct.
Q. Okay.
Are you aware of any other levels that have been found to be correlated with kidney cancer other than the ones listed in 1 through 23 of your report?
A. I'm not aware of any, or I'm certainly not thinking of any right now off the top of my head.
MR. BU: Okay.
All right. I think this is a good place to stop.
THE VIDEOGRAPHER: The time right now is 12:55 p.m., and we're off the record.

(Luncheon recess taken.)

- - -

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

- - -

THE VIDEOGRAPHER: The time right now is 1:32 p.m., and we're back on the record.

BY MR. BU:

Q. Dr. Weiss, during the break, did you discuss your deposition testimony with anyone?

A. I did not.

Q. Is there anything you've testified to today that you'd like to clarify or correct?

A. No.

Q. In your report, you describe Mr. Fancher's exposure as substantial.

Do you recall writing that?

A. I do.

Q. Okay.

You don't quantify what a substantial exposure is, do you?

A. I don't quantify it. I said

substantial because based upon Dr. Reynolds' charts and graphs and calculations as to what his exposure level was, the amount that he was exposed in comparison to what the medium, low, and high risk exposure was considered in the Bove papers, I felt that it was a substantial amount that he was exposed to, but I don't quantify that.

Q. Okay.

Similarly, you don't identify a threshold amount of exposure to VOCs whereby an individual is as likely as not to develop a kidney cancer, do you?

A. I do not.

Q. For Mr. Fancher's exposures, did you independently calculate the amount of his exposures?

A. I did not.

Q. Okay.

A. I reviewed Dr. Reynolds' calculations and what inputs she used into developing those charts, but I used her charts.

Q. Okay.

Did you use anything other than Dr. Reynolds's charts to calculate Mr. Fancher's exposures?

MS. SULPIZIO: Object to the form.

A. I did not.

Q. Can you turn to page 10 of your report for me, please?

A. Okay.

Q. Do you see that paragraph starting "In sum, Mr. Fancher's exposure"?

A. I do.

Q. And there are four items listed?

A. I do.

Q. The third item it reads: The intensity of the exposure as shown by the ATSDR water modeling data and other data.

Do you see that?

A. I do.

Q. Okay.

What other data did you consider to determine the intensity of the exposure?

A. Well, this -- this was based upon -- this was what Dr. Reynolds used to calculate this chart. It wasn't me. And she utilized what the ATSDR had -- had estimated a marine on base would have been exposed to, or actually just ingested, 'cause it's actually an underestimate, just what the ATSDR data would have suggested that a marine on base for that amount of time would have been -- would have ingested.

And other data -- I don't know, to be honest with you. I -- I think it's -- you know, the ATSDR data and -- and I guess the other data would be the known calculations like from column 1 on what his microgram per liter months, you know, was.

Q. Okay.

Do you see the next full paragraph on page 10?

A. Yes.

Q. All right.

And it reads: I formed my

opinion that Mr. Fancher had substantial exposure solely based upon Mr. Fancher's deposition, the elevated levels of concentrations in the water, and additional documents from Mr. Fancher's file.

Do you see that?

A. I do.

Q. What additional documents did you consider in determining whether Mr. Fancher had substantial exposure?

A. Some of the -- the depositions by Mr. Fancher in terms of, you know, how often he was living on base, when he was living off base, how often was he showering on base, how many meals was he eating on base. You know, he goes into pretty good detail as to, you know, how much exposure he would have had while he was working there or while he was, you know, living across the street and still working at the base. So that would be the other information from his deposition.

Q. Okay.

Other than the deposition transcript, were there other documents you considered in determining whether Mr. Fancher had substantial exposure?

A. I don't -- I don't think so.

Q. Okay.

When you -- sorry, to go back to item 3, the intensity of the exposure.

Are you referring to the concentration or something else?

A. The intensity of the exposure would be the -- the concentration of the exposure in combination with the length of time. I wouldn't say exposure really. It's -- it's ingested based upon this chart. So, you know, how often he ate on base, how often he showered, how often he, you know, drank water while he was training, how many days a week he was training. All of that, kind of, factored into it.

Q. Okay.

What concentrations would you consider intense?

MS. SULPIZIO: Object to the form.

A. I mean, I think anything that's, you know, in the moderate to, you know, high exposure level, based upon Bove, I would say is intense.

Q. Do you know how those exposure classifications were determined for Bove?

A. I don't recall.

Q. So for example, like item 7, the low exposure is cumulative exposure of one to 3,100 micrograms per liter month of TCE, right?

A. Correct.

Q. And you're not aware of why the cutoff is at 3,100 micrograms per liter rather than 4,100 micrograms per liter month or 2,100 microgram per liter month.

Is that fair to say?

A. I don't remember why they made those -- those cutoffs.

Q. Okay.

And Dr. Reynolds provides a total mass of ingested chemicals; is that

right?

A. She does.

Q. Okay.

Do you know whether total mass of ingested chemicals is a standard exposure assessment metric in risk assessment?

MS. SULPIZIO: Object to the form.

A. I mean, I believe that total mass ingested is -- is used, but I don't know how you would define, you know, standard, I guess.

Q. Okay.

Does it matter over what period of time that total mass is ingested?

A. It may.

Q. Are you aware of other epidemiological studies that measure exposure based on total mass of ingested chemicals?

A. I'm not sure I understand the question, to be honest with you, because I -- I mean, I'm aware of plenty of

studies that talk about total exposure level as a -- I mean, I think they're a total mass, but I'll be honest, I'm not sure. I'm not sure your line of questioning.

Q. Okay.

Is exposure more commonly measured in concentration multiplied by a duration of exposure, like a ppm year or a ppb month?

A. Yes.

Q. I guess like a smoking pack year is kind of measuring the same thing, right?

A. Yes, it's very similar to, you know, pack year history.

Q. Would you agree that the same exposure may affect people differently based on body weight?

A. I mean, I think it -- it -- it may, but that's probably more pertinent when there's extremes in -- in body weight.

Like for instance, with Mr.

Fancher, he -- his BMI was, like, pretty normal when he was -- you know, when he was exposed to these chemicals. You know, clearly these chemicals are metabolizing and distributed throughout the body. So if someone is in, like, an extreme of weight, it may have an effect on -- on the overall cancerogenic potential. But -- but to be honest with you, I don't know if we know that either way.

Q. Okay.

In your clinical practice, are there medications that you need to dose based on body weight?

A. Yes.

Q. What are some examples?

A. Like blood thinners like Lovenox, it's dependent on the fat distribution.

Q. And why do these -- why do blood thinners need to be dosed based on body weight?

A. Because the way that -- the way the drugs are metabolized, you need a

larger dose for a larger patient.

Q. You need a larger dose for a larger patient to see the same effect. Is that fair to say?

A. That's correct.

Q. Okay.

A. And it's -- it's also very common in children because children are rapidly growing, that you -- that you weight-base their medications. But it's much less common in adults. You know, there's -- there's far fewer medications that are dosed based on weight because there's not as much variation in -- in -- in the weights.

Q. Dr. Reynolds calculates exposures for TCE, PCE, vinyl chloride, and benzene; is that right?

A. That's correct.

Q. Did Dr. Reynolds calculate a total VOC exposure?

A. Yes, I believe she did.

Q. Did you calculate that total VOC exposure?

A. I didn't calculate it, but I --
Q. Okay.
A. I relied on Dr. Reynolds' calculations.
Q. All right.
And -- okay. I think you mentioned that you think that Dr. Reynolds's calculations underestimate the true exposure for Mr. Fancher; is that right?
A. I do.
Q. Okay.
And that's because it -- Dr. Reynolds's calculations account only for ingestion and not for inhalation and dermal exposure; is that right?
A. That's correct.
Q. Do you know whether the risks for these chemicals is different between ingestion, inhalation, and dermal exposure?
A. No, I don't.
Q. If Dr. Reynolds's ingestion overestimated Mr. Fancher's actual

ingestion, is it possible that her charts would not underestimate his total exposure?

MS. SULPIZIO: Object to the form.

A. I don't understand the question. Say it -- say it again, please.

Q. Sure.

So, Dr. Reynolds calculates based on Mr. Fancher's deposition testimony and some field manuals that he ingested about 577,000 micrograms of TCE; is that right?

MS. SULPIZIO: Object to the form.

A. That's correct.

Q. Okay.

And that calculation is based, in part, on the ATSDR's water model; is that correct?

A. It's based in part on the ATSDR modeling, but it's also taking into account -- for the 577,000. It's also taking into account Mr. Fancher's

deposition and testimony about how -- how often he was drinking, how often he was showering, et cetera.

Q. Okay.

Part of that calculation is determining the concentrations of TCE in Camp Lejeune water at the time Mr. Fancher was at Camp Lejeune; is that right?

A. That's correct.

Q. Okay.

Do you know where those concentrations come from?

A. They come from the ATSDR.

Q. Okay.

So I guess what I'm really trying to ask is if those concentrations are greater than what they actually were at Camp Lejeune, is it possible that the 577,000 micrograms overestimates Mr. Fancher's total ingestion?

MS. SULPIZIO: Object to the form.

A. Yeah, I mean, it -- so what you're saying is if the ASDR -- ATSDR

reported concentrations in the water supply were wrong and actually the concentration was much lower, then would that mean this is an overestimation of what Mr. Fancher was exposed to, then yes.

Q. Okay.

A. But we're relying on the ATSDR for the -- you know, for the concentrations that were present, or at least that's what Dr. Reynolds relied on.

Q. And if the 577,000 microgram ingestion isn't overestimate, it might not then underestimate the total exposure if the, I guess, the error is greater than the inhalation and dermal exposures, if that makes sense.

MS. SULPIZIO: Object to the form.

A. I mean, we don't know what the inhalation expose -- I mean, we -- we -- the inhalation exposure and the dermal contact isn't factored into this equation. So we're getting into, kind of, hypotheticals that are really hard to come

up with, but it would depend on how -- how far they were off on the ATSDR estimation of the concentrations versus how much more inhalation and dermal contact, and, you know, you'd have to compare those two.

Q. Right.

Are you aware of any calculations for Mr. Fancher's inhalation or dermal exposures?

A. I'm not.

Q. All right.

So, if there is some underestimation of the concentration at Camp Lejeune, we wouldn't be able to determine whether Dr. Reynolds's calculations overestimate or underestimate Mr. Fancher's total exposure?

MS. SULPIZIO: Object to the form.

A. I mean, if the -- now you're -- if there's an underestimate of the concentration of chemicals by the ATSDR, then that would mean that all of these concentrations in Dr. Reynolds' actually

should be -- should be higher.

Q. Okay.

Would -- if ATSDR overestimated the concentration levels, we wouldn't be able to determine whether Dr. Reynolds's calculations overestimate or underestimate Mr. Fancher's total exposure because we don't know the delta for the inhalation or dermal exposures.

Is that fair to say?

MS. SULPIZIO: Object to the form.

A. It wouldn't be that we couldn't calculate it because we don't know the dermal and inhalation. It would be because if -- if their estimates were wrong, then we wouldn't be able to rely on their estimates.

Q. Okay.

Did Mr. Fancher have a cumulative exposure to 27 to 44 milligrams of PCE?

A. How much, I'm sorry? Say that again?

Q. Sure.
A. Did he have a cumulative exposure of?
Q. Of 27 to 44 milligrams of PCE.
MS. SULPIZIO: Object to the form.
A. 27 -- I'm not sure I'm reading you correctly. I'm not -- I don't think I'm understanding you correctly.
You're asking what was Mr. Fancher's cumulative exposure to PCE?
Q. Yes.
A. I mean, his cumulative exposure's, based upon Dr. Reynolds, was, you know, 247.
Q. 247 what?
A. Micrograms per liter month.
Q. Okay.
Does Dr. Reynolds use that to calculate possible consumption in milligrams or micrograms?
MS. SULPIZIO: Object to the form.
A. No.

Q. Okay.

Did Mr. Fancher have an exposure to 1,580 ppb years of TCE?

MS. SULPIZIO: Object to the form.

A. I'm not sure -- I'm not sure I understand where that -- where you're getting that number from.

Q. Sure.

So, go back to page 8 of your report.

A. Yeah.

Q. And these are the levels you've listed that are found to be correlated with kidney cancer.

A. Okay.

Q. Right.

And the first level is cumulative exposure to 27 to 44 milligrams of PCE.

A. Okay.

Q. We can skip the second one.

The third one is cumulative exposure to greater than 1,580 ppb years

of TCE, right?

A. Okay.

Q. So based on your understanding of Dr. Reynolds's report, did Mr. Fancher have an exposure to 1,580 ppb years of TCE?

MS. SULPIZIO: Object to the form.

A. Again, you're -- the problem with a lot of these studies is they -- people interchangeably use "parts per billion" as a concentration as opposed to the "micrograms per liter months."

So, it -- it's very difficult for me to compare the two directly.

Q. Okay.

Did Mr. Fancher have a sustained exposure to zero to 25 parts per billion TCE?

A. I mean, I think his exposure --

MS. SULPIZIO: Object to the form.

A. I think his exposure was probably higher than that.

Q. Higher in terms of the concentration of TCE?

A. It's probably higher in terms of total amount mass ingested.

Q. What do you think -- do you have an opinion about the total mass ingested in the Andrew 2022 study?

A. No.

Q. Okay.

Do you recall if they described total mass ingested in that study?

A. I don't recall.

Q. All right.

And earlier we discussed that the study was looking at 5-, 10-, and 15-year residential exposures, right?

A. Correct.

Q. Okay.

Did Mr. Fancher have a five-year residential exposure?

A. No.

Q. Okay.

His exposure was less than one year, right?

MS. SULPIZIO: Object to the form.

A. His exposure was from, you know, October of -- you know, October of '79 to June of '81. So it was, like, 337 days.

Q. So less than one year?

MS. SULPIZIO: Object to the form.

A. Less than one year.

Q. Okay.

And for the Parker study, items 5 and 6 on your report, you don't know how long the duration of those exposures in the -- the Parker study were, right?

A. That's correct.

Q. Okay.

A. 'Cause it's just reported as a total.

Q. Okay.

So we can -- we can't determine whether Mr. Fancher's exposure was similar in duration to those in the Parker study.

Is that fair to say?

MS. SULPIZIO: Object to the

form.

A. I think that's fair to say.

Q. Okay.

And because Parker was looking at residential exposures in Woburn, is it fair to say that Mr. Fancher's one-year exposure at Camp Lejeune was shorter than those in the Parker study?

MS. SULPIZIO: Object to the form.

A. I think it's hard -- I think it's really it's hard to know because these were -- this is like a community retrospective study and you don't -- you don't have nearly as much information about those people and where they were living and for how long and what they were exposed to as you did -- as you do in Mr. Fancher's case where because he was in the military, you know what days he was living there and what days he was living off campus.

So I think you -- you have a lot more information about Mr. Fancher than

you could possibly have in a retrospective, kind of, community observational study.

Q. Okay.

So do you think the durations of exposure in Parker are comparable to Mr. Fancher's one-year exposure?

MS. SULPIZIO: Object to the form.

A. I don't think I can say.

Q. Was Mr. Fancher exposed to PCE at 21 parts per billion?

MS. SULPIZIO: Object to the form.

A. I mean, again, the -- the calculations by Dr. Reynolds were not in parts per billion. So, you know, I think you would -- I think you would have to literally, you know, do a calculation to -- to convert, but I think it would be higher. I think it would be higher, to be honest with you.

Q. Okay.

And why do you think it would be

higher than 21 parts per billion?

A. Because of the -- because of the level that was, you know, in the water, the concentration, and the period of time he was there. I think it would have -- it was a very high -- it was a high concentration of TCE, PCE, all of these, you know, compounds.

Q. Okay.

MR. BU: Sharon, can we pull tab 25, please?

(Weiss Exhibit 17, Appendix 7 David William Fancher (Kidney Cancer), was marked for identification, as of this date.)

BY MR. BU:

Q. I've handed you what's marked as Exhibit 17.

Have you seen this document before?

A. Boy, that is really small print. Yes.

Q. And what is it?

A. It's a -- it's a table that was

calculated by Dr. Reynolds looking at specifically for Mr. Fancher and potential exposure to each of the organic compounds, and it's an estimate first based upon if you just assumed he had a one liter per day ingestion and then by utilizing what the ATSDR had said that a, you know, a marine staying on base would have been exposed to, and then lastly, it's a -- it's a combination of the ATSDR -- ATSDR used for the concentration of exposure combined with Mr. Fancher's testimony as to how often he was, you know, felt to be ingesting --

Q. Okay.

A. -- the water.

Q. Can you turn I guess it's to the third page of this exhibit?

A. Okay.

Q. And there's a table with blue headers on the right-hand side for TCE, PCE, VC, and BZ.

Do you see that?

A. Is it Chart 1 "Days on Base and

Cumulative"?

Q. Yes.

A. Okay. I see blue headers.

Q. All right.

And are these blue headers describing the concentration of contamination for the different VOCs at these different points in time?

A. Yes.

Q. Okay.

And for PCE, most of these concentration levels are less than 21 micrograms per liter month; is that right?

MS. SULPIZIO: Object to the form.

A. Yeah, it looks like all of them but two.

Q. Okay.

A. No, three.

Q. And you said there's some uncertainty about translating micrograms per liter month to ppbs; is that right?

MS. SULPIZIO: Object to the form.

A. I think that, unfortunately, at least based upon my review of these literature, these -- these concentrations are almost -- almost seemed seem to be used interchangeably. And so it's hard for me -- it's hard to compare.

Q. Okay.

Looking at item 13 of your Fancher report, would you agree that Mr. Fancher's cumulative exposure was not greater than 7,700 micrograms per liter month of TCE?

MS. SULPIZIO: Object to the form.

A. Mr. Fancher's exposure was believed to be a little over, you know, 5,340 to TCE.

So is the question is that less than 7,000? Yes.

Q. Okay.

And he was not exposed to greater than 380 micrograms per liter month of PCE, right?

A. That's correct. He was exposed

to 200 -- estimated to be 247.

Q. Okay.

And he was not exposed to more than 12,250 micrograms per liter month of all compounds at Camp Lejeune?

MS. SULPIZIO: Object to the form.

A. I mean, I think, again, these are all estimates. So I can't say that he was not exposed to that much.

The -- the estimates of total organic compounds exposed to, what -- what number did you -- did you say again?

Q. Sure.

This is item 15 in your report. Mr. Fancher was not exposed to greater than 12,250 micrograms per liter month to all compounds at Camp Lejeune?

MS. SULPIZIO: Object to the form.

A. I think based upon these un -- and, again, what I thought were underestimates by Dr. Reynolds, they would be less than -- his exposure would be less

than 12,250, but, again, based upon what I think are underestimates by Dr. Reynolds.

Q. Okay.

Are you able to determine the magnitude of that underestimate?

A. I'm not.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Are you able to determine, based on that underestimate, how likely it would be for Mr. Fancher to have been exposed to more than 12,250 micrograms per liter month of all compounds?

MS. SULPIZIO: Object to the form.

A. I can't.

Q. Okay.

Looking at item 20, similarly, you would agree that Mr. Fancher was not exposed to greater than 11,030 ppb months of PCE?

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Right?

A. I -- I can't say.

Q. And why can't you say?

A. Well, again I think, you know, we're -- ask the question again? I'm sorry, I'm getting confused by your question.

Q. That's fine.

Would you agree that Mr. Fancher's cumulative exposure to TCE was not greater than 11,030 ppb months?

A. I don't think I can -- I don't think I can -- I don't think I can comment on that or calculate that.

Q. And why don't you think you can comment on that?

A. Because again we're looking at, you know, parts per billion months, and much of the analyses that I've used to look at Mr. Fancher's exposure have been based upon Dr. Reynolds, who's utilizing a, you know, micrograms per liter months, and -- and unfortunately they -- they seem

to be using them interchangeably, but it's difficult to compare the two.

Q. Okay.

So you can't compare Mr. Fancher's exposures to item 20 either way?

MS. SULPIZIO: Object to the form.

A. I think -- I mean, I think you can. I think, you know, some of these studies, like the ATSDR, you know, that -- that's -- you know, just because they use that metric don't mean they're -- they can't be compared.

You know, I'm relying on Dr. Reynolds who's an expert on -- in putting these calculations together and then -- and then using those calculations that she's made to look at the existing literature, a/k/a the Bove paper, to say what's your -- you know, what's your -- a reliable, you know, exposure measures.

Q. Okay. Well, I think let's stay focused on this.

Item 20 comes from ATSDR's 2018

morbidity study, right?

A. Yes.

Q. Okay.

Are you able to compare Mr. Fancher's exposures described in Dr. Reynolds's report to the ppb month exposures described in the morbidity study?

MS. SULPIZIO: Object to the form.

A. I mean, I think you can compare all of these things. They're just -- they're just not an exact, you know, conversion, but you can compare. I believe that Mr. Fancher's exposures are in line with these exposures, which is what I delineated in this.

But I just -- I can't -- I can't give an exact number. I can't give an exact number. I can't calculate it.

Q. Okay.

So, is your opinion that Mr. Fancher's TCE exposures as described by Dr. Reynolds are consistent with item 20

cumulative exposure greater than 11,030 ppb months of TCE?

MS. SULPIZIO: Object to the form.

A. I think they can -- I think they're -- I think they can be compared. I think that these levels are, you know, based upon her calculations, are in the realm of -- of being similar values.

Q. And how do you determine that they're similar values?

A. Based upon -- you know, based upon review of the literature. Based upon reviewing the ATSDR. Based upon, you know, her statement in -- in formulating these. Putting all them together.

Q. Okay.

So, Dr. Reynolds describes Mr. Fancher's TCE exposure as 5,340 micrograms per liter month, right?

A. For TCE?

Q. For TCE, yes.

A. Yes.

Q. Can you walk me through how you

compare that to the cumulative exposure greater than 11,030 ppb months of TCE in item 20 of your Fancher report?

MS. SULPIZIO: Object to the form.

A. I can't. I can't cal -- I mean, I can't do a calculation here. I don't -- I'm not sure there is a calculation that can be done to convert those.

What -- what I'm -- you know, what I'm utilizing is her calculation, which is believed to be a, you know, an underrepresentation of what the exposure level was and then trying to compare it to the existing literature on parts per billion.

Q. Okay.

But can you walk me through how you make that comparison to this 11,030 ppb month of TCE in item 20 of your Fancher report?

MS. SULPIZIO: Object to the form.

A. No, I can't.

Q. Can you explain how you would compare Mr. Fancher's PCE exposure as calculated by Dr. Reynolds to item 21 cumulative exposure greater than 711 ppb months of PCE?

MS. SULPIZIO: Object to the form.

A. Ask the question again?

Q. Sure.

Similar to TCE, Dr. Reynolds also calculates Mr. Fancher's PCE exposure, correct?

A. Correct.

Q. All right.

And she expresses that PCE exposure in micrograms per liter month, correct?

A. Correct.

Q. And she determines that Mr. Fancher's cumulative exposure to PCE was 247 micrograms per liter month, correct?

A. Correct.

Q. All right.

And 247 micrograms per liter

month is the exposure that you rely on for your report.

Is that also correct?

A. That's correct.

Q. Okay.

Can you walk me through how you would compare that 247 micrograms per liter month PCE exposure to item 21 in your Fancher report, cumulative exposure greater than 711 ppb months of PCE?

A. I mean, really I'm relying on --

MS. SULPIZIO: Object to the form.

A. Really I'm relying on the experts to develop these numbers, Dr. Reynolds, and then I'm comparing these numbers to the existing literature, but in particular for -- for TCE, PCE, VC, I'm really -- I'm really comparing it to the low, moderate, and high risk groups from the -- from the Bove paper because that allows me to compare apples to apples, because, you know.

Q. Okay.

So would it be fair to say you're not really comparing Dr. Reynolds's calculations to the ATSDR morbidity study?

MS. SULPIZIO: Object to the form.

A. I wouldn't say that's fair to say. I mean, I still -- I'm still able to compare it, or at least, you know, take it into -- into -- into thought, but -- but it's much more challenging to compare the two. I mean, you're not -- but I'm still using it to compare.

Q. Okay.

And, I'm sorry, I'm still a little unclear on this. How do you make that comparison between 247 micrograms per liter month to 711 ppb months?

A. I rely on the experts that calculate, you know, this. I -- I rely on the -- the -- the expert causation statements as to exposure, but most of my comparison for these values that were -- that were developed by Dr. Reynolds, I'm really utilizing to put into a low,

moderate, high risk category to compare to the Bove paper.

Q. Okay.

Looking at item 16, you would agree that Mr. Fancher did not reside on base more than 18 months, right?

MS. SULPIZIO: Object to the form.

A. Correct.

Q. All right.

And you would also agree that he was not employed on base for more than 2.5 years?

A. Correct.

Q. And looking at item 23 on the next page, Mr. Fancher did not spend more than 21 quarters on base as a civilian worker, right?

A. Correct.

Q. Can you turn to page 9 of your report?

A. Okay.

Q. Do you see the paragraph starting "According to ATSDR" at the

bottom of that page?

A. I do.

Q. Okay.

And do you see the third sentence after footnote 5?

A. Okay.

Q. And you wrote. According to ATSDR/Bove, it is likely that during training, the water supplied in the field came from the Hadnot Point Water System with both measured and estimated levels of TCE and PCE substantially higher than their MCLs.

Did I read that correctly?

A. Yes.

Q. Okay.

What is your understanding of what an MCL is?

A. It's been a while since I put this together, but -- I think -- I'll be honest, I don't recall. I think it's basically just in comparison to, you know, a water system that wasn't the Hadnot system, would be like an expected

exposure.

Q. Okay.

So your recollection is this is not comparing to the levels at which we would expect to see health effects.

Is that fair to say?

MS. SULPIZIO: Object to the form.

A. I -- I'll be honest with you, I don't -- I don't recall. I don't recall.

Q. Okay.

And how do you define whether estimated levels are substantially higher?

A. I mean, substantially higher would mean that, you know, it's having a -- a significant, you know, effect or it's -- it's a subjective term "substantially."

Q. Are there any articles or guidelines you consulted to determine whether a level of contamination is substantially higher?

A. No.

MS. SULPIZIO: Nathan, when

you're at a point, can we take maybe five minutes?

MR. BU: Sure. Give me just one more question.

MS. SULPIZIO: That's fine.

BY MR. BU:

Q. Are you able to express the probability of cancer risk for exposures at a given level?

MS. SULPIZIO: Object to the form.

A. No.

Q. And do you know what the MCLs for TCE and PCE are?

A. I mean, I'm sure I did, but I don't right now.

MR. BU: All right. We can stop there.

THE VIDEOGRAPHER: The time right now is 2:17 p.m., and we are off the record.

(Recess taken.)

THE VIDEOGRAPHER: The time right now is 2:33 p.m., and we're back

on the record.

BY MR. BU:

Q. Doctor, during the break, did you discuss your deposition testimony with anyone?

A. I have not.

Q. Is there anything that you've testified to today that you'd like to clarify or correct?

A. I don't think so.

Q. Okay.

Do you know whether people are exposed to background levels of TCE in everyday life?

MS. SULPIZIO: Object to the form.

A. I mean, I don't know that. I would have no way of knowing it.

Q. Okay.

Would you agree that people generally are exposed to background level of carcinogens?

A. Yes.

Q. And it's impossible to live a

life completely free from all cancerogenic exposures.

Is that fair to say?

A. Yes.

Q. Did you consider whether Mr. Fancher was exposed to background levels of TCE in your report?

A. I mean, I -- I think in the differential I -- I thought about lots of things for Mr. Fancher and what he may or may not have been exposed to.

The reality is is that there's no way for me to know what he was exposed to other than the fact that I know he was exposed to TCE, PCE, vinyl chloride, benzene. That's all I can know.

Q. So you don't make any comparison to what you know about Mr. Fancher's TCE exposures to what might be reasonable background levels of TCE, do you?

MS. SULPIZIO: Object to the form.

A. I do not.

Q. Okay.

Do you offer any opinions about why the exposure to TCE in Camp Lejeune water would be the cause of Mr. Fancher's cancer but not exposure to background levels of TCE?

MS. SULPIZIO: Object to the form.

A. I mean, I don't -- I would have no way of knowing what type of, you know, low level carcinogenic exposures anyone could have, let alone Mr. Fancher. All I have is known documented exposure to these volatile compounds. So that's all I could really pass judgment on or -- or measure.

You know, obviously in -- in developing a differential, I put forth all of the, you know, known causes of kidney cancers. For instance, one of those is exposure to heavy metals like cadmium, and we don't have any -- any data based upon his testimony or his deposition that he had had high exposure to -- to any of the -- you know, to that substance or any other carcinogen.

Q. Okay.

Did you consider how frequently TCE is detected in water supplies or in groundwater?

MS. SULPIZIO: Object to the form.

A. I didn't. I -- I -- I think I recall reading somewhere about a very, very low level being in, you know, in other water supplies, but I -- and again, I don't remember the numbers, but I remember thinking they were much, much, much smaller than what was seen in the Hadnot Water Plant.

Q. Okay.

Did you consider whether TCE occurs frequently at -- in ambient air?

A. I did not.

Q. Okay. All right.

MR. BU: Sharon, can we grab the next exhibit, the TCE tox profile?

(Weiss Exhibit 18, ATSDR Toxicological Profile For Trichloroethylene June 2019, was

marked for identification, as of this date.)

THE WITNESS: Okay.

BY MR. BU:

Q. Have you seen -- I've just handed you what's been marked Exhibit 18.

Have you seen this document before?

A. I -- I don't remember reading this document. It doesn't mean I haven't seen it before. Again, I reviewed a lot of documents, in particularly in the beginning when I was, you know, first started reviewing this case, but I -- I don't recall this one in particular. It doesn't mean I didn't review -- that I didn't read it.

Q. Okay.

A. To be honest.

Q. Okay.

Does it appear to be a toxicological profile on TCE by the ATSDR?

A. It does.

Q. Okay.

And you reviewed and relied upon other ATSDR publications for your report, right?

A. Yeah. I mean, I relied on multiple ATSDR, you know, documents.

Q. Can you turn to page 322 for me, please?

A. Okay.

Q. And do you see the section heading "6.4.1 Air"?

A. I do.

Q. All right.

The ATSDR reports that trichloroethylene is widely detected in ambient air, correct?

MS. SULPIZIO: Object to the form.

A. That's what it says here.

Q. All right.

Do you have any reason to disagree with that statement?

A. No.

Q. All right.

Do you know whether TCE is also

detected in food?

A. I do not know.

Q. Okay.

Would it surprise you to find that TCE is detected in low levels in different food products?

MS. SULPIZIO: Object to the form.

A. I mean, it wouldn't surprise me that TCE is found in ambient air or in food products, but the reality is is that everyone is inhaling ambient air, and everyone is being exposed to food products. So -- so you would think in these comparison groups and in cohort studies, et cetera, that everyone would have that same level of exposure, at least at baseline.

And then in this case, you're looking at, you know, additional documented increased levels of exposure. But it wouldn't surprise me if there's some level of TCE in the air or in food.

Q. Okay.

Can you turn to page 333, Table 6-8?

ATSDR reports various ppb TCE contamination in different food products, right?

A. Yes.

Q. Okay.

And for your report, you didn't -- did not consider whether Mr. Fancher may have been exposed to TCE through these different types of food products, did you?

MS. SULPIZIO: Object to the form.

A. I mean, I did not -- I did -- I did not take into account him having any type of, you know, different exposures outside of the camp that the general population wouldn't have. So I didn't take into -- you know, how many slices of American cheese does he eat or how many, you know, nuts does he eat as compared to the general population.

I would assume that his

low-lying ambient exposure would -- would be the same as the general population with the addition of his time on Camp Lejeune.

Q. And you don't try to quantify what that ambient exposure would be for the general population; is that correct?

A. I do not.

Q. Okay.

Can you turn to page 335 for me, please?

A. Okay.

Q. Do you see the middle paragraph starting "Assuming a typical air concentration"?

A. Okay.

Q. And it reads: Assuming a typical air concentration range of 100 to 500 ppt and a breathing rate of 20 cubic meters air per day, the average daily air intake of trichloroethylene can be estimated at 11 to 33 micrograms per day.

Did I read that correctly?

A. You did.

Q. And a ppt is a part per

trillion, right?

A. Yes.

Q. And ATSDR goes on to report that: Average daily water intake of trichloroethylene can be estimated at 2 to 20 micrograms per day assuming a typical concentration range of 2 to 7 ppb in consumption of two liters water per day.

Did I read that correctly?

MS. SULPIZIO: Object to the form.

A. You read it correctly, yes.

Q. Do you have any reason to disagree that the average daily air intake of trichloroethylene can be estimated at 11 to 33 micrograms per day?

MS. SULPIZIO: Object to the form.

A. I mean, I -- I truthfully have no way of knowing. I mean, it's -- it's in this report. I have no reason to believe it's wrong or -- or right. I mean, I -- I have no -- no basis for passing judgment on that.

Q. Okay.

Do you have any reason to disagree that the average daily water intake of TCE can be estimated at 2 to 20 micrograms per day?

A. I have no reason to disagree, and I have no reason to agree.

Q. Do you know whether benzene is commonly found in ambient air?

A. I don't, but my guess is 'cause you're asking that it will be.

Q. And do you know whether benzene is also found in foods?

A. Yeah, benzene I -- I -- I do believe is found in some foods, yes.

Q. Okay.

And do you know if benzene is found in gasoline products?

A. I believe it is, but I can't say definitively.

Q. Okay.

MR. BU: Sharon, can we pull up the next exhibit, please?

(Weiss Exhibit 19, U.S.

Department of Health and Human Services Toxicological Profile For Benzene August 2007, was marked for identification, as of this date.)

THE WITNESS: I will say that the more I'm seeing these, the more that I'm realizing that the estimate of Mr. Fancher's exposure is probably even more of an underestimate because he, in addition to having the time on the base, would have the same exposure as the general population or people walking around and eating or drinking. I don't think his exposure, you know...

MR. BU: Okay.

BY MR. BU:

Q. Do you know that general population exposures are comparable to the Camp Lejeune water exposures?

MS. SULPIZIO: Object to the form.

A. I would have no way of knowing.

Q. Okay.

So you would have no way of knowing how much the Camp Lejeune water exposures underestimate Mr. Fancher's total exposure?

MS. SULPIZIO: Object to the form.

A. I -- I'm sorry, I don't -- explain the question again.

Q. Right.

A. Say the question again.

Q. Sure.

Earlier you had said that the estimated Camp Lejeune exposures are probably an underestimate given Mr. Fancher's, sort of, general population exposures, correct?

A. Yes.

Q. Okay.

What I'm asking you is do you know how significant that underestimate would be? Is it double? Is it triple? Is it 25 percent?

A. No, I don't know.

MS. SULPIZIO: Object to the

form.

BY MR. BU:

Q. All right.

Can you turn to page 272 of Exhibit 19?

A. Okay.

Q. And, I'm sorry, I should have mentioned, Exhibit 19, have you seen this document before?

A. I have not.

Q. Okay. So, this is a toxicological profile for benzene by the ATSDR.

And on page 272, table 6-3 ATSDR reports different benzene concentrations in food.

Is that right?

A. Yes, that's what it appears.

Q. Okay.

And do you have any reason to disagree with the concentrations reported here in Table 6-3?

A. Truthfully, I would have no way of knowing whether this is true or not.

But I have no reason to disagree with it, but I have no reason to agree with it either.

Q. Okay.

A. I -- and I just -- I just want to say that in particular for these documents, like the toxicology profile for benzene, I haven't had a chance -- I haven't seen this before. I haven't had a chance to review it in totality, so it's a little bit difficult for me to comment on it.

Q. Okay.

A. Like for this Table 6-3, I -- I haven't had a chance to analyze, like, what is this they're listing here, number of cases. Like did -- was there only two total cases with cheese that had benzene in it?

So I -- I really struggle with this.

Q. Okay.

For Mr. Fancher, you didn't review any chemical tests to determine

whether his cancer was caused by a toxic exposure, did you?

A. I --

MS. SULPIZIO: Object to the form.

A. No, not that I'm -- no.

Q. Are you aware of any chemical tests or biomarkers for TCE-induced kidney cancer?

A. I'm not aware of any like clinically -- clinical test or any -- any lab test. I -- I would assume there's -- you know, when the exposure's occurring that you may be able to measure, like, you know, metabolites, but I'm unaware of any that being available or done in this case or any cases, to be honest.

Q. Are there clinical features that are more consistent with a chemically-induced kidney cancer?

A. Not that I'm aware of.

Q. Outside of litigation, have you ever diagnosed a patient with chemically-induced cancer?

A. Yes, I believe I have.
Q. Okay.
In what context?
A. Currently in New York we're seeing a lot of patients that are developing malignancies that are related to 9/11, and some of those chemical exposures range from just aerosolization of, you know, asbestos, they range from heavy metals from when the towers came down, cadmium actually. So -- so I do see patients that frequently are part of that situation. I'm not involved in any way in that litigation or the lawsuit or if there even is a lawsuit related, but I see patients frequently through the 9/11 group that's being sent to me for cancers that are felt to be 9/11-related.
Q. Okay.
Are you referring to the World Trade Center Health Program?
A. Yes.
Q. Okay.
And so you treat patients that

are referred to through the World Trade Center Health Program; is that right?

A. I -- I see patients that are part of that program because frequently as part of that program, we -- you know, they -- they require medical records and documentation about their cancer diagnosis. So -- so I see patients that are part of that. They're not being directly referred to me through that program.

Q. Okay.

When they come to you, has someone else already made a determination that they're eligible for medical care through the health program, or are you making that determination?

A. It depends. They're not -- they're not always already part of that program, but frequently when they have the diagnosis, then -- and again, I don't -- I don't put them into the program, but -- but they take the medical records to see if they're applicable for that program and

they should be accepted, so.

I don't make that determination.

Q. Do you know whether that program applies any presumptions to determine whether an exposure caused cancer?

A. I don't know.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Do you know how the World Trade Center Health Program determines whether a cancer's caused by exposure to 9/11?

MS. SULPIZIO: Object to the form.

A. I do not.

Q. You mentioned earlier that Mr. Fancher's diagnosis was much younger than is typical.

Do you recall that?

A. I do.

Q. Okay.

What is a -- what is the typical age range for a kidney cancer diagnosis?

A. I mean, the -- the vast majority

of kidney cancers are diagnosed in people in their sixth and seventh decades of life, but, you know, when you look at the ultra extremes, you know, less than 40 is an extreme. I would say less than 5 percent of kidney cancers are -- are diagnosed in people under the age of 40.

Q. On page 9 of your report, you also describe the latency period for Mr. Fancher's cancer. This is the third full sentence in Section 8.

A. I'm sorry, page 5?

Q. Page 9.

A. Page 9.

Q. Yeah.

A. Okay.

Q. And you write that: The latency period between exposure and the development of cancer is normal.

Do you see that?

A. I'm not going to find it here, but I -- but I -- yes. I mean, I wrote that and I know that, so.

It's very common to have a

latency period from the exposure to when the cancer actually develops.

Q. And Mr. Fancher's latency period was 16 years, correct?

A. That's correct.

Q. What is a normal latency period between exposure and kidney cancer?

MS. SULPIZIO: Object to the form.

A. I mean, I think it -- I think it's dependent -- I don't think we know with a hundred percent certainty. It's probably dependent on what the exposure is to whether it be TCE, PCE or whether it's to a different carcinogen like smoking, but many cancers related to, you know, to -- to environmental exposures have a latency period of 10, 15, 20, sometimes even longer years. Probably that exposure risk probably continues for the life of the individual that was exposed. So -- so I don't think we -- you know, I don't think we can say the, quote, normal latency. It's frequently 10, 15, 20

years.

Q. Okay.

So then would you say a cancer that is diagnosed less than 10 years or longer than 20 years after an exposure is less likely to be caused by that exposure?

A. No.

Q. Okay.

Is a cancer that is diagnosed between 10 and 20 years following exposure more likely to be caused by that exposure?

MS. SULPIZIO: Object to the form.

A. Not necessarily.

Q. And why is latency not necessarily indicative of causation?

A. I'm not sure I understand the question.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Right.

So -- so, in your report you make a comment, right, that Mr. Fancher's

latency was normal, right?

A. Yeah, that it was normal to have a latency between an exposure and when the cancer develops. That's very common.

Q. Okay.

Can you turn to page 12 of your report for me, please?

A. Okay.

Q. The last sentence of this section before Section 10 you write: After exposure to carcinogenic chemicals, there is well-established latency period before the actual development of cancer and 16 years is well within the normal range.

Do you see that?

A. I do.

Q. All right. And so I had asked you what is a normal range.

Do you recall that?

A. I do.

Q. All right.

And so would a normal range be between 10 and 20 years?

A. So, I think -- I think I misunderstood your question before because I thought you meant just what's normal range for a latency for cancer -- I thought you were specifically saying for kidney cancer.

This is referring to a latency period for cancerogenic exposure for a cancer development in general, and -- and we have a lot more experience in things like lung cancer, frequently, you know, 10, 15, 20 years. I don't think -- it's not like a confidence interval. We can't say 10 to 20 years. But 16 years is -- is within a, like, a normal range for how latency periods work for cancer development.

Q. Okay.

A. My point is it's not uncommon for it to take 16 years from the exposure to develop a -- to develop a cancer.

Q. All right.

Your understanding of a normal range for latency is based on what we know

about smoking and latency.

Are there other latencies that we know about that you've considered?

MS. SULPIZIO: Object to the form.

A. I mean, there's lots of latency periods that we know about. We know about latency periods for skin cancer, you know, frequently 15, 20 years after a high-intensity skin -- skin damage or skin burning.

I'm just trying to think off the top of my head.

You know, chronic pancreatitis, people can have chronic pancreatitis for 10, 15 years and then they develop pancreas cancer. There's been a nidus, you know, 15 years after the development. So -- so many, many cancerous will develop 15, 20 years after the -- the exposure or the nidus or what we believe is the cause.

Q. And just so as there are latencies, sort of, within this normal range, there must be latencies outside of

that normal range, right?

MS. SULPIZIO: Object to the form.

A. Yeah, I'm sure there are.

Q. Okay.

So to go back to what I was trying to get at earlier, if a latency is outside of that normal range, whatever it is, would you agree it's less likely to be caused by the exposure?

MS. SULPIZIO: Object to the form.

A. Not necessarily. Not necessarily. I think, you know, different people have, you know, different thresholds. Different people may have longer or shorter latencies based upon, you know, their own susceptibility to cancer. And so I can't say that if you're outside of the normal range that now it's less likely to be related to that exposure.

I -- my comment is just that it's pretty normal to have a -- a latency

period of 16 years like -- like Mr. Fancher had.

Q. Okay.

Do you know whether the latency periods vary by different types of cancer?

A. Yeah, I mean, there's, yeah, different cancers, different environmental exposures may have a different, you know, latency period. Like, you know, when they dropped an atomic bomb in Japan, those patients developed cancer pretty quick when they were exposed to high intensity iodizing radiation and different types of cancer. So, yeah, I think there's probably a range.

Q. You also mentioned you don't think Mr. Fancher's cancer's related to a genetic predisposition.

Do you recall that?

A. I do recall it.

Q. Okay.

All else being equal, Mr. Fancher's young age of diagnosis would be considered suspicious for a genetic

predisposition.

Is that fair to say?

A. I wouldn't say that it's suspicious. I mean, you know, when you have a young patient that develops a cancer, you're -- you're obviously trying to figure out why.

You know, a familial predisposition usually has a family history of such cancers, like kidney cancers. In a -- in a young patient, you're usually looking for did they develop a cancer in both kidneys or just one kidney, which wasn't him. You're looking for was it multiple tumors in the -- in the same kidney or was it one. He had a solitary lesion.

Just age in of itself I don't think would push you are towards saying this is a genetic predisposition for this individual. He really had no other risk factors.

Q. Okay.

In your report on page 11

towards the bottom of that paragraph -- or, bottom of that page, I'm sorry, you write: His history would not warrant genetic testing under the current standard of care due to the extreme low likelihood of revealing a genetic effect.

Do you see that?

A. I do.

Q. Okay.

Were there guidelines or resources you consulted for determining the current standard of care for genetic testing?

A. No. I mean, there -- there may be guidelines, but I didn't consult any of the guidelines. I looked at his -- I looked at his medical history, his family history, his presentation and what the disease looked like at presentation, and I based -- and -- and I based the opinion that -- that it -- you know, there was no clear indication that there -- there was no indication that this was genetically caused.

Q. Okay.

Can you turn to page 13 of your report for me, please?

A. Okay.

Q. So, you opine that the treatment and care Mr. Fancher has received and is now receiving is fair, reasonable, and medically necessary, right?

A. I agree.

Q. Okay.

What treatment and care are you referring to?

A. Well, the original treatment of, obviously, undergoing the nephrectomy, the radical nephrectomy.

Can you rephrase the question? Do you want to know what he's undergoing currently or whether -- what he underwent?

Q. Is there treatment Mr. Fancher is currently receiving that's related to his kidney cancer?

A. I don't know if he's still being followed. You know, clearly he is now 20 -- more than 25 -- yeah, more than 25

years out. So the chances of recurrence are very low, but they're still possible. So, many times these patients are followed for life for a potential recurrence.

I would assume that he's being followed for the fact that he has one kidney, so he's at least at risk for developing renal insufficiency, liver -- you know, renal failure in the opposing kidney. So far at least, based upon the most recent records, his -- his remaining kidney is functioning, but I got to believe that he's being followed for that.

I'm unaware of any other, you know, treatment that he's receiving in terms of mental health and so forth. Obviously patients that are diagnosed with a cancer, it's a -- it's a dramatic event for them. They're told they have a cancer, then they're undergoing surveillance, and, you know, sometimes they have long-term sequelae related to that. And -- and I'm not aware of treatment that he's receiving for that,

but it's certainly possible.

Q. Is there any other treatment or care that you are aware of that Mr. Fancher is receiving surveillance?

A. No.

Q. Okay.

A. I believe he has a hernia or at least a diastasis or an outpouching from where he had the incision 'cause I know he had a wound infection after surgery and frequently those patients get hernias. I don't think he's undergone any interventions or treatment for that, but they can sometimes be somewhat, you know, lifestyle-altering having that.

Q. Other than the hernia and the kidney removal, are there any other permanent effects that you attribute to Mr. Fancher's kidney cancer diagnosis?

A. I mean, the tough thing for these patients that have been treated for cancer, truthfully, is some of the, you know, lifelong anxiety of is it going to come back and the -- the mental health,

you know. Some of these patients, you know, even can get, sort of like, post-traumatic distress disorder for being treated for their cancers.

But my understanding is -- is, you know, his tumor was removed. It was a T3N0 tumor. He has not had a recurrence of that cancer yet. Although, you know, again the likelihood is low, but it still could happen. I've seen recurrences of kidney cancers this far out. It happens. Pretty uncommon.

And then I would assume routine health as part of that related to the fact that he has one kidney and needs to be followed for that and managed appropriately, and it seems -- seems like he's, you know, in relatively decedent shape.

Q. Okay.

Do you know whether the standard of care would be that Mr. Fancher requires cancer surveillance?

A. So, the standard of care would

say that he could -- that he could have stopped surveillance, but I personally have experience with patients that have recurred long after the standard of care.

So, standard of care I would say at this point he probably does not need to undergo surveillance anymore.

Q. Okay.

A. But that doesn't mean that he can't recur at this point.

Q. When you were discussing Mr. Fancher's hernia, is that the same or different from his surgical scar?

A. It's different.

Q. Okay.

Do you know whether Mr. Fancher's surgical scar requires any interventions?

A. Currently I don't -- I don't believe it has.

Q. Okay.

On page 13 you also opine for item 4: The medical billing relating to Mr. Fancher's kidney cancer diagnosis, the

surgery to remove his kidney, and the follow-up treatment related to his kidney cancer was reasonable and medically necessary.

Do you see that?

A. Yes.

Q. Okay.

How did you determine whether medical billing is reasonable?

A. I -- I didn't review actual billing for this, but the billing of performing a -- you know, a -- a radical right nephrectomy and the aftercare and everything else would be appropriate. You -- you would bill for those services in someone that has cancer. But I certainly didn't review, like, the actual bills and make sure that the bills were the proper amounts and so forth.

Q. Okay.

So you're not opining to the specific costs for medical services.

Is that fair to say?

MS. SULPIZIO: Object to the

form.

A. I mean, I didn't review the actual costs of his procedures.

Q. Okay.

When did you first become aware of the Camp Lejeune water litigation?

A. I think the first I became aware of it was -- was -- was when I was contacted by Mr. Mandell about, you know, would I be willing to review a case to see if, you know, I wanted to be a -- a witness in the case.

In hindsight maybe I saw some -- you know, on TV maybe a commercial here or there, but to be honest, there's so many of these commercials that I don't remember the specifics. But I think the first I really became aware of the case was -- was last year when I was contacted by Mr. Mandell, you know.

Q. When you say "Mr. Mandell," do you know if that was Mark Mandell or Zach Mandell?

A. It was Zach Mandell.

Q. When was this that Zach Mandell reached out to you?

A. Like the -- the middle of 2024, I want to say back in maybe September or August, around that time. I can't give you the exact date, but I think that's when he first reached out to me, I believe.

Q. At that initial meeting in -- whenever you had your first contact with Zach Mandell, do you recall how the Camp Lejeune water litigation was described?

A. I don't.

MS. SULPIZIO: Object to the form.

To the extent that it has any questions in regard to, you know, privilege, I instruct you not to answer.

BY MR. BU:

Q. Do you recall when you were retained by plaintiffs' counsel in this litigation?

A. When I was contacted, I was

essentially retained. I don't do a lot of, you know, witness testimony and -- and basically when I do, I -- if I'm retained, that's to review the -- the materials. It was probably -- it -- it must have been around the same time, September.

Q. When you say "retained to review materials," do you mean as like a consulting expert or a medical consultant?

A. I'm not sure I understand the difference --

MS. SULPIZIO: Object to the form.

A. -- to be honest.

Q. Sure. Let me ask it this way.

How much of your annual income would you say is related to assisting in legal proceedings?

A. Very little. I could calculate it, I guess, but it's probably far under 5 percent. Maybe 5 percent.

Q. And of the legal proceedings that you've assisted in, I think you mentioned you testified in maybe six; is

that right?

A. I've been deposed about five or six times. I maybe will do, you know, either a medical consulting or a legal case maybe -- maybe once or -- or twice a year, tops. It doesn't represent very much of my efforts in my income.

Q. Okay.

When you were retained by plaintiffs' counsel, did you execute a retainer agreement or some other type of contract?

MS. SULPIZIO: Object to the form.

A. If I -- if I'm involved in a case like this, I have a standard retainer sheet just that describes what, you know, the hourly rate will be and so forth.

And so, I'll be honest, I don't understand the -- I should, I guess. I don't understand the legal back-and-forth of what I was, quote, retained versus I just -- you know, I give them a -- basically a fee schedule so that they know

what I charge and then that's it.

MR. BU: Okay.

Sharon, can we pull up tab 27, please?

(Weiss Exhibit 20, invoices and fee schedule of Matthew J. Weiss, MD, MBA, Bates CL_PLG-EXPERT_WEISS_0000000001-006, was marked for identification, as of this date.)

BY MR. BU:

Q. I'm handing you what's been marked Exhibit 21.

A. Okay.

Q. Have you seen this document before?

A. I have.

Q. And what is this document?

A. This is a copy of an invoice that I sent to the Mandells on November of 2024.

Q. Okay.

And if you flip to, I guess, the next few pages, there's also a fee

schedule for deposition and for court appearances.

Do you see that?

A. Correct, yes.

Q. Okay.

Is this the retainer sheet that you were describing earlier?

A. This is.

Q. Okay.

Do you know whether there are any other agreements that you signed with plaintiffs' counsel?

MS. SULPIZIO: Object to the form.

A. Not that I'm aware of.

Q. Okay.

And you drafted this retainer agreement; is that correct?

A. This was drafted by me.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Okay.

Do you know whether you would

have done any work on this case before September 25th, 2024?

A. I don't recall the exact dates, to be honest. I don't remember. I -- I don't think so.

Oh, wait now. Here. Here, 'cause I just -- yeah, September 25th, 2024, that would have been the first day that I worked on it, correct.

Q. Okay.

Is there work in this litigation that you performed but did not bill for?

MS. SULPIZIO: Object to the form.

A. I don't think so.

Q. Okay.

So you wouldn't have, like, written off any time?

MS. SULPIZIO: Object to form.

A. No, I don't think so.

Q. Okay.

Is there any compensation you expect to receive other than your hourly rate for casework, testimony fees, and

travel costs?

A. No.

Q. Is your fee schedule on this case the same as your fee schedule used in other cases?

A. Exactly the same, yes.

Q. The second invoice is from February 5th, 2025.

Do you see that?

A. I do.

Q. Okay.

Have you submitted any invoices to plaintiffs' counsel since February 5th, 2025?

A. I have not.

Q. Have you performed work on this case since January 25th, 2025?

A. I have.

Q. All right.

About how much time have you spent on this case since January 25th, 2025?

A. I would say maybe five or six hours re-reviewing, 'cause obviously

things that I reviewed back in September of 2024, in preparation for the deposition I wanted to go back and re-review. I would guess, I don't know, five or six hours.

Q. Did you speak with any other plaintiffs' experts in the course of preparing your reports in this case?

A. I did not.

Q. You mentioned spending five to six hours on depo prep.

Can you tell me what you did to prepare for your deposition today?

MS. SULPIZIO: Objection only to the extent that it reveals communications between him and counsel.

Go ahead, you may answer.

A. I went back and reviewed what I remember being the most critical papers related to this case. I went back and reviewed depositions by Mr. Fancher. I reviewed his medical record again. I reviewed some of the -- the expert, I

wouldn't call it testimony, but the experts of -- of Dr. Reynolds, Dr. Hatten, Dr. Bird. Basically it was just reading through a lot of documents that I had read before, but I wanted to refresh my memory.

Q. Okay.

Without telling me what you may have discussed, did you meet with any attorneys to prepare for your deposition?

MS. SULPIZIO: Object to the form.

A. I did.

Q. Okay.

How many times did you meet with attorneys?

A. Two short meetings.

Q. How long were those meetings?

How long was the first meeting, I'm sorry?

A. Maybe a half an hour.

Q. Do you remember which attorneys you met with?

A. I guess the question is can I -- can I say that?

MS. SULPIZIO: You can answer.

A. Yeah, Mr. Mandell and -- and Gabby.

Q. Okay. So, there are two Mandells.

Do you know if you met with Zach or Mark?

A. Sorry. I met with Zach.

Q. How long was the second meeting with the attorneys?

A. About the same, about a half an hour. It was a -- it was -- it was really a phone call.

Q. And who did you meet with for that second meeting?

A. The same.

Q. Zach Mandell and Gabby?

A. Zach Mandell and Gabby.

Q. Okay.

Was anyone other than a plaintiffs' attorney present for either of those meetings?

A. No.

Q. Have you had communications with

anyone other than a plaintiffs' attorney to prepare for your deposition?

A. No.

Q. Have you had any communications with Mr. Fancher?

A. No.

Q. Have you had any communications with any of the plaintiffs in the Camp Lejeune water litigation, that you're aware of?

A. No.

Q. Have you had any communications with any of Mr. Fancher's treating physicians?

A. No.

Q. The cases in which you've served as an expert witness, how many of those cases, other than this one, have involved exposures to toxic substances?

A. None of them.

Q. Were the other cases medical malpractice cases?

A. Yes.

Q. Other than this litigation, is

all of your work in litigation related to medical malpractice?

MS. SULPIZIO: Object to the form.

A. I would say up -- yes. Yes. Yes.

Q. There are no other types of cases that you've worked on?

A. No.

Q. Prior to this case, have you ever worked as an expert witness for the Mandell law firm?

A. No, never before.

Q. Okay.

Have you ever worked as an expert witness for The Bell Legal Group?

A. No.

Q. To the best of your knowledge, have you ever worked as an expert witness for any of the other plaintiffs' firms involved in the Camp Lejeune water litigation?

A. I don't know what all the other plaintiffs' firms are, but I -- I can't

imagine that I have.

Q. Okay.

Have you ever worked as an expert witness in a case involving the United States?

A. No.

MR. BU: Could we go off record?

THE VIDEOGRAPHER: The time right now is 3:21 p.m., and we're off the record.

(Recess taken.)

THE VIDEOGRAPHER: The time right now is 3:30 p.m., and we're back on the record.

BY MR. BU:

Q. Dr. Weiss, during the break, did you discuss your deposition testimony with anyone?

A. I did not.

Q. Okay.

Is there anything that you've testified to today that you would like to clarify or correct?

A. No.

Q. Can you turn to page 12 of your report, Exhibit 1, for me, please?
A. Okay.
Q. In that first full paragraph you write: Mr. Fancher was also exposed to PCE, benzene, and VC, which are known carcinogens and --
A. Hold on. I'm having trouble finding you.
You said page 12 of my -- of my report, correct.
Q. Yes, page 12 of your report.
A. Okay.
Q. The first full paragraph at the top, the third line of that paragraph beginning "Mr. Fancher was also exposed."
A. Okay. Yes.
Q. So, you write: Mr. Fancher was also exposed to PCE, benzene, and VC, which are known carcinogens and also associated with the development of kidney cancer.
Did I read that correctly?
A. Yes.

Q. And you go on to write: This exposure likely and probably would have been additive or synergistic when combined with his TCE exposure.

Did I read that correctly?

A. Yes.

Q. Can you explain what you mean by "additive or synergistic"?

A. What I mean is that he had -- you know, if he has -- obviously has the TCE exposure which has its own carcinogenic, you know, potential, and that these other substances, PCE, benzene, and VC, also have a carcinogenic potential, and then since he was exposed to all four, he's -- each of them would have its own cancer potential. It's not like one would be protective over the other. So I would say that they're at least additive.

And it's even possible that some carcinogens when they're combined can have a synergistic effect, meaning they can make them even more potent than just being

additive.

Q. Did you review any literature about how these different chemicals interact with one another?

A. I did not.

Q. Okay.

Did you review any literature about how they are metabolized together?

A. Not together.

Q. Okay. Okay.

Are there any additional materials you feel would be relevant to your report that you did not have a chance to review?

A. No.

Q. Okay.

And do you -- do you consider yourself an epidemiologist?

A. No.

Q. All right.

Have you ever been a principal invest -- investigator for an epidemiological study?

A. No.

Q. Do you consider yourself a toxicologist?
A. No.
Q. Do you hold yourself out as an expert in environmental health?
A. No.
Q. Do you hold yourself out as an experiment in occupational medicine?
A. No.
Q. Do you hold yourself out as an expert in risk assessment?
A. Just general risk assessment? I wouldn't say an expert. I mean, we utilize risk assessment daily life in -- in medical care and in reviewing the literature all the time, but I wouldn't say I'm an expert in -- in risk assessment, per se.
Q. Okay.
Have you ever published peer-reviewed literature regarding the effects of TCE on cancer?
A. I have not.
Q. Have you ever published

peer-reviewed literature regarding the effects of PCE, benzene, or vinyl chloride on cancer?

A. I have not.

Q. To the best of your knowledge, have you ever treated a patient with cancer related to exposure to water at Camp Lejeune?

A. Not that I'm aware of.

Q. Okay.

Have you ever been asked to write a letter to the Department of Veterans Affairs related to benefits for Camp Lejeune exposures?

A. No.

Q. Does your practice require training on --

MR. BU: Let me rephrase that. Sorry.

Q. Does your practice require training on the potential health risks related to TCE exposure?

MS. SULPIZIO: Object to the form.

A. I mean, not specifically for TCE exposure, no.

Q. What about specifically for PCE, vinyl chloride, or benzene?

MS. SULPIZIO: Object to the form.

A. No. I mean, my practice requires knowing about environmental exposures and their -- and their of developing cancer, but I wouldn't say specifically these.

Q. Other than in this case, have you ever offered an expert opinion in another case involving exposure to TCE, PCE, vinyl chloride, or benzene?

A. No.

Q. Okay.

Other than in this case, have you ever offered an opinion about the etiology of a kidney cancer?

A. No.

Q. Other than in this case, have you ever offered an expert opinion on the etiology of a -- of any type of cancer?

MS. SULPIZIO: Object to the form.

A. No, I don't believe so.

Q. Do you feel that your testimony that you've given today is complete and accurate to the best of your ability?

A. I do.

Q. Is there anything that you've testified to today that you'd like to clarify or correct?

A. No.

MR. BU: Okay.

No further questions.

MS. SULPIZIO: I just have one quick question for you, Doctor.

EXAMINATION BY

MS. SULPIZIO:

Q. You were asked some questions today about prospective randomized trials, and you talked about the connections between them and causation.

Do you recall that conversation?

A. I do.

Q. Would you agree that you are

able to prove causation in cases like Mr. Fancher without prospective randomized control trials?

A. Absolutely.

Q. Okay.

A. A prospective randomized trial for a situation like this would -- would be unethical, and the only way you can draw conclusions is by using the best available retrospective data to -- to formulate an opinion and a conclusion.

Q. And that's exactly what you did in your report to make your opinions for Mr. Fancher, correct?

A. Correct.

MS. SULPIZIO: I don't have any further questions.

THE VIDEOGRAPHER: The time right now is 3:37 p.m., and we're off the record.

MS. SULPIZIO: Doctor, you're going to read and sign, right?

THE WITNESS: Yes.

(Time noted: 3:37 p.m. EDT)

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.

A C K N O W L E D G M E N T

STATE OF)
:ss
COUNTY OF)

I, MATTHEW J. WEISS, M.D., MBA, hereby certify that I have read the transcript of my testimony taken under oath in my deposition of June 30, 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.

_____
MATTHEW J. WEISS, M.D., MBA

Signed and subscribed to before me this _____ day of _____, 20__.

_____
Notary Public, State of

E R R A T A

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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, MATTHEW J. WEISS, M.D., MBA, was duly remotely 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 not financially interested in the action.

_____

COURT REPORTER
Registered Merit Reporter
Certified Realtime Reporter
Notary Public
Dated: July 9, 2025

LAWYER'S NOTES

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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.

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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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