Exhibit 167

IN THE UNITED STATES DISTRICT COURT
FOR THE EASTERN DISTRICT OF NORTH CAROLINA
SOUTHERN DISTRICT
No. 7:23-CV-897

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IN RE: :
: :
CAMP LEJEUNE WATER LITIGATION :
: :
This Document Relates to: :
ALL CASES :
- - - - - - - - - - - - - - - - - - - -X

Videotaped deposition of AMELIA K. BOEHME, Ph.D, MSPH, taken at the offices of Weitz & Luxenberg, 700 Broadway, New York, New York, before Clifford Edwards, Certified Shorthand Reporter and Notary Public, in and for the State of New York on Friday, May 2, 2025, at 10:02 a.m. ET.

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(Reporter's Note: Exhibits retained by the court reporter and forwarded to Golkow for production.)

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

Today's date is May 2nd, 2025 and the time is 10:04 a.m.

This deposition is taking place at 700 Broadway, New York, New York, in the matter of Camp Lejeune Water Litigation. The deponent is Dr. Amelia Boehme.

Counsel, please identify yourselves for the record.

MR. MICELI: David Miceli for the plaintiffs.

MS. MIRSKY: Sara Mirsky for the United States.

MS. PLATT: Elizabeth Platt for the United States.

MS. ELLISON: Anna Ellison on behalf of the United States.

MS. LAMACCHIA: Leslie LaMacchia on behalf of PLG.

MR. TELAN: Patrick Telan on behalf of PLG.

THE VIDEOGRAPHER: The court

reporter is Cliff Edwards and will now swear in the witness.

AMELIA KATHERINE BOEHME

having first been duly sworn, deposed and testified as follows:

DIRECT EXAMINATION

BY MS. MIRSKY:

Q Good morning, Dr. Boehme.

A Morning.

Q My name is Sara Mirsky. We just met before going on the record and I represent the United States.

Do you understand that this is a court proceeding even though we are not in a courtroom?

A Yes.

Q And you are under oath; correct?

A Correct.

Q Do you understand that you are obligated to tell the truth?

A Yes.

Q A court reporter is taking down everything that we say. It's important that you answer any question verbally. So for example, you must say "yes" or "no" rather than nodding or shaking your head.

Do you understand that?

A Yes.

Q Please talk at a reasonable pace so the reporter can record everything.

And we should also try not to interrupt one another, otherwise the reporter will not be able to record us accurately. Please wait until I have finished my question before you start to answer and I will not interrupt you while you're speaking.

Once the deposition is complete, you'll be given an opportunity to read a transcript of your testimony and to make any corrections. You will then be asked to sign it.

Does all of that make sense?

A Yes.

Q If you don't understand a question, please let me know and I'll try to clarify. And if you don't ask for a clarification, I will assume you've understood the question; is that fair?

A Understood.
Q During the deposition you may hear Mr. Miceli say, Objection.
But unless Mr. Miceli instructs you not to answer the question, please answer the question after the objection has been made; does that make sense?
A Understood.
Q Okay. Is there any reason why you're unable to give your most truthful and accurate testimony today?
A No.
Q Is there any reason your memory might be impaired today?
A No.
Q Are you currently taking any medication that might impair you?
A No.
Q We will take occasional breaks. You may ask for breaks.
Please just answer any pending question that I've asked before we take a break; okay?
A Understood.
Q Now, before we get started, there are

some technical terms that are involved in your report and the related studies and I would just like to get a com- -- few common abbreviations on the record to ensure that we are on the same page and for facility of reference moving forward.

So trichlorethylene is TCE; correct?

A Correct.

Q And tetrachloroethylene is PCE or PERC?

A Correct.

Q Parkinson's disease can be abbreviated as PD?

A Correct.

Q And 1-trichloromethyl-1,2,3,4-tetrahydro-beta-carboline, can we talk about that as TaClo, please?

A Sure.

Q Great. Thank you.

And the Agency for Toxic Substances and Disease Registry is abbreviated as ATSDR; correct?

A Yes.

Q So it will be clear if I use these abbreviations moving forward?

A Yes.

Q Okay.

MS. MIRSKY: So I'd like first to enter as Exhibit 1.

(Whereupon, Boehme's Exhibit 1, Subpoena for Amelia Boehme, was marked for identification.)

BY MS. MIRSKY:

Q Do you recognize this document?

A Yes.

Q And it is the subpoena for your deposition today; correct?

A Correct.

Q Have you reviewed the request for production of materials at the end?

A Yes.

Q Do you have any responsive materials to produce?

A No. Other than I believe my bills and invoices were sent previously.

Q Okay. And --

MR. MICELI: And we provided the updated CV.

MS. MIRSKY: Correct.

BY MS. MIRSKY:

Q Are you withholding any documents that

would be responsive?

A No.

Q Let's just quickly go through this Attachment A.

So what was requested includes, number one, all e-mails, letters, correspondence, text messages, conversations chats, voicemails, data, technical files, or other communications pertaining to Camp Lejeune sent or received prior to your retention as an expert in this matter, including but not limited to, from, or with Morris Maslia.

Do you have any of those communications with Morris Maslia?

A None.

Q Do you have any of those communications with David Savitz?

A None.

Q Okay. Number two, all e-mails, letters, correspondence, text messages, conversations, chats, voicemails, data, technical files, or other communications pertaining to Camp Lejeune, including but not limited to...

Do you have any of those communications with Robert Faye?

A No.
Q Jason Sautner?
A No.
Q Rene Suarez-Soto?
A No.
Q Susan Martel?
A No.
Q Scott Williams?
A No.
Q Frank Bove?
A No.
Q Mike Partain?
A No.
Q Jerry Ensminger?
A No.
Q Lori Freshwater?
A No.
Q Paul Rosenfeld?
A No.
Q Do you have any communications with Dr. Samuel Goldman?
A No.
Q Do you have any communications with Dr. Ray Dorsey?

A No.
Q You have published with Dr. Dorsey several times before; is that correct?
A I don't believe so.
Q Okay. We can go back to that.
A I would have to check my CV.
Q Okay. Do you have any e-mails, letters, correspondence, text messages, conversations, chats, voicemail, or other communications to, from, or with any individual who has filed a claim with the Department of Navy or the Eastern District of North Carolina pursuant to the Camp Lejeune Justice Act of 2022?
A No.
Q And as discussed, you provided your invoices today?
A Yes.
Q Did you bring any documents to the deposition today?
A Just a copy of my report.
Q Okay. Are there any notations on that report?
A None.
Q Okay. And as you just referenced, you

prepared a report in this litigation; correct?
A Correct.
Q Okay.
MS. MIRSKY: I'd like Tab 2, please.
(Whereupon, Boehme's Exhibit 2, Report by Amelia Boehme Dated December 8, 2024, for Litigation, was marked for identification.)
BY MS. MIRSKY:
Q Okay. Do you recognize this document?
A Yes.
Q And this is your complete December 8th, 2024 report submitted in this litigation; correct?
A Yes.
Q And on page 22 of this report is your signature; correct?
A Yes.
Q Okay. So I'll ask you first to turn to the green tab.
Is this the CV -- is this your CV as it existed on December 8th, 2024?
A This was the one I had provided prior to that. There might be some missing publications. But otherwise it should contain most of --

everything I had done up until May 1st, 2024.

Q Okay. And as Mr. Miceli referenced, this morning we were provided with an updated CV?

MS. MIRSKY: And I'd like to mark that as Exhibit 3, please.

(Whereupon, Boehme's Exhibit 3, Updated CV of Amelia Boehme, was marked for identification.)

BY MS. MIRSKY:

Q This is your current CV; is that correct?

A Correct.

Q And does this document represent a complete representation of your educational and professional background?

A It represents my education, my profession, and anything that would pertain to tenure.

Q Okay. And is there any information outside of those categories that you did not include?

A So I don't include my expert witness work on my CV. I don't include volunteer work. I serve as a methodologist occasionally for organizations and that's outside consulting and not to be

1 considered for tenure, so I don't con- -- include
2 that here.
3 Q Okay.
4 A I also don't include media appearances or
5 community outreach work.
6 Q Okay. Do you have that information
7 compiled elsewhere?
8 A I could put it together, yeah.
9 Q But you don't have it readily --
10 A Not readily --
11 Q -- available?
12 A -- available.
13 Q Okay. Is there any information since
14 April 4th, 2025, which is the date that this CV was
15 updated, about the categories that you described
16 including in your CV that is not in this document?
17 A It should all be in here.
18 Q Okay. You have a bachelor of science;
19 correct?
20 A Yes.
21 Q And that's from the University of
22 Alabama; correct?
23 A Alabama in Huntsville.
24 Q Okay. What was your major?

A Biological sciences and chemistry.
Q And you have a science -- a ma- -- masters of science in public health in epidemiology; correct?
A Yes.
Q And that is from the University of Alabama at Birmingham?
A Yes.
Q And you have a Ph.D. in epidemiology; correct?
A Yes.
Q And that is also from the University of Alabama in Birmingham?
A Yes.
Q You hold yourself to be an expert in epidemiology; correct?
A Yes.
Q What is the basis of your expertise in epidemiology?
A So my expertise is multifactorial, based off of my education, my post-doctoral experience in my post-doc doing a neuroepidemiology fellowship. Then from there, I have many publications in epidemiology, neuroepidemiology.

I transitioned into academia after my fellowship. I have served as one of the co-PIs of the neuroepidemiology training program at Columbia. And in that role I was training neuroepidemiologists.

I've served as the head of the doctoral committee for the qualifying exam, which is what is used to test doctoral students on their epidemiologic knowledge. I serve as a peer reviewer for many different journals.

Additionally, I am a reviewer for NIH grants, specifically with NINDS, the National Institute of Neurological Disorders and Stroke, where I serve as an epidemiologic methodologist during grant reviews there.

I have mentored many students, medical students, doctoral students, and master students, through epidemiology. I teach epidemiology courses at NYU and Columbia, and I've been functioning as an epidemiologist for over a decade.

Q Okay. Thank you.

MR. MICELI: Excuse me, you might want to slow down for him.

And I don't mean to --

THE WITNESS: Oh, apologies.
MR. MICELI: -- interrupt and to be inappropriate. Just --
THE WITNESS: That's okay.
MR. MICELI: -- you may want to slow down for the court reporter before his fingers start smoking.
THE WITNESS: Apologies.
BY MS. MIRSKY:
Q Okay. And do you hold yourself out to be an expert in neuroscience?
A Not in neuroscience, no.
Q Okay. Do you hold yourself out to be an expert in toxicology?
A No.
Q And are you qualified to judge the accessi- -- acceptability of toxicological findings?
A No.
Q Have you ever been qualified as an expert in toxicology before?
A Never.
Q Do you hold yourself out to be an expert in genetics?
A No.

Q And are you qualified to judge the acceptability of findings regarding genetics?

A No.

Q Have you ever been qualified as an expert in genetics before?

A No.

Q Are you qualified to diagnose Parkinson's in humans?

A No.

Q Parkinson's in humans is diagnosed based on motor function; correct?

MR. MICELI: Object to the form.

A My understanding from reading the literature and working with my epidemiology -- neuroepidemiology fellows is it's a clinical diagnosis. There is no blood test, per se, or imaging that can be done, and so it's based off of clinical diagnosis.

BY MS. MIRSKY:

Q And in that clinical diagnosis, a neurologist would perform a physical evaluation to look for symptoms such as bradykinesia, tremors, rigidity; correct?

A That is my --

MR. MICELI: Object to the --
Excuse me.
THE WITNESS: Oh, sorry.
MR. MICELI: Object. Foundation.
A That is my understanding from reading information on how PD is diagnosed.
BY MS. MIRSKY:
Q Okay. You have never published on TCE; correct?
A Correct.
Q You've never published on PCE; correct?
A Correct.
Q You've never published on vinyl chloride; correct?
A Correct.
Q You've never published on benzene; correct?
A Correct.
Q You've never published on DCE; correct?
A Correct.
Q But you have published on Parkinson's before; correct?
A Yes.
Q Okay.

MS. MIRSKY: 5tab 3, please.

MR. MICELI: Thank you.

(Whereupon, Boehme's Exhibit 4, Study Titled "Parkinson's disease Aggravation in Association with Fine Particle Components in New York State," Co-Authored by Amelia Boehme, was marked for identification.)

BY MS. MIRSKY:

Q So what's been marked as Exhibit 4 is a study titled "Parkinson's disease" -- excuse me -- "Parkinson's disease aggravation in association with fine particle components in New York State."

Do you recognize this document?

A Yes.

Q And you are an author on this study; is that correct?

A Yes.

Q This study did not analyze TCE, PCE, vinyl chloride, benzene, or DCE, specifically; correct?

A Correct.

Q And this study did not analyze any solvents; correct?

A Correct.
Q This study also did not analyze the causation of Parkinson's; correct?
A Correct.
Q Okay.
MS. MIRSKY: Tab 4, please.
MR. MICELI: Are you done with that one?
MS. MIRSKY: Yes.
MR. MICELI: Okay. Thank you.
MS. MIRSKY: There you go.
(Whereupon, Boehme's Exhibit 5, Study Titled "Motor Phenotype Classification in Moderate to Advanced PD in BioFIND Study," Co-Authored by Amelia Boehme, was marked for identification.)
BY MS. MIRSKY:
Q And what's been marked as Exhibit 5 is a study called "Motor Phenotype Classification in Moderate to Advanced PD in BioFIND Study."
Do you recognize this document?
A Yes.
Q And you're an author on this study;

correct?

A Correct.

Q This study did not analyze TCE, PCE, vinyl chloride, benzene, or DCE; correct?

A Correct.

Q And this study did not analyze any solvents; correct?

A Correct.

Q This study did not analyze the causation of PD; correct?

A Correct.

Q Okay.

MS. MIRSKY: Next.

MR. MICELI: You might want to put them face down when you are done with them.

(Whereupon, Boehme's Exhibit 6, Study Titled, "Comparative study of cerebrospinal fluid alpha-synuclein seeding aggregation assays for diagnosis of Parkinson's disease," Co-Authored by Amelia Boehme, was marked for identification.)

BY MS. MIRSKY:

Q Okay. What's been marked as Exhibit 6 is a study entitled, "Comparative study of cerebrospinal fluid a-synuclein seeding aggregation assays for diagnosis of Parkinson's disease."

Do you recognize this document?

A Yes.

Q And you are an author on this as well; correct?

A Yes.

Q This study did not analyze TCE, PCE, vinyl chloride, benzene, or DCE; correct?

A Correct.

Q And this study did not analyze any solvents; correct?

A Correct.

Q This study did not analyze the causation of PD; correct?

A Correct.

Q Okay. Let's go back to your CV, which was marked as Exhibit 3.

A Uh-huh.

Q That's your updated CV.

On page two at the top, there are two

entries for a company spelled A-e-t-i-o-n.
Can you pronounce that, please?
A Aetion.
Q Aetion.
MR. MICELI: Can you tell me the page again? I apologize for interrupting.
MS. MIRSKY: That's okay.
Two.
MR. MICELI: Thanks.
BY MS. MIRSKY:
Q What is Aetion?
A Aetion is a -- it's a company that does two functions.
They've developed software that helps analyze longitudinal studies in epidemiology, and they also do scientific consulting.
I -- my function within Aetion is through scientific consulting as a methods expert.
Q Okay. And it says that from 2022 to 2025 you served as the director of science; is that correct?
A Yes.
Q And what did that position entail?

A So I was serving as a methodologic expert and assisting in study design, assisting in assessment of causality for certain studies, and really building into thought leadership on how to use epidemiologic data for a number of different situations.

Q And can you give a couple of examples of what those situations would be?

A They are currently ongoing and I do have an NDA, but I can -- I am allowed to speak to who they are with. They are with the FDA.

Q Okay. And your CV states that starting in 2025, you are now the senior director of science; is that correct?

A Yes.

Q What's the distinction between the two roles?

A I was promoted for functioning as a senior director through my thought leadership work.

Q And is there any difference in terms of your responsibilities as a senior director as opposed to director?

A No. I had been functioning as a senior director.

Q Okay. Understood.
And you are also currently working as a professor at Columbia University; is that correct?
A Yes.
Q And you also are a professor at New York University; is that correct?
A Yes.
Q How do you split your time between these three jobs?
A It depends on the semester and it depends on the consulting work that's ongoing.
Q So on average, is there a percentage between teaching and Aetion that you could specify?
A It really ranges and it -- it really does depend on my class load, it depends on what students I'm mentoring, etc.
Q Does your position at Aetion ever rise above 50 percent of your working time?
A Sometimes, yes.
Q Okay.
(Whereupon, there was a discussion off the record.)
(Whereupon, Boehme's Exhibit 7, Publication from Aetion Website, was

1 marked for identification.)
2 BY MS. MIRSKY:
3 Q So what's been marked as Exhibit 7 is a
4 publication from the Aetion website.
5 Do you recognize this?
6 A Yes.
7 Q Okay. And this is an article that you
8 wrote for Aetion in February 2025; correct?
9 A Yes.
10 Q Would you please look at the second
11 paragraph?
12 And there's an abbreviation, RWE.
13 Can you tell me what that stands for,
14 please?
15 A Real world evidence.
16 Q Okay. The second -- beginning with the
17 second sentence it states, RWE must be rigorous,
18 transparent, and reproducible to be relevant in
19 regulatory decision-making. Not all RWE studies
20 meet these criteria and inconsistencies in data
21 sources and methodologies can undermine their
22 credibility. Ensuring that RWE studies adhere to
23 scientific and regulatory standards is essential for
24 RWE use in premarket approvals, post-market

surveillance, and health technology assessments, parentheses, HTA.

Did I read that correctly?

A Yes.

Q And then on the second page, it -- underneath Aetion's role in generating regulatory grade RWE, the second paragraph, it states that, Aetion works with NEST to implement the NEST mark approach for evaluating the regulatory feasibility of RWE studies helping manufacturers identify data sources that align with regulatory expectations; structure real-world studies to generate credible, interpretable findings; apply rigorous methodologies to ensure transparency, reproducibility, and regulatory alignment.

Did I read that correctly?

A Yes.

Q And what does NEST stand for?

A Hold on, or I'm going to get this wrong.

It is the National Evaluation System for health Technology.

Q Okay. Thank you.

Would you agree that these principles apply to epidemiology broadly?

MR. MICELI: Object to the form.

A Could you restate the question, please?

BY MS. MIRSKY:

Q Would you agree that the three criteria that -- or three indented lines in this article apply to epidemiology broadly?

A So this is a little bit different in that what NEST is trying to do is emulate target trials. And so in that vein, they need their data sources to match what would be done in a clinical trial.

In epidemiologically more broadly, applying rigorous epidemiologic methods is at the forefront. We are always focusing on that. And that is always taken in context with what is being studied.

So what is being done for regulatory facing might be different than exploratory studies where a clinical trial is not an option.

Q Thank you.

So you would agree that rigorous methodologies are important in "epidon-" -- epidemiology; correct?

A Absolutely.

Q And rigorous methodologies are needed in

epidemiology to ensure transparency; correct?

A I mean, transparency is separate from rigorous methodologies.

Transparency is informing your reader everything that you've done within your studies so that the reader can interpret the findings within the context of what has been done and the context of the limitations.

So, you know, there's transparency and there's rigorous methodologies.

Q Understood.

So would you agree that transparency is needed in analyzing -- in -- in -- strike that.

Would you agree that transparency is required when putting forward opinions about epidemiological studies?

A I'm not sure I understand the question.

Q Let me try to rephrase.

Would you agree that transparency is needed to inform readers about how you analyzed epidemiological studies?

A Me, personally, when I'm doing my report, is that what you are referring to, or just in general?

Q In general.

A This is where the peer review process comes in; right? So, you know, when I'm reviewing a journal article or when I'm publishing something, we aim to provide as much information to be transparent, and when I'm reviewing as a peer reviewer, I look for that information so that I can understand what has been done and how the authors came to their conclusions.

Q And more specifically, when working on your report?

A When working on my report, it's a little bit different in that I'm also weighing in on my scientific expertise and drawing conclusions from a broader evaluation of the literature and the evidence as a whole and taking many other factors into consideration.

Q Okay. Thank you.

Would you agree that rigorous methodologies are needed in epidemiology to ensure reproducibility?

A So rigorous methodologies are needed to be understood so that others can build on the studies that have been done and if warranted,

reproduce the studies in different populations.

Q Okay. Thank you.

Going back to your report on page two --

MR. MICELI: Are we done with this for now?

MS. MIRSKY: Yes.

MR. MICELI: Thank you.

BY MS. MIRSKY:

Q The second sentence of the first full paragraph states, Epidemiology as a field incorporates statistics and epidemiologic methods to assess causal factors in observational studies. These methods have been developed in order to apply rigorous research methodology to observational studies of clinical questions, many of which are not appropriate to study using randomized control trial.

Did I read that correctly?

A Yes.

Q You then write two sentences later, This report will utilize epidemiologic and evidence-based medicine approaches to evaluate findings reported in the literature, particularly the Bradford Hill criteria, which are now referred to as Hill's List of Standards for Inference in Epidemiological Text,

and referred to as "Bradford Hill considerations" in this report, as not all of the considerations are needed to determine causality.

Is that correct?

A Yes.

Q Are these consistent with the standards that we just discussed?

A To an extent.

Q Can you explain how they might be inconsistent?

A Well, there's more to it; right?

So Bradford Hill criteria was written in the '60s and so our understanding of scientific methodology has rapidly increased since then.

Epidemiologic methods have seen a rapid increase in the ability of what can be studied and how, in addition to how we can address concerns around bias, confounding, and other factors that could influence observational studies.

So a lot has been built upon this and will continue to be built upon this.

Q You've taught a number of courses on epidemiology; correct?

A Yes.

Q And that's at Columbia University and New York University; correct?
A Yes.
Q Do you teach about levels of association in your courses on epidemiology?
A What do you mean by levels of association?
Q Do you teach about what a finding of association might entail in -- in your courses?
A Like how to calculate a measure of association or --
Q Sure.
A I -- I do teach about how to calculate measures of association. I also teach about how to interpret measures of association within the context of your study design and what you are evaluating.
Q Okay. Do you teach about analyzing risk or hazard or odds ratios in a study's results in your courses on epidemiology?
A Yes.
Q And would you agree that it's important to analyze a risk or hazard or odds ratio in a study's result when analyzing the study overall?
MR. MICELI: Object -- excuse me.

Object to the form.

A Could you repeat the question?

BY MS. MIRSKY:

Q Would you agree that it's important to analyze a risk or hazard or odds ratio in a study's result when analyzing a study overall?

A It depends on the study. It depends on what you are studying and how you are studying it.

Q Okay. A risk ratio indicates the level of association; is that correct?

A The risk ratio provides a magnitude of association.

Q And what does -- what do you mean by magnitude of association?

A So what the risk ratio is indicating is whether or not one group, exposed, has a difference in the risk of disease than the unexposed.

And this ratio can indicate whether or not those who are exposed are protected from disease, so if the risk ratio is less than one, or at risk for the disease.

And so what the risk ratio is really trying to estimate is we understand that there's an inherent risk of disease in a general population,

what we often term as the source population.

And when we are evaluating specific risks, we want to understand how much more does that risk factor, in this case, an exposure, increase the probability of disease.

And so the risk ratio is estimating that excess that would be driven by that specific exposure as opposed to other potential factors that could influence disease.

Q Okay. And you talked about if a risk ratio is above one or below one, it indicates something one way or the other.

If there's a risk ratio of one, that indicates no association; correct?

A Correct.

Q If you go to page ten of your report, please, a few lines up from the -- the bottom of the main paragraph there you are listing a few hazard ratios and there's a hazard ratio of 1.59; do you see that?

A Yes.

Q Okay. I can --

A After the line --

MR. MICELI: You are referring to

four lines up from the --
MS. MIRSKY: Yes.
MR. MICELI: Okay.
MS. MIRSKY: Yes.
BY MS. MIRSKY:
Q Okay. And so you say in -- in respect to this 1.59 hazard ratio, that there is a strong association between Camp Lejeune and risk of PD mortality; do you see that?
A Yes.
MR. MICELI: Well, just --
THE WITNESS: Sorry.
MR. MICELI: -- object to the form. You got to just --
THE WITNESS: I apologize.
MR. MICELI: Okay.
MS. MIRSKY: Okay.
MR. MICELI: -- allow me to object.
A Could you repeat the question, please?
BY MS. MIRSKY:
Q You state that a hazard ratio of 1.59 is a strong association between Camp Lejeune and PD mortality?
MR. MICELI: Same objection.

A I am referencing all of those hazard ratios, not just the 1.59.

BY MS. MIRSKY:

Q Would you agree that a hazard ratio of 1.59 is a strong association?

A I would agree that that is a large measure of association.

Q But would you characterize it as a strong association?

A Yes.

Q Okay. What level of increased risk reflects a slight association in your opinion?

A It depends on whether or not our exposure is dichotomous, categorical or, continuous. That's going to influence what would be considered a strong association.

Q I was asking about a slight association.

A Or a slight association.

Q Okay.

A It -- it -- same.

Q So you don't have a -- you know, a range in mind for what would constitute a slight association?

A It's very dependent on what type of

exposure you are assessing, whether it's continuous, categorical, or dichotomous.

Q Okay. Association is not the same as causation; correct?

A It is not.

So when we think about causation, association is a factor in assessing causation.

Q Okay. And so would you agree that generating an opinion on causation is not as simple as pointing to a study that shows a -- an association between a substance and a disease?

A It's larger than that. So we are looking at the entirety of the literature.

And so when you see that there are a number of studies that have very strong associations within those studies, this starts to point to consistency. And what we want to understand when we are assessing causality is the bulk of the research.

We don't assess or determine causality off of a single paper. We look at what has been done in the context of everything that's been published.

And strong associations within multiple peer-reviewed publications are part of establishing

1 causality.
2 Q You -- we were just talking about what
3 constitutes a strong association and you said that
4 it depends; correct?
5 A It depends on the type of exposure, yes.
6 Q So when you are looking to determine
7 whether there's consistency of strong associations,
8 how do you determine whether a particular
9 association is strong?
10 A So, again, it depends on if our exposure
11 is continuous, categorical, or dichotomous.
12 And so when you're reading the studies,
13 you would understand whether your exposure is a
14 continuous variable, categorical variable, or a
15 dichotomous variable. And it's all within the
16 context of what you're seeing in regards to how the
17 exposures are measured.
18 Q You talked about looking at the entirety
19 of the literature.
20 In your opinion, would it be possible to
21 have a study showing a statistically significant
22 association between exposure to a substance and a
23 disease and still not be able to conclude that the
24 substance in question can cause the disease?

MR. MICELI: Object to the form.

A Could you repeat the question?

BY MS. MIRSKY:

Q Would it be possible to analyze a study showing a statistically significant association between a substance and a disease and still not to be -- still not be able to conclude from that study that the substance in question could cause that disease?

MR. MICELI: Same objection.

A So when we're looking at a single study, we do start to talk about causality in the context of what's been done so far.

And so in a discussion section of a paper like that, you might start to speak towards, this is in line with what has also been published.

However, in terms of causality, we really look at the entirety of the body of literature, not just a single paper. We don't build our case off of a single paper. We need to see more evidence than just a single study.

BY MS. MIRSKY:

Q And so to that point, you wouldn't draw a conclusion about causation from a single study;

correct?
A You could start to talk towards causation. But, again, when we look at the en- -- we'd need to look at the entirety of the evidence.
Q So you couldn't draw that conclusion from just one study; correct?
A Depending on the methods you're using, you could start to say that this is -- that something is on a causal pathway.
Q But it wouldn't be determinative?
A What do you mean by that?
Q It -- one study in and of itself would not be sufficient to determine causation?
A When we are talking on a global public health scale, we are not going to make causal determinations off of a single study.
Q Okay. And you would, as you were saying, want to assess all of the available epidemiological literature in evaluating causation; correct?
A And it's not just the epidemiologic literature, it's everything as a whole. We need to understand the mechanisms of what's occurring, we need to have an understanding of, you know, the animal models, whether there are changes over time,

etc.

Q Okay. Typically the authors of an epidemiology study provide statistical results that could indicate the levels of any risk observed; is that correct?

MR. MICELI: Object to form.

A What do you mean by that?

BY MS. MIRSKY:

Q That a study will include statistical results to support any conclusions related to increased or decreased risk?

MR. MICELI: Same objection.

A Are you talking about statistical significance?

BY MS. MIRSKY:

Q Any statistical numbers or evaluations.

MR. MICELI: Same objection.

A Well, there's, you know, calculating your measures of effect and then there's calculating whether something is statistically significant or not.

BY MS. MIRSKY:

Q Okay. So what about statistical significance?

A So statistical significance is a little funny in that it is very dependent on your sample size.

And especially when you are looking at something like Parkinson's-- which is, you know, in the grand scheme of things, relatively rare -- it becomes harder to reach statistical significance because of small numbers and small populations of people with Parkinson's.

And so in situations where we know that we are not powered for statistical significance, the -- then you start looking at the magnitude of association, you look at how the study was designed, and whether the study did what they could to reduce bias, and recognize that in a fully powered study, you would likely see statistical significance.

Q Okay. For epidemiological findings, what is the difference between showing an increased risk of developing a disease and showing an association?

A What do you mean by that?

Q We talked about how epidemiological studies can discuss whether or not there's an association between an exposure and a disease; correct?

A Yes.
Q Do epidemiological studies also analyze whether or not there's an increased risk of developing a particular disease?
A Calculating risk is one way of calculating an expen- -- an association. There are a number of different ways to calculate associations.
Q So calculating what the risk of developing a disease is part of the analysis of determining whether there's an association?
A It depends on the study design. So certain study designs are able to calculate risk, certain study designs are not.
So for instance, the cross-sectional study, we never calculate risk in a cross-sectional study.
Q Okay. Back to your report on page two, at the bottom of the main paragraph, the last two sentences read, The measure of association, such as odds ratios or risk ratios, and 95 percent confidence intervals (95 percent CIs) will be presented for key findings in this report. The measure of association provides information on the

strength of association and the 95 percent CI provides information on the level of precision around the measure of association.

Did I read that correctly?

A Yes.

Q Confidence intervals provide a measure of precision; is that correct?

A Yes.

Q And a narrower confidence interval indicates more precision; is that correct?

A Generally, yes.

Q A wider confidence interval indicates less precision; is that correct?

A Yes.

Q In interpreting the confidence interval, one should consider if the underlying test or sample population is biased; is that correct?

MR. MICELI: Object to the form.

A Could you repeat the question?

BY MS. MIRSKY:

Q In interpreting a confidence interval, one should consider if the underlying test or sample population is biased?

MR. MICELI: Same objection.

A So one of the things that we do with our measures of association and our confidence intervals is we interpret these results within the context of our limitations. And limitations could include bias.

And so when we are thinking about bias in studies, we do what we can to minimize bias during our study design.

There are methods that can be done to estimate what the measure of association would have been if bias was attenuated, if there was any concern for bias, as well as if there's any concern for selection bias.

So there are methods where we can approximate what the measure of association and subsequent confidence intervals would be if there had been bias present, for instance.

BY MS. MIRSKY:

Q So then consideration of any potential bias is necessary when interpreting a confidence interval; correct?

MR. MICELI: Object to the form.

A Again, it's interpreting your results within the context of your limitations, and

limitations include bias.
BY MS. MIRSKY:
Q Okay.
A Or potential bias, I should say.
Q Okay. Would you agree that a confidence interval is less meaningful where the sample of the population is not representative of the underlying population?
A What do you mean by that?
Q When -- when analyzing a confidence -- excuse me, a confidence interval, you mentioned that understanding potential limitations is important; correct?
A Uh-huh.
Q And a lack of representativeness in the sample population compared to the larger underlying population could be a limitation; correct?
A Not necessarily. There are many instances where we want a less representative population, and this could be because we are trying to evaluate specific effects, this could be because it may not apply to a larger population as a whole.
But generally, when we are looking at a population that is not generalizable, we would make

those interpretations within the context of that population.

So say, for instance, we are only studying school children and we are looking at the risk of paying attention if you consume sodas in schools.

Those -- the results from that would only be interpreted within the context of the school children and could be applied to other school children.

Would it be applied to the greater U.S. as a whole? Possibly.

There are many circumstances where even if our study population is not representative of a larger population, we can and do extrapolate sometimes.

Q We talked about statistical significance a bit.

Do you agree that statistical significance measures the probability of the null hypothesis being true compared to the acceptable level of uncertainty regarding the true answer?

MR. MICELI: Object to the form.

A Could you repeat the question, please?

BY MS. MIRSKY:

Q Would you agree that statistical significance measures the probability of the null hypothesis being true compared to the acceptable level of uncertainty regarding the true answer?

MR. MICELI: Same.

A So essentially what you are looking at is whether or not what you are seeing, or more extreme than what you are seeing, is due to chance, and so you are trying to eliminate that chance.

But statistical significance is highly dependent on sample size.

I have had situations where I did calculations in a very large population. My risk ratio was one and it was statistically significant. That is a null value.

And that's why we pull in the magnitude of association, especially when we are looking at outcomes where they are more rare, where we do see lower numbers, because we know we don't have statistical significance.

And so we look at this in the context of we are looking at a rare disorder, we know we are not going to be powered for statistical

significance, so we start looking at the magnitude of effect.

And as long as the magnitude of effect is large, then we do know that if we had been sufficiently powered, we would still see an increased risk.

BY MS. MIRSKY:

Q So then is it your opinion that statistical significance is irrelevant in evaluating the results of a study?

MR. MICELI: Object to the form.

A I would say that it is not irrelevant. It is one of many components and it is not the end-all, be-all.

There's been a movement away from statistical significance being the end-all, be-all because there is the overarching recogni- -- recognition that it is so dependent on sample size and often we are not able to obtain the necessary sample size. And so it's looking at a larger context.

BY MS. MIRSKY:

Q Okay. You -- you said that there's a movement away from emphasizing statistical

1 significance.
2 But is it generally accepted in the field
3 of epidemiology to disregard statistical
4 significance?
5 A I'm not saying to disregard. I'm saying
6 that it needs to be considered as a whole.
7 So something can be statistical
8 significant but clinically or from a public health
9 perspective meaningless.
10 Something may have a very large magnitude
11 of effect which has a large clinical relevance or
12 public health relevance but is not statistical
13 significant.
14 And so we recognize that since
15 statistical significance is due to sample size, it
16 is one component. It is not the end-all, be-all.
17 Q Okay. At the bottom of page two of your
18 report going onto page three, you write, Statistical
19 significance will not be used in determining
20 evidence for causality as the limitations to
21 significance testing outweigh the benefits.
22 Statistical significance is determined by sample
23 size (including sample size within exposure or
24 outcome groups) and strength of the measure of

association; correct?
A Correct.
Q Would you use a finding that has a relative risk of 1.1 that was not statistically significant in determining causation?
MR. MICELI: Object to the form.
A So, again, this would -- that finding would be very dependent on whether the exposure was continuous, categorical, or dichotomous.
BY MS. MIRSKY:
Q Okay. Speaking more broadly about your report, does this report contain all the opinions you've formed to date in this case?
A It contains an overview of my opinions. As more information might come in, my opinions may elaborate.
Q Are you planning to form any additional opinions?
A I do not know.
Q What was your assignment in this litigation?
MR. MICELI: Do not repeat anything that we discussed.

BY MS. MIRSKY:
Q Yeah. Do not testify to any privileged information, but if you can --
A Essentially to compile the epidemiologic literature on the relationship between TCE, PCE, and Parkinson's disease, and to give an overview and scientific opinion on the epidemiologic evidence for the relationship between TCE and/or PCE and Parkinson's.
Q Okay. On page one of your report, right at the top you state, The purpose of this report is to analyze the role, if any, of TCE and PCE as a cause of PD. I provide a summary of the literature on PD epidemiology, environmental risk factors for PD, TCE, and PCE as causes of PD, and mechanisms behind the relationships; correct?
A Yes.
Q Okay. If you could turn to page 22 of your report, please.
(Whereupon, there was a discussion off the record.)
BY MS. MIRSKY:
Q Okay. You have three opinions bulleted at the -- in the middle of the page; is that

correct?

A Yes.

Q And the three opinions are: It is at least as likely as not that exposure to the water at Camp Lejeune contaminated with TCE can cause Parkinson's disease; that it as least as likely as not that exposure to the water at Camp Lejeune contaminated with PCE can cause Parkinson's disease; it is at least as likely as not that exposure to the water at Camp Lejeune contaminated with TCE and PCE and other volatile organic compounds can cause Parkinson's disease.

Did I read that correctly?

A Yes.

Q Is it fair to say that these are your overall opinions included in this report?

A Yes.

Q If called at trial, would you offer any other expert opinions?

A It depends on what I'm being asked to offer.

Q Okay. Would you agree that your report is a complete statement of all opinions you will express and the basis and reasons for them at this

point?

MR. MICELI: Object to the form.

A It depends on if I'm being asked for more opinions on epidemiologic methods, anything epidemiologic based.

BY MS. MIRSKY:

Q Okay. In each of these opinions, you use the phrase "at least as likely as not"; correct?

A Yes.

Q Where does this language originate from?

A What do you mean?

Q How did you arrive at this standard or this language?

A Could you elaborate your question?

Q What does "at least as likely as not" mean to you?

A So to me, it means that when evaluating the evidence at large, that more often -- that -- that the combined evidence indicates that TCE increases the incidence of PD, PCE increases the incidence of PE -- PD, sorry -- and that it's -- essentially -- that are measures of association indicate risk factors.

Q And have you ever used this standard in

any of your publications?
A I don't know. I have many publications and I would have to go back and look through what my language is in my publications.
Q Do you recall at this point ever using it previously?
A I have no idea.
Q Okay. Do you recall ever seeing this standard used in other scientific articles?
A I don't know. I read a lot of articles daily. There's a lot of different language uses -- uses used.
Q You've served as an expert previously; correct?
A Yes.
Q Have you ever written an expert report stating your opinions using the "at least as likely as not" language?
A I would have to go back to my reports and see what language I used.
Q But as you sitting here today, do you recall any instances?
A I have no idea. I don't remember my reports off the top of my head.

MS. MIRSKY: Okay. Could you get Tab 7, please?

MR. MICELI: Is this going to be Number 8?

MS. MIRSKY: Yes.

MR. MICELI: Okay.

MS. MIRSKY: This has a bit of a bent staple, so just be careful.

(Whereupon, there was a discussion off the record.)

(Whereupon, Boehme's Exhibit 8, May 18, 2022 affidavit, was marked for identification.)

BY MS. MIRSKY:

Q Do you recognize this document?

A Yes.

Q This is your May 18th, 2022 affidavit in the case Burkes vs. Munster (phonetic), et al.; is that correct?

A Correct.

Q Do you recall what this case was about?

A I was asked to serve as an epidemiologic witness in this case. I do not off the top of my head remember exactly what it was. This was a while

ago.

Q Do you remember the general subject of your opinions?

A I do not remember.

Q Okay. On page two in paragraph two, the third sentence says, I'm aware that the legal cause of an injury is a cause, which in a natural continuous sequence produces injury or harm without which the injury of harm would not have occurred; correct?

MR. MICELI: Where are you reading from?

MS. MIRSKY: It's the third sentence in the second paragraph.

MR. MICELI: Okay. Okay.

A Yes.

BY MS. MIRSKY:

Q Do you know where -- do you know how you became aware of that definition of a legal cause of an injury?

A I don't remember. This was three years ago.

Q Okay.

A Actually, I think, a little more.

Q Have you ever used this definition or standard in other scientific contexts?

MR. MICELI: Object to the form.

A I honestly have no idea.

BY MS. MIRSKY:

Q Okay. You then state that you were, quote, aware that the legal cause of an injury or harm is a cause which is a substantial factor in bringing about the injury or harm; correct?

MR. MICELI: Object to the form.

A That's what is in -- yes.

BY MS. MIRSKY:

Q And do you recall where this language originated from?

A I don't remember what I wrote in here. I honestly don't. I'm sorry. I write a lot of publications and it's been a long time since I wrote this.

Q Okay. Do you know whether you have ever used this language in another publication?

MR. MICELI: Object to the form.

A I don't know. I have a lot of -- again, I have a lot of publications and we use a lot of different language. I don't remember.

BY MS. MIRSKY:

Q Okay. Going back to page 22 of your report -- you can put that to the side -- your -- the third bullet includes the phrase "volatile organic compounds"; correct?

A Yes.

Q You do not include a definition for this term; correct?

A Correct.

Q Is volatile organic compounds, or VOCs, intended to include any compounds other than TCE or PCE?

A What do you mean?

Q Well, how would you define "VOCs" in this context?

A So I was using the definitions from publications. And so in the review of things like solvents, it discussed VOCs. And with solvents containing TCE and PCE, it's all wrapped within that.

I personally do not come up with a definition of "VOC."

Q So "VOC" as it's used in your report, does it refer to compounds other than TCE or PCE in

solvents?

A Depending on what paper I'm referencing, possibly.

Q Okay. Do you have any specific opinions on any other VOCs?

A Not at this moment.

Q Okay. Are there any other specific VOCs discussed in your report?

A So my report, again, goes over some of the publications, the epidemiologic studies that look at the broader VOCs. And, again, those are going to be specific to each paper that's being reviewed and what their definitions are and what they've included.

Q Okay. You do not offer an opinion on benzene and Parkinson's; correct?

A Correct.

Q And you do not offer an opinion on vinyl chloride and Parkinson's; correct?

A Correct.

Q Are you aware that other experts for the plaintiffs considered vinyl chloride and benzene and their relationship to Parkinson's?

MR. MICELI: Object to the form.

A I am not.
BY MS. MIRSKY:
Q Okay. Were you asked to evaluate these chemicals and their relationship to Parkinson's?
MR. MICELI: Object to the form.
And do not discuss anything that we discussed.
A I was asked to focus on TCE and PCE.
BY MS. MIRSKY:
Q Okay. Can you please describe the methodology that you used in forming your opinions in this report?
A Absolutely. So I start with a very broad approach to reviewing the literature and, you know, using search terms that are very broad, open-ended. And I go through publications that are found through those search terms. And I was really focusing on epidemiologic studies. So a lot of the search terms did produce results for reviews, opinions, etc.
But I was focusing on epidemiologic studies. And within epidemiologic studies, I did also include case studies and case series as that helped to set the stage for why the epidemiologic studies were done.

And so I was -- again, I start with a very broad approach, narrow it down. I'm focusing on publications that had done what they could to decrease the impact of limitations. I was trying to focus on studies that really used more recent information that had good measures for exposures and good measures for outcomes and were relevant to what I was evaluating.

Q Okay. Could you please turn to the red tab in your report?

A Uh-huh.

Q And this is your reference list; correct?

A Correct.

Q Is this list complete for the references cited in your report?

A For the references cited, yes.

Q Okay. And if you could please go to page 30, this is your additional materials reviewed list; correct?

A Yes.

Q And is this list complete for the additional materials you reviewed aside from the documents in your reference list?

A For the report, yes.

Q Okay. If I refer to these combined lists as your materials considered list for ease of reference, will that be clear to you?

A Yes.

Q If there's a distinction between what I'm talking about, I'll make it clear, but --

A Yes.

Q Okay. Thank you.

MR. MICELI: Just -- just for the record, in doing that there are going to be two, 1s through 27.

MS. MIRSKY: Yes.

MR. MICELI: Just...

MS. MIRSKY: Thank you.

MR. MICELI: Yup.

BY MS. MIRSKY:

Q So you previously, sort of, broadly discussed how you took an approach to gather the literature that you reviewed; correct?

A Yes.

Q Would you say that you conducted a literature review?

A I would say I conducted a literature review, but I did not do a systematic literature

review.

Q What is a systematic literature review?

A So a systematic literature review is where you set out with very precise inclusion/exclusion criteria for the studies you want to include.

And I wanted my literature review to be more broad because, again, I did want to include case series, case studies. I wanted to have the opportunity to read through publications that may have had significant limitations but evaluate whether or not the authors did what they could for the limitations and ensure that I captured as much information as possible.

Q And you mentioned running some searches.

Did you conduct a PubMed search?

A I used PubMed, Medline, and Google Scholar.

Q And what were the search terms that you put into PubMed?

A Some examples -- oh.

MR. MICELI: Object.

Go ahead. I'm sorry. I won't -- I won't object to that.

A I have them written down, but some examples included environmental health and Parkinson's, TCE and Parkinson's, PCE and Parkinson's, with a lot of other more broad search terms as well.

BY MS. MIRSKY:

Q So you mentioned that you have the search terms written down.

Do you still have that list?

A I could probably find it, yes.

Q Okay.

MS. MIRSKY: So we would request that list, please.

THE WITNESS: Okay.

BY MS. MIRSKY:

Q And you said you did a search on Medline as well; is that correct?

A Correct.

Q And do you know what the search terms are that you --

A I used the same search terms for all three.

Q Okay. So that would be for PubMed, Medline and Google Scholar; is that what you said?

A Yes.
Q Okay.
A May I pause for a second?
I think an alarm is going off. I apologize.
Q Sure. We can take a break -- we've been going for about an hour -- if you like.
A No, I just somehow set an alarm. I apologize.
Q No, that's okay.
MR. MICELI: Do you want -- are you at a breaking point? Or do you want to just keep going? It's fine --
MS. MIRSKY: I'm fine to keep going, but if you want to take a break --
THE WITNESS: I'm fine to keep going. I just didn't want --
MR. MICELI: Can we just go off the record just for one second? Because it's eleven o'clock. I want to ask y'all something.
THE VIDEOGRAPHER: The time right now is 11:08 a.m. and we are off the record.

(Whereupon, there was a recess taken from 11:08 a.m. to 11:09 a.m.)

THE VIDEOGRAPHER: The time right now is 11:09 a.m. and we are back on the record.

BY MS. MIRSKY:

Q So we were just talking about the searches that you ran on PubMed, Medline, and Google Scholar and you said that those are probably still in your possession; correct?

A I have to go find them, but yes.

Q But they are not incorporated into your report; correct?

A Correct.

MR. MICELI: For the record, we appreciate she's going to look to make sure she still has them; right?

THE WITNESS: Yes.

MR. MICELI: Okay. I've got it written down.

MS. MIRSKY: Thank you.

BY MS. MIRSKY:

Q Are there any other searches that you conducted aside from those three?

A No.
Q Okay. Did you review all of the results from these searches?
A I did personally, yes.
Q And did you exclude any studies from your review?
A Oh, yes.
Q And why would you exclude a study from your review?
A If it was not relevant, if it was evaluating something else. So for instance, using the "environmental health and Parkinson's" search term, there were many things that were not relevant to this and so did not include those.
I didn't include opinion pieces or speculation or argumentative pieces.
Q Okay. Are there any other reasons that you would exclude a study from your review?
A So if I found that the study -- or if I thought that the study was poorly done, I would not include it as well.
Q And how do you determine whether a study is poorly done?
A So it's really through my scientific

1 expertise. So that's, you know, weighing what's
2 been done within the context of the study design,
3 within the context of how they are capturing the
4 exposure definitions and the outcome definitions,
5 what statistical methods they are applying, and the
6 interpretation of the results.
7 Q Okay. And these reasons that you just
8 discussed for excluding studies from your review are
9 not incorporated in your report; correct?
10 A I did not explicitly list out how I did
11 that, no.
12 Q Okay. Did you consider any studies that
13 were inconsistent with your opinions?
14 A Oh, yes.
15 Q And are they in your materials considered
16 list?
17 A I'm -- I would have to look.
18 Q Okay. You mentioned that you included
19 case studies in addition to epidemiology studies.
20 Can you explain what the distinction is
21 between the two?
22 A Absolutely. So a case study is often --
23 it's one of the first publications we'll see in
24 terms of a new exposure-disease relationship. So

it's a unique finding that hadn't been explored before.

So case studies will range from a case report, where it's just a report of one person, to a case study where it's a handful of people who may have a similar outcome after a similar exposure or experience.

Case reports and case studies are often then used to justify conducting larger scale studies to explore those relationships.

Q So would you agree that findings from case studies are not as persuasive as findings from epidemiological studies?

MR. MICELI: Object to the form.

A Again, it's about the entire context; right?

So we're not -- there are circumstances, actually, I should say, where we may form a basis off of a single case study or a case report. It's going to be very dependent on the exposure of interest and the outcome of interest. There are many things in epidemiologic literature that may not be conducive to large-scale studies, especially rare diseases.

BY MS. MIRSKY:

Q Would you say that the findings of a case study would be sufficient to determine association between an exposure and a disease?

A It really depends on what is being evaluated and whether or not you can go to larger populations or not. And, again, that is very dependent on how rare the exposure and/or outcome is.

Q Okay. How did you decide whether to include certain documents in your materials considered list based on your overall review?

A So I was looking for publications that had large sample sizes. I was trying to focus on U.S. based. There are some publications that are non-U.S. based, but really wanted to focus on how the exposure is being determined in particular since, you know, establishing exposure in this case is not a straightforward as other epidemiologic questions.

Q Okay. And so if a study did not have a large sample size, did you include it in your materials considered list?

MR. MICELI: Object to the form.

A I included some. There were some smaller studies and, you know, when you are working with a population of, like, 70 people, it's -- you are running into sample size constraints.

BY MS. MIRSKY:

Q Do you have a definition for what a large sample size would include?

A So a sample size is, again, going to be dependent on what you are studying. So what's large for one aspect may not be large for another.

Q Do you have a definition for what a large sample size would be in the context of this report?

A I was really trying to focus on, you know, studies that were cohorts so we have their temporality met, and where we would have at least a hundred people in the exposed group since the exposure is rare.

Now, that does not negate the smaller studies, but it is -- it is a lot harder if you only have a handful of people in your exposure group. It can be done and there's important information to be gleaned. But overall, in terms of the epidemiologic findings for these explorations, they were very consistent.

Q Okay. You also mentioned that how exposure is determined in a study was something that you considered when putting together your materials considered list; correct?

A Yes.

Q And what are the ways in which a study determined exposure that would encourage you to include a study in your materials considered list?

A So if there was really robust assessments for occupation. So occupation as a proxy is -- you know, it's a proxy. So there are limitations with using a proxy.

However, the more granular and detailed any occupation assessment is, the better the proxy of exposure would be.

Additionally, you know, using the Camp Lejeune, Camp Pendleton, understanding the exposures at Camp Lejeune versus Camp Pendleton, were more robust than an occupational exposure. You know, understanding where they are drawing these assumptions from.

Q Okay. Did counsel provide you with any documents in connection with this matter?

A They had sent me the ATSDR 2017.

Q Anything else?
A That's it.
Q Okay. Were there any documents that you reviewed but decided to not include in your materials considered list that impacted your opinions?
A No.
Q Did you review any other expert reports from this litigation while preparing your report?
A I reviewed Dr. Cannon's report.
Q Any of the water modeling reports?
A No.
Q Other than Dr. Cannon's report, did you review any of the other general causation reports?
A No.
Q Why did you review Dr. Cannon's report?
A As he's a toxicology expert, to have a understanding of the mechanism and ensure that, you know, my brief blurb on mechanism was appropriate.
Q Did you ask to review Dr. Cannon's report?
MR. MICELI: Object to the form.
Do not discuss what we discussed, so I would instruct you not to answer --

THE WITNESS: Okay.
MR. MICELI: -- because that's -- you are asking about our discussions.
THE WITNESS: Okay.
BY MS. MIRSKY:
Q Did you ask to review anything specific on toxicology?
MR. MICELI: Object to the form.
And don't answer -- I'm instructing you not to answer about conversations you had with me.
If you are referring to other conversations, I -- I can't object to that so --
A Could you repeat the question?
BY MS. MIRSKY:
Q That's okay.
Have you reviewed the transcripts of any of the other general causation experts in this case?
A I've only reviewed the transcript of Dr. Cannon and Dr. Miller.
Q Okay. Did you review the transcripts of any other depositions in this case?
A No.

Q On page 30 of your report, the numbers one and two of additional materials reviewed --

A Oh.

Q -- lists the two deposition transcripts from Dr. Frank Bove; correct?

A Yes. And I did review those in terms of establishing that the ATSDR report was peer reviewed.

Q Which ATSDR report are you referring to?

A The one Dr. Bove wrote.

Q Dr. Bove authored multiple reports.

Are you referring to one in particular?

A I would have to look at what I referenced.

Q Okay. That's okay.

(Whereupon, there was a discussion off the record.)

(Whereupon, Boehme's Exhibit 9, Dr. Amelia Boehme Supplemental Materials Considered, was marked for identification.)

BY MS. MIRSKY:

Q Okay. What's been marked as Exhibit 9 says at the top, Dr. Amelia Boehme supplemental

materials considered.

Do you recognize this document?

A Yes.

Q Have you seen it before?

A This specific document?

Q Yes.

A I believe so.

Q Did you prepare this?

A I did not prepare this.

Q Were you aware this was being prepared --

A Yes.

Q -- on your behalf?

Let me just finish the question --

A Oh.

Q -- before you answer.

A Apologies.

Q That's okay.

Were you aware that this was being prepared on your behalf?

A Yes.

Q Are you aware that Plaintiff's counsel recently submitted this list on behalf of five of the Parkinson's general causation experts?

A Could you --

MR. MICELI: Objection to the form but --

A Could you repeat the question?

BY MS. MIRSKY:

Q Are you aware that Plaintiff's counsel has submitted this list on behalf of other general causation experts?

MR. MICELI: Same.

THE WITNESS: Does that mean I don't answer or --

MR. MICELI: No, no. You --

THE WITNESS: Okay.

MR. MICELI: I'm just noting my objection for the record.

THE WITNESS: Okay.

A I know that this was something that we've discussed.

BY MS. MIRSKY:

Q Do you know that it was submitted for other general causation experts as well?

MR. MICELI: Same objection.

A I don't remember our conversation around that, honestly.

BY MS. MIRSKY:

Q Okay. Did you review either of the items listed on this supplemental materials considered list in forming the opinions in your report?

A I did.

Q And why were they not included in your materials considered list?

A So the proxy use of occupation with the way that they were doing the linkage had me very concerned about underestimating TCE exposure.

Q Then why are you including this on your list now?

A Because I did review it and I've since rereviewed it.

It does not contradict any of my opinions and it was something I had considered but I did not include in my report.

Q Is there anything else that you didn't include in your lists that need to be added at this point?

A Not that I'm aware of.

Q Okay. On page three of your report, in the full paragraph right after footnote three, the report states, A number of factors are associated

with the risk of PD. However, PD is largely thought of as a disorder brought about due to environmental contaminants.

Did I read that correctly?

A Yes.

Q Is that your opinion?

A It is not only my opinion, but it is what is considered broadly in the PD world.

Q You did not cite any sources in support of this statement; correct?

A It is in textbooks, it's what I teach in my neuroepidemiology course. It's considered accepted. And I could provide references if you would like.

Q Okay. You don't define what "environmental contaminants" means in this sentence; correct?

A Correct.

Q And you also don't define a required level of exposure to qualify as a risk factor; correct?

A What do you mean?

Q Well, in this statement, you say that PD is largely thought of a disorder brought about due

to environmental contaminants.
But this doesn't include any required level of exposure to environmental contaminants to bring about PD; correct?
MR. MICELI: Object to the form.
A This is an overview statement to set the stage and exposure is going to be dependent on what contaminant.
BY MS. MIRSKY:
Q Okay. A few sentences later you state, quote, The most common PD symptoms result from death or damage to neurons in the substantia nigra, an area near the base of the brain, resulting from the loss of 60 to 80 percent of dopamine -- dopamine-producing cells in this area; correct?
A Yes.
Q Is that your opinion to a reasonable degree of scientific certainty?
MR. MICELI: Object to the form.
A That is not my opinion; that is an overview of PD.
BY MS. MIRSKY:
Q You agree with that sentence; correct?
MR. MICELI: Object to the form.

A I would agree that this is what is discussed in terms of, you know, our understanding of PD on a larger scale, yes.

BY MS. MIRSKY:

Q Okay. And your citation to this is the Bastiaan Bloem, et al. publication, Parkinson's disease in The Lancet; correct?

If you want to flip to your references cited, that's fine.

MR. MICELI: You are referring to number five, reference number five?

MS. MIRSKY: Yes.

MR. MICELI: Okay.

A Oh, sorry.

BY MS. MIRSKY:

Q It should be in the --

A It's all --

Q -- tabbed --

A Oh, okay. Tabbed. Thank you.

Q Yeah.

A Yes.

Q Okay.

(Whereupon, there was a discussion off the record.)

(Whereupon, Boehme's Exhibit 10, Bastiaan Bloem Article from The Lancet, was marked for identification.)

BY MS. MIRSKY:

Q This is the Bloem publication included as citation 5; correct?

A Correct.

MR. MICELI: Did you mark this as ten?

MS. MIRSKY: I -- I believe so, yes.

MR. MICELI: Okay.

MS. MIRSKY: Can you please turn to page 2288?

A Yes.

BY MS. MIRSKY:

Q In the first paragraph on the left, the sentence beginning with "Environmental factors" states, Environmental factors that perhaps had an effect decades ago or that accumulated over time, possibly in interaction with genetic features, is not possible to assess; correct?

MR. MICELI: Where -- I'm sorry, I apologize. Where --

MS. MIRSKY: It's in the sentence starting with "environmental factors," in the first --

MR. MICELI: Thank you.

MS. MIRSKY: -- paragraph.

MR. MICELI: I apologize.

MS. MIRSKY: That's okay.

BY MS. MIRSKY:

Q Is that correct?

A Yes.

Q Okay. And it then says, We assume there is a continuum between causes and risk factors and counteracting factors of different effect sizes that occur in unique constellations. Notably, factors increasing the risk for developing Parkinson's disease are not necessarily identical to those modifying the disease course which can only be assessed in large-scale longitudinal studies; correct?

A Correct.

Q Would you agree that these sentences taken together indicate that evaluating whether environmental exposure could be a risk factor for Parkinson's disease may not be possible?

MR. MICELI: Object to the form.
A Could you repeat your question?
BY MS. MIRSKY:
Q Would you agree that these sentences taken together indicate that evaluating whether environmental exposure could be a risk factor for Parkinson's -- Parkinson's disease may not be possible?
MR. MICELI: Object. Foundation.
A Not necessarily, no.
BY MS. MIRSKY:
Q Would you agree that an individual's actual exposure to a contaminant is critical to identify whether environmental exposure is a risk factor?
A I'm looking at populations and not individuals and so are epidemiologic studies. And when we are looking at the epidemiologic studies, we are looking at, you know, this excess risk, so to speak, with specific exposures.
And when we are looking at that excess risk, we are really drilling down on that one exposure to see, okay, in those that have had this exposure, is there a higher incidence of Parkinson's

than what we would have expected in our general population.

And so that's what really allows us to start exploring these environmental health contaminants or any exposures really. And in doing so, you know, and especially in doing so in repeated studies in different populations over different timeframes, we are really able to assess holding all the other potential causes of Parkinson's, how much more risk do you see in those who are exposed to this particular exposure of interest.

Q Would you agree then that Parkinson's is a multifactorial disease?

MR. MICELI: Object to the form. Foundation.

A Could you repeat the question, please?

BY MS. MIRSKY:

Q Would you agree that Parkinson's disease is a multifactorial disease?

MR. MICELI: Same.

A There's a lot we don't know about Parkinson's. And it could be that in certain populations it's multifactorial and it could be that in other populations it is not.

We still have a lot to learn about the extent of all the different causes of Parkinson's and how that reacts in different populations.

BY MS. MIRSKY:

Q Okay. On the next page of the Bloem publication under the heading, "Nongenetic factors associated with an altered Parkinson's disease risk," it states, quote, Several toxins can produce a clinical picture resembling Parkinson's disease, such as Parkinsonism, resulting from exposures to the neurotoxin MPTP. In addition to these direct causes, various environmental and lifestyle factors have been evaluated as contributors to the risk of Parkinson's disease. This literature is difficult to interpret. Many factors have never been replicated. Conflicting results exist. A plausible mechanistic explanation is often scarce and the observed associations could have been false; correct?

A Correct.

Q It continues, One persuasive risk factor is exposure to environmental toxins, such as pesticides, for which there is converging and consistent evidence. The introduction of pesticides

after World War II, which was required to feed a fast growing world population, could partially explain the current rise of Parkinson's disease. Another well established health factor -- excuse me, another well established risk factor is head injury; correct?

A Correct.

Q In the sentence on page four of your report for which you --

A What page?

Q On page four of your report?

A Page four.

Q -- where you say, Risk factors for PD include increasing age, male sex, familiar history, and environmental exposures, you do not list pesticides as a risk factor; correct?

MR. MICELI: Object to the form.

A That's within environmental exposures.

BY MS. MIRSKY:

Q Okay. And you do not list head injuries as a risk factor; correct?

A I don't list it here, but I do talk about it later.

Q Okay. You also cite to "Parkinson's

Disease and its Management" by George DeMaagd -- DeMaagd and others; correct?

That would be citation six, I believe. Yes.

A Yes.

MR. MICELI: Are we done with --

MS. MIRSKY: Yes.

MR. MICELI: -- with Boom -- or Bloem?

No, Boehme -- Boehme is here. Bloem.

THE WITNESS: Thank you.

(Whereupon, Boehme's Exhibit 11, Publication Titled "Parkinson's Disease and its Management," was marked for identification.)

BY THE WITNESS:

Q And this is that study; correct? Or that publication?

A Correct.

Q On the first page there's a table, table one, that lists risk factors; correct?

A Correct.

Q And some of the factors we've already

discussed, such as head trauma and pesticides, are included here; correct?

A Correct.

Q There are others that we have not discussed, such as elevated cholesterol and increased body mass index; correct?

A Correct.

Q And these are not included in your list of risk factors on the top of page four of your report; correct?

A What I said was they include. I did not say that those were the entirety of the identified risk factors.

Q On the next page, on page 505, the second sentence of the second paragraph under "Pathophysiology" starts with "Typically."

A Oh, okay.

MR. MICELI: Under "pathophysiology," second sentence, did you --

MS. MIRSKY: Second paragraph.

MR. MICELI: Progressive degenerative.

THE WITNESS: So it's --

MR. MICELI: Oh, I see it. Thank you. Yup.

BY THE WITNESS:

Q It says, Typically patients experience the motor features of PD only after 50 percent to 80 percent of dopaminergic neurons have been lost; correct?

A Correct.

Q And that's fairly consistent with the statement -- the earlier statement in your report that we discussed where you said that 60 to 80 percent of dopamine-producing cell loss is necessary before motor manifestations of PD can be seen; correct?

A Correct.

Q Okay. Further down on page four of your report -- and you can put this to the side for now.

A Okay.

COURT REPORTER: Can we go off the record for a moment?

MS. MIRSKY: Sure.

THE VIDEOGRAPHER: The time right now is 11:35 a.m. And we are off the record.

(Whereupon, there was a recess taken from 11:35 a.m. to 11:57 a.m.)

THE VIDEOGRAPHER: The time right now is 11:57 a.m. We are back on the record.

BY MS. MIRSKY:

Q Dr. Boehme, in the -- on page four of your report, in the main paragraph a few sentences in, it says, quote, Age itself is not thought of as a risk factor for PD. However, increased age allows for more exposure time to environmental exposures, intoxicants, and enough time for PD to manifest.

Did I read that correctly?

A Yes.

Q And is that your opinion?

A That is more of a discussion in the PD literature in general. And considering the long latency period for PD, you know, it's understood that it takes a while for this to develop and so the longer someone could be exposed to something that is associated with PD, the more time that goes on, allows for the ability for Parkinson's to develop.

Q And so you agree with that statement; correct?

1 MR. MICELI: Object to the form.
2 A It's what's in the literature.
3 BY MS. MIRSKY:
4 Q But you agree with the statement as
5 written in your report?
6 MR. MICELI: Same objection.
7 A Well, in terms of thinking of exposures
8 and diseases, particularly diseases that have long
9 latency periods, you know, from an epidemiologic
10 perspective you have to have enough time to develop
11 the disease.
12 So if you die from something else at an
13 early age, you're not -- you don't have the
14 opportunity to develop disease, something we call a
15 competing risk.
16 And so we do recognize that for diseases
17 that -- and Parkinson's in particular, with the
18 average onset being over the age of 65 -- you need
19 to at least survive long enough to develop it.
20 BY MS. MIRSKY:
21 Q So -- but my question is just whether you
22 agree with the statement as written in your report?
23 A I agree with the statement.
24 Q Okay. And do you agree that increased

1 age would allow for more time for an individual to
2 be exposed to more pesticides?
3 A Increased time could allow for the
4 opportunity from any exposure to increase the risk
5 of Parkinson's. And so whether this is more time
6 where someone is exposed to solvents or any other
7 risk factor.
8 And, again, the long latency period is
9 the bigger factor here. It takes a long time from
10 an exposure to symptoms. And so you have the
11 underlying changes that are occurring that don't
12 manifest until symptoms occur.
13 And because Parkinson's in particular
14 doesn't have a test, it's solely based on clinical
15 symptoms, there's really no way to identify
16 pre-Parkinson's like there could be for other
17 disorders. And so because of that, age really plays
18 a factor.
19 Q And so my question was whether increased
20 age would allow for more time for an individual to
21 be exposed to more pesticides; correct?
22 MR. MICELI: Object to the form.
23 A It depends; right. So it could allow for
24 more time for exposure.

But it could allow for -- you know, it depends on the exposure period, how long someone is exposed to something for, whether they come into contact, etc.

BY MS. MIRSKY:

Q And it could also allow for additional time for any head injuries to occur; correct?

A Well, it depends on what you mean by "head injury."

Q Well, head injuries is defined as a risk factor for Parkinson's; correct?

A Right. But there's a wide range of head injuries and for Parkinson's in particular, it's severe head injuries, which is quite different than your run of the mill -- like what you might experience if you trip and fall in the park, for instance.

Q But nevertheless, if someone has an -- you -- strike that.

Nevertheless, an increased age would allow for more time for someone to experience a head injury; correct?

A Possibly. But severe head injury is relatively rare as well.

Q And increased age would increase the likelihood of someone developing any other risk factor as well; correct?

A Well, what do you mean by that?

Q Well, we discussed that increased body mass index is a risk factor and higher cholesterol could be a risk factor.

So increased age could increase the likelihood of someone developing or coming into contact with any other risk factor; correct?

A It depends; right? So not everyone who ages has an increase in body mass index; not everyone who ages has an increase in cholesterol; so it really does depend on what's happening.

Q But my question was whether it could increase the likelihood of someone developing or coming into contact with any of these risk factors?

A I mean, again, the longer someone lives, then there is more opportunity possibly to come into contact with these risk factors.

However, the one thing to remember is because of this long latency period, even though people are living longer, more often than not the exposures that happen for Parkinson's happen a long

time before Parkinson's symptoms present.

So if an exposure -- say someone increases their body mass index five years before Parkinsonian symptoms are present, it's likely not going to be the body mass index; right. Because the disease course takes a long time to develop prior to symptoms happening.

Q Right before the sentence that I just read on page four of your report, it -- you write, Aging alone is not responsible for the increasing rate of Parkinson's disease; is that correct?

A Correct.

Q And your citation to that number four is, "Global, regional, and national burden of Parkinson's disease, 1998 to 2016, A systematic analysis for the global burden of disease" study by Ray Dorsey and others; correct?

A Correct.

MS. MIRSKY: And we'll mark this as Exhibit 12.

(Whereupon, Boehme's Exhibit 12, Study Titled "Global, Regional, and National Burden of Parkinson's Disease, 1998 to 2016, A Systematic

Analysis for the Global Burden of Disease," was marked for identification.)

A Thank you.

BY MS. MIRSKY:

Q And this is that article; correct?

A Yes.

Q If you could turn to page -- oh no, sorry, the first page, 939, in the last paragraph on the second column, the third sentence says, The most important risk factor is age; correct?

A Well, this is in the introduction. This is setting the stage; right?

And so yes, what you've read is correct, but they're introducing what is known prior to what they are evaluating.

Q But this article does state that the most important risk factor is age?

MR. MICELI: Object --

A Yes --

MR. MICELI: -- to the form.

A Yes, it says that.

BY MS. MIRSKY:

Q And then it goes on to say that

Parkinson's disease, quote, also appears to be associated with industrial chemicals and pollutants such as pesticides, solvents, and metals; correct?

A Yes.

Q TCE and PCE are not specifically referenced here; is that correct?

A Correct.

Q If you could turn to page 941, please.

In the paragraph under "Risk Estimation" right at the bottom, the second column, it states, Of the 84 risks quantified in GBD 2016, only smoking was judged to have sufficient evidence for a relationship with Parkinson's disease with smoking associated with decreased risks; correct?

A Correct.

Q Okay. Turning back to page four of your report, you state that, The majority of PD is labeled idiopathic. Idiopathic is a term used in clinical medicine when there is no known cause of a disease or condition. However, the term can also be used when other known causes have been ruled out.

Did I read that correctly?

A Yes.

Q And do you agree with those sentences?

A Yes.
Q You did not cite any sources in support of this section; correct?
A Correct.
Q Are you aware of any specific studies where idiopathic is used when other known causes have been ruled out?
A What do you mean by this?
Q Well, you state that idiopathic can be used when other known causes have been ruled out; correct?
A Yes.
Q And are you aware of any studies where that definition is used?
A I mean, I've seen idiopathic Parkinson's discussed in the literature.
You know, when we are doing epidemiologic studies, though, we are looking at specific exposures. And so, you know, idiopathic, particularly in Parkinson's, is really referencing, you know, when there's no specific genetic cause for certain.
And so whether or not -- you know, what other risk factors are being evaluated is part of

the epidemiologic study.

Q But have you use- -- have you seen this definition of idiopathic used in other scientific studies?

A The term "idiopathic" is quite ubiquitous in epidemiology and clinical medicine and it's used in textbooks, it's used in reviews. It's -- it's pretty abundant.

Q And can the phrase still -- or can the term still be used when certain known causes have been ruled out?

A Well, right. So if you know that someone did not have exposure to those known causes, then they would be termed as idiopathic.

Q Okay. And is it your opinion that idiopathic Parkinson's only occurs when there isn't a known genetic cause?

MR. MICELI: Object to the form. Foundation.

A So I am not a neurologist. And my understanding is if there is no known cause, then someone is termed as having idiopathic Parkinson's.

Generally speaking, the idiopathic is really used more in reviews, again, textbooks, you

1 know, a person is not going to go to their
2 neurologist and necessarily receive a diagnosis.
3 There's no diagnosis code that's attributed to
4 idiopathic Parkinson's.
5 BY MS. MIRSKY:
6 Q Okay. On page five of your report, you
7 have a section titled "Environmental Solvent
8 Exposures and Risk of PD."
9 And halfway through the first paragraph
10 you say, quote, The first report of Parkinsonian
11 symptoms induced by solvent abuse was a 1994 case
12 report describing acute Parkinsonian -- Parkinsonism
13 following heavy abuse of a solvent; correct?
14 A Correct.
15 Q And your reference number 22 there is to
16 "Parkinsonism Induced by Solvent Abuse" by R.G. --
17 R.J. Uitti; is that correct?
18 A Correct.
19 THE WITNESS: Thank you.
20 BY MS. MIRSKY:
21 Q And is Exhibit 13 that communication?
22 A Yes.
23 Q Okay. In the summary paragraph right up
24 top it states that the patient developed

Parkinsonism acutely following heavy abuse of lacquer thinner; correct?

A Correct.

MR. MICELI: Excuse me. Just to interrupt, I know we are still on the record.

What is your ZIP code for the card that we are using?

MS. PLATT: Oh, 23455.

MR. MICELI: 23455. Okay. Okay. I will let them know.

MS. PLATT: Okay. Thank you.

MR. MICELI: Just give me one second to put these in.

COURT REPORTER: While he's doing that, are we marking a new exhibit?

MS. MIRSKY: Yes, that was exhibit --

THE WITNESS: 13.

MS. MIRSKY: -- 13.

(Whereupon, Boehme's Exhibit 13, Article Titled "Parkinsonism Induced by Solvent Abuse" by R.J. Uitti and Others, was marked for

identification.)

MR. MICELI: 23455. All right. Thank you.

BY MS. MIRSKY:

Q Okay. This study does not reference TCE or PCE; correct?

A Correct.

Q Back on page five of your report, you then cite to a, quote, 1998 case control study I -- which identified a relationship between well water use for at least ten consecutive years and occupational exposure to chemicals, specifically organic solvents, with increased risk of PD; correct?

A Correct.

Q And this reference number 23 is referencing "A case-control study of occupational and environmental risk factors for Parkinson's disease in the Emilia-Romagna region of Italy" by A. Smargiassi and others; correct?

A Correct.

MS. MIRSKY: Can I have Tab 13, please? Thank you.

And this is Exhibit 14 -- marking

Exhibit 14.

(Whereupon, Boehme's Exhibit 14, Study Titled, "A Case-control Study of Occupational and Environmental Risk Factors for Parkinson's Disease in the Emilia-Romagna Region of Italy" by A. Smargiassi and others, was marked for identification.)

BY MS. MIRSKY:

Q The exposure data in this study was collected by a questionnaire; correct?

A Correct.

Q And family medical history was also collected by questionnaire; correct?

A Correct.

Q On table one on page 711, cranial trauma has an OR of 2.88; correct?

A Correct.

Q And occupational chemical exposure has an OR of 2.13; correct?

A Correct.

Q You do not mention the finding of cranial trauma as a risk factor in this report in the discussion in your report; correct?

A Correct.
Q And there were no measurements of the contaminants in the well water in this study; correct?
A Correct.
Q And there was no information on any specific chemicals in this study; correct?
A Correct.
Q On page 712 in the middle of the third paragraph, it states, It was impossible to analyze the role of specific agents; correct?
A Correct.
Q You did not mention this in your report; correct?
A I didn't.
Q You state that this study further supported, quote, the burgeoning hypothesis implicating environmental and occupational risk factors in the development of PD, end quote; correct?
A So that statement is really again building the history as to why there were further investigations into these exposure-disease relationships. You know, with case report, early

case-control studies, you are starting to see a pattern that indicates more research should be done.

Q When you're referring to a "burgeoning hypothesis," are you referencing any studies beyond the Smargiassi and the Uitti studies?

A I referenced 23.

Q That is I think the --

MR. MICELI: Smar- --

BY MS. MIRSKY:

Q Smargiassi?

A Yeah.

Q So are you referencing any other studies besides those two?

A So in general, it's also building upon the case studies and the case series, which are referenced in both of those papers. And it's -- again, it's referencing the historical information of how the other research papers came to be.

Q Did you -- but you didn't cite any other studies in this section of your report?

A On that --

Q On that --

A -- sentence?

Q -- sentence?

A I did not.
Q And did you review the other cited case studies in the Smargiassi or the Uitti studies?
A I did.
Q And why did you not include them in your report?
A So I'm just trying to, again, present some of the historical information. It's -- there's more case studies involved and it's setting the stage for how the epidemiologic studies really came to be.
Q Okay. You then say that, A study of patients with PD found that exposure to solvents was directly responsible for earlier PD symptom onset and more severe symptoms; correct?
A Correct.
Q And you cite to "Hydrocarbon exposure and Parkinson's disease" by G. Pezzoli and others for that statement; correct?
A Yes.
Q Okay.
MS. MIRSKY: So this is Exhibit 15.
THE WITNESS: Thanks.
MS. MIRSKY: Mark as Exhibit 15.

(Whereupon, Boehme's Exhibit 15, Study Titled "Hydrocarbon Exposure and Parkinson's Disease" by G. Pezzoli and Others, was marked for identification.)

BY MS. MIRSKY:

Q And this is that study; correct?

A Correct.

Q This study compared features of Parkinson's patients who had solvent exposure history to those without solvent exposure history; correct?

A Correct.

Q And the result of this study was that exposed patients were diagnosed at 55; correct?

A I -- let me --

Q If you look in the abstract --

A It says exposed patients were younger.

Q And then in the parenthesis --

A Younger age at onset --

Q -- it says --

(Whereupon, the court reporter requests clarification.)

BY MS. MIRSKY:

Q And then in the parenthesis it says 55.2 plus or minus 9.8 versus 58.6 plus or minus ten years?

A Correct.

Q Okay. So these age ranges overlap; correct?

A With their standard deviation, yes.

Q And does this indicate that there was not strong evidence for this finding?

MR. MICELI: Object to the form.

A So what this indicates is that there's a lot more we needed to explore. Again, it's setting the stage for the epidemiologic studies.

And what we do when we are looking at case reports and case series, we'll often start quite broad -- in this case, solvents, hydrocarbons -- and then start to go into specific exposures.

And this is a small study and it's, you know, building upon other studies that have been done. And, you know, the authors determined that in these cases for these particular people, the solvent exposure was directly responsible for earlier PD

symptoms.

BY MS. MIRSKY:

Q But the overlapping age ranges would indicate that there was not strong evidence for this finding; correct?

MR. MICELI: Object to the form.

A Not necessarily.

So, you know, when they are looking at this and when they start to explore this, they are looking -- you know, in this abstract they just have this younger age at onset here. But when you start looking more -- where is it -- it's looking at the -- the entirety of this.

You know, they start showing symptoms earlier. The difference in the age at onset, you know, it's a younger age. And this is one of the studies that starts to suggest that solvent exposure -- or in this case, hydrocarbon exposure -- not only is on the pathway to Parkinson's, but there may be faster progression.

BY MS. MIRSKY:

Q Okay. In your report in that same paragraph you say, quote, A two thou- -- on page five -- A 2003 meta-analysis evaluating pesticides

and solvents on PD risk found a strong relationship between solvents and PD risk, parenthesize -- parenthesis, standardized odds ratio, or SOR, 1.35, 95 percent confidence interval, parenthesis, CI, 1.09 to 1.67, closed parenthesis, with the relationship being even stronger in higher quality studies, parenthesis, SOR 1.58, 95 percent confidence interval 1.23 to 2.04, end parenthesis; correct?

A Correct.

Q And earlier we talked about the strength of association.

You would consider an odds ratio of 1.35 to be a strong association?

A So an --

MR. MICELI: Object -- excuse me. Object to the form.

A An odds ratio of 1.35 indicates that someone has a 35 percent increased odds of disease.

And, you know, this again illustrates that people who have been exposed have a higher risk than those who have not been exposed. There's differences due to the exposure of the solvents in this case.

BY MS. MIRSKY:
Q But you categorized the standardized odds ratio of 1.35 as strong?
A Yes.
Q Correct?
And so that's your opinion that that is a strong standardized odds ratio?
A Yes.
MR. MICELI: Object to the form. Just take a breath so I can --
THE WITNESS: Sorry.
MR. MICELI: -- not overlap with you. Or talk over you. Sorry.
THE WITNESS: Okay.
BY MS. MIRSKY:
Q You cite to "Exposure to pesticides or solvents and risk of Parkinson's disease" by G. Pezzoli and E. Cedera (phonetic) -- excuse me, Cereda for this statement; correct?
A Yes.
MS. MIRSKY: Mark this as Exhibit 16.
(Whereupon, Boehme's Exhibit 16, Study Titled "Exposure to Pesticides

or Solvents and Risk of Parkinson's Disease" by G. Pezzoli and E. Cereda, was marked for identification.)

BY MS. MIRSKY:

Q Is this that document?

A Yes.

Q Okay. This study did not evaluate TCE specifically; correct?

A Correct.

Q And it did not evaluate PCE specifically; correct?

A Correct.

Q Do you believe that this study included all relevant studies on TCE and PD at that time?

A So this study is looking for studies that had pesticides or solvents, which would include other studies that include TCE and PCE.

Q Do you believe that the study included all relevant studies on TCE and -- and Parkinson's at that time?

MR. MICELI: Object to the form.

A Could you explain your question more, please?

BY MS. MIRSKY:

Q Do you believe that -- can you explain what a meta-analysis is?

A Right. So in a meta-analysis the authors go through publications that are associated to their exposure of interest and their outcome of interest.

And they generally have inclusion-exclusion criteria to determine what studies will be included in their meta-analysis.

And based on the inclusion-exclusion criteria, they'll review what they have pulled from their literature review to see what studies meet their inclusion-exclusion criteria and then incorporate them.

And in terms of inclusion-exclusion criteria, this study was specifically -- hold on -- inclusion included prospective -- so cohort studies -- and case-control studies. They were specifically looking for original studies, so no reviews, no other meta-analyses.

They did not include studies addressing fetal or early life exposure. They did not include studies using mortality data.

Yup. And that's what they were really

1 looking for.
2 Q And so based on these criteria and -- and
3 your analysis of the literature, do you believe that
4 this study reviewed all relevant studies on TCE and
5 Parkinson's?
6 MR. MICELI: Object to the form.
7 And foundation.
8 A Are you asking that at the time that this
9 study was conducted, did they include every
10 potential paper that had -- that met their
11 inclusion-exclusion criteria?
12 BY MS. MIRSKY:
13 Q Based on your review of the literature.
14 MR. MICELI: Object to the form.
15 A So I did not limit my review of the
16 literature to when this study would have been
17 conducted. And so I would have to limit to the
18 timeframe that they did their study in and use their
19 same search history -- or search terms in PubMed,
20 EMBASE, and CINAHL.
21 And they were looking at publications up
22 until December 2011.
23 BY MS. MIRSKY:
24 Q Okay. Let's go back to your report,

page 13, section titled "Mechanism of Action."
This relates to your opinions regarding the toxicological studies of TCE and PCE; correct?
A So this is an overview of the mechanism of action to help support the Bradford Hill considerations when evaluating the entirety of the body of literature.
Q So do you have any -- would you say that you have any opinions regarding the toxicological studies discussed in this section?
A I am not --
MR. MICELI: Object to the -- object to the form. Foundation.
A I am not a toxicologist.
BY MS. MIRSKY:
Q Okay. On page 14 you state in the last paragraph, It is widely accepted that high-dose exposure to TCE lead to PD or PD-like symptoms; correct?
A Yes.
Q But you don't define "widely" here; correct?
A Correct.
Q You cite to number 68, which is:

"Trichlorethylene: An update on an environmental contaminant with multiple health effects" by Lauren Lash -- excuse me, Lawrence Lash; correct?

A Yes.

MS. MIRSKY: Mark this as Exhibit 17.

(Whereupon, Boehme's Exhibit 17, Study Titled "Trichlorethylene: An Update on an Environmental Contaminant with Multiple Health Effects" by Lawrence Lash, was marked for identification.)

BY MS. MIRSKY:

Q This is that publication; correct?

A Yes.

Q Okay. If you would turn to page 514, please.

It states at the bottom of 514, Hence, there seemed to be multiple potential mechanisms by which TCE can cause neurodegeneration. What is uncertain, however, is the role of each mechanism and the tissue dosimetry that results in sufficient exposures to TCE, especially in the environment, that can cause these neurological effects; correct?

A Correct.
Q Do you agree with this statement?
MR. MICELI: Object to the form. Foundation.
A I am not a toxicologist.
BY MS. MIRSKY:
Q So do you have any basis to evaluate the findings in this paper?
A Could you elaborate on your question?
Q What are your qualifications to evaluate the findings in this paper?
A So this paper is a review and it's a review of the literature and it's been peer reviewed and published in the annual reviews.
And, again, it is to -- it was referenced and reviewed to set the stage of potential mechanisms of action, particularly when thinking through certain aspects of the Bradford Hill consideration, specifically biologic plausibility and consistency.
Q So do you have -- my question was, you know, what are your qualifications to analyze any of the findings in this study?
MR. MICELI: Object to the form.

A I didn't analyze the findings.
BY MS. MIRSKY:
Q So -- but you are using this study to support statements in your report; correct?
A Yes.
Q So how did you determine that this study or publication supports the statements in your report?
A It's peer reviewed. It's a review of the literature. It's summarizing findings in the mechanism of action literature and generally speaking, you know, peer-reviewed publications are accepted.
Q So other than the fact that this is peer-reviewed publication, you have no independent basis to analyze whether the findings in this publication are correct?
MR. MICELI: Object to the form. Foundation.
A You don't analyze reviews; right? We use reviews to help us summarize what has been done; we don't analyze them.
And so reviews are often referenced because it's a nice summarization of the information

1 to date.
2 And since I'm not a toxicologist, I did
3 rely on a review by someone who is respected in the
4 field, peer-review published review to help
5 summarize potential mechanisms of action.
6 But, again, there's no analysis done on
7 reviews.
8 BY MS. MIRSKY:
9 Q So the statements in this section of your
10 report are summaries of what is included in the
11 citations that you have in -- in this section; is
12 that correct?
13 A Correct.
14 Q Okay. And you have -- do you have any
15 independent basis to provide additional information
16 beyond what is in these studies?
17 A I am not a toxicologist.
18 Q Okay. And are you providing any
19 additional opinions beyond what is in these studies
20 with respect to mechanisms of action?
21 A Again, I'm not a toxicologist so I'm not
22 providing any opinions on anything related to the
23 toxicology.
24 Q Okay. So are you able to answer any

1 questions about any findings within the publications
2 cited in this section of your report?
3 MR. MICELI: Object to the form.
4 Foundation.
5 A I'm not a toxicologist. I can discuss
6 what's in those publications, but I am, again, not a
7 toxicologist. That is not my area of expertise.
8 BY MS. MIRSKY:
9 Q Okay. So do you know whether the
10 mechanism that would cause TCE exposure to lead to
11 PD or PD-like symptoms is uncertain?
12 MR. MICELI: Object to the form.
13 A I'm not a toxicologist.
14 BY MS. MIRSKY:
15 Q Okay. At the end of this discussion
16 you -- sorry, the -- let me start over.
17 At the top of page 14, you have a
18 citation to footnote two; do you see that?
19 A Yes.
20 Q And I think the text of footnote two is
21 on the previous page, page 13; correct?
22 A Correct.
23 Q And footnote two states, The fact that
24 PCE has received less attention in the scientific

1 literature is discussed in greater detail in the
2 report of Jason Cannon, Ph.D. Dr. Cannon, a
3 neurotoxicologist, explains the structural and
4 functional similarities of PCE and TCE, their
5 toxicities, and the relative attention devoted to
6 TCE and PCE in the scientific literature.
7 I reviewed the Cannon report for context
8 and to further understand the relative similarities
9 and toxicities of VOCs. I have -- I have, however,
10 conducted my own review of the literature in forming
11 my opinion stated in this report.
12 Did I read that correctly?
13 A Yes.
14 Q Do you agree with the opinions stated in
15 Dr. Cannon's report?
16 A So when I say that I've conducted my own
17 review of the literature in forming my opinions, I'm
18 really referencing the Bradford Hill considerations.
19 I'm an epidemiologist. I'm an expert in
20 epidemiology. I'm not an expert in toxicology or
21 neurotoxicology, so therefore I am not capable of
22 forming my own opinion on the research done behind
23 the information in the mechanism of action section.
24 What is important to me, though, as an

epidemiologist is understanding that there is robust literature on mechanism of action to inform biologic plausibility so that when we are considering all of the Bradford Hill considerations, that is enveloped within those considerations.

Again, I'm not a neurotoxicologist.

Q And so do you have any opinion one way or the other about the statements in Dr. Cannon's report?

A No.

Q Okay. And do you adopt Dr. Cannon's report as your own opinions?

MR. MICELI: Object to the form.

A I --

MR. MICELI: Foundation.

A They are not my opinions. I'm not a neurotoxicologist, so therefore, you know, the information on mechanism of action, again, is more around supporting the evaluation of the Bradford Hill considerations.

BY MS. MIRSKY:

Q And you incorporated what is doc -- in Dr. Cannon's report and what you have read here into your Bradford Hill considerations; correct?

MR. MICELI: Object to the form.
A So, again, what I've done here is just ensure that there is literature to support biologic plausibility.
BY MS. MIRSKY:
Q But you have not evaluated it one way or the other; correct?
MR. MICELI: Object to the form. Foundation.
A I'm not a neurotoxicologist.
BY MS. MIRSKY:
Q Okay. Do you know whether there are any animal studies investigating the effects of PCE specifically on the substantia nigra dopamine cells?
A I'm not a toxicologist.
Q So you don't know one way or the other?
A It's not my field.
Q So you don't know one way or the --
MR. MICELI: Object to the form. Foundation.
MS. MIRSKY: Can I finish my sentence, please?
MR. MICELI: Sure.

BY MS. MIRSKY:
Q So you don't know one way or the other; is that correct?
A Correct.
MR. MICELI: She finished. Let me object.
THE WITNESS: Okay.
BY MS. MIRSKY:
Q On the -- excuse me. In the last paragraph on page 13, you state in the middle of the -- in the middle of that paragraph, The neurotoxicity of TCE has been well described for decades, whereas the underlying mechanisms behind TCE and subsequent neurodegeneration are a recent development with multiple potential mechanisms hypothesized behind TCE causing neurodegeneration and PD.
Did I read that correctly?
A Correct.
Q You have no citation for this statement; correct?
A Correct. But I can provide a citation. It's within the citations in this entire -- entire section.

Q So in other words, you are incorporating the citations within this section to that statement?

A I would have to go and identify which publication that this statement came from, but any of the statements here are from the references within this section.

Q Even if there isn't a citation put after it?

A Yes.

Q So you don't know without checking what sources you would have relied on in writing this statement?

A It would --

MR. MICELI: Object.

Excuse me.

Object to the form.

A It would have been one of the citations within this section.

BY MS. MIRSKY:

Q Okay. You reference that there are multiple potential mechanisms hypothesized behind TCE causing neurodegeneration and PD; correct of?

A Where do you --

Q It's in the -- it's the last part of that

sentence that I just read?

MR. MICELI: Which sentence? Just on -- on 13?

MS. MIRSKY: Yes.

MR. MICELI: Okay.

A Could you repeat that so I just make sure --

BY MS. MIRSKY:

Q That there are multiple potential mechanisms hypothesized behind TCE causing neurodegeneration and PD; do you see that?

A Yes.

Q Okay. What hypotheses are you referring to here?

MR. MICELI: Object to the form. Foundation.

A I'm not a neurotoxicologist. I'm referring to what has been discussed in the literature in this section.

BY MS. MIRSKY:

Q So you can't identify any specific hypotheses within this statement at this point?

A That is not my expertise.

Q Okay. Are you aware whether any of these

hypotheses have been confirmed?

MR. MICELI: Object to the form.

A I'm not a neurotoxicologist.

BY MS. MIRSKY:

Q Okay. You then state, quote, The neurotoxicity of PCE has received less attention in the scientific literature. However, the evidence that is available implicates PCE as just as toxic, if not more so, than TCE.

Did I read that correctly?

A Yes.

Q And you have no citation for this statement; correct?

A Correct.

Q Did you rely on any sources in forming this opinion?

A So that statement is, again, relying on the references within this section.

I could find the exact reference that that relies on and update the report, if needed. I'm more than happy to do that.

Q Okay. Thank you.

You then state, The structural similarities between TCE and PCE, the commonality of

known metabolites, and the co-occurrence of TCE and PCE in exposures support extrapolating the toxic effects identified in TCE studies to PCE; is that correct?

A Yes.

Q And this is the sentence that has the footnote to Dr. Cannon's report; correct?

A Correct.

Q And you have no other citations for this statement; correct?

A Correct.

Q Is the common metabolite that you are referring to in this sentence known as TaClo?

MR. MICELI: Object to the form. Foundation.

A I would have to go back to the report to make sure that I am stating the correct metabolite.

BY MS. MIRSKY:

Q By "the report," you mean Dr. Cannon's report?

A Correct.

Q Do you know whether TaClo has been proven to be a metabolite for TCE?

MR. MICELI: Object to the form.

Foundation.

A I'm not a toxicologist.

Q Do you know whether TaClo has been proven to be a metabolite for PCE?

MR. MICELI: Same objection.

A I'm not a toxicologist.

BY MS. MIRSKY:

Q Okay. You do discuss TaClo earlier on page 13; correct?

A Yes.

Q Okay. And in this discussion of TaClo, you cite to "Trichlorethylene, A ubiquitous environmental contaminant in the risk for Parkinson's disease" by De Miranda and Greenamyre; is that correct?

A Yes.

MS. MIRSKY: And I will mark this as Exhibit 18.

(Whereupon, Boehme's Exhibit 18, Study Titled "Trichlorethylene, A Ubiquitous Environmental Contaminant in the Risk for Parkinson's Disease" by De Miranda and Greenamyre, was marked for identification.)

THE WITNESS: Thank you.
MR. MICELI: Are we done with 17?
MS. MIRSKY: Yes.
MR. MICELI: For now? Okay.
MS. MIRSKY: Yes.
MR. MICELI: All right.
BY MS. MIRSKY:
Q Is Exhibit 18 that document?
A Yes.
Q Dr. De Miranda is another expert that Plaintiffs have disclosed in this litigation; is that correct?
A Yes.
Q But you have not reviewed her report?
A Correct.
Q If you turn to page eight of this paper, the bottom of the first paragraph states, These data indicate that chronic exposure to TCE may be required to produce TaClo at a concentration that contributes to dopaminergic toxicity --
A Could you hold on?
Q Oh.
A I'm trying to find it.
Q Sure.

A Could you say that --
Q Sorry. It's the last sentence of the first paragraph.
A Oh, the first paragraph. Apologies. Okay.
Q No, that's okay.
Did you find it?
A Yes.
Q Okay. I'll start the quote over again.
These data indicate that chronic exposure to TCE may be required to produce TaClo at a concentration that contributes to dopaminergic toxicity. However, more studies are needed to assess whether TaClo production is the ultimate dopaminergic toxicant following chronic TCE exposure.
Did I read that correctly?
A Yes.
MR. MICELI: Just so I'm clear where you are reading from, is it here?
MS. MIRSKY: Yes. You are not looking at the --
MR. MICELI: Are you page ten or 11?
THE WITNESS: Page eight.

MS. MIRSKY: Page eight.
MR. MICELI: Oh, that makes sense then.
BY MS. MIRSKY:
Q Do you know whether those studies that are referenced as being needed here have been completed?
MR. MICELI: Object to form. Foundation.
A I'm not a toxicologist.
BY MS. MIRSKY:
Q So you don't know?
A That's not my area of expertise. I don't know what studies are ongoing or not.
Q Okay. Okay. In -- in your report in the sentence that I've read before -- sorry, now let me find it -- you state that the co-occurrence of TCE and PCE in exposures support extrapolating the toxic effects identified in TCE studies to PCE --
A Is this on page 13?
Q It starts -- it's in the sentence that starts on page 13 and then goes to page 14.
MR. MICELI: The one that ends with "see footnote two"?

MS. MIRSKY: Yes.
MR. MICELI: Okay.
THE WITNESS: Oh, okay.
Oh, okay. I see it.
MS. MIRSKY: Okay.
THE WITNESS: Could you restart that? Sorry.
MS. MIRSKY: Sure. That's fine.
BY MS. MIRSKY:
Q So you also state that the co-occurrence of TCE and PCE in exposures support extrapolating the toxic effects identified in TCE studies to PCE; correct?
MR. MICELI: Object.
A I state that.
MR. MICELI: Yeah.
BY MS. MIRSKY:
Q Okay. How does the co-occurrence of TCE and PCE in exposures extrapolate the toxic effects identified in TCE studies to PCE?
MR. MICELI: Object to the form. Foundation.
A I'm not an expert in that area.

BY MS. MIRSKY:
Q So you -- you can't explain how what's written in your report, The co-occurrence of TCE and PCE in exposures support extrapolating the toxic effects in TCE studies to PCE?
MR. MICELI: Object to the form. Foundation.
A Could you clarify exactly what you mean by that? Are you talking about the metabolism? Are you talking about the --
BY MS. MIRSKY:
Q I'm asking about the co-occurrence of TCE and PCE in exposures and why that supports extrapolating the toxic effects in TCE studies to PCE, as is written in your report?
MR. MICELI: Same objection. Foundation.
A Again, I am not a toxicologist and so this is based off of my understanding of what the toxicologists have written in their body of literature.
BY MS. MIRSKY:
Q Okay. Do epidemiological studies generally consider co-occurrence of contaminants to

be a limitation of a study?

A It depends.

Q Can that be considered a limitation of a study?

A Again, it depends. Not necessarily.

Q But it could be; is that correct?

A It really depends on what you are trying to study and how.

Q If you were trying to study whether TCE exposures can be -- let me start over.

If you were trying to determine whether the toxic effects of TCE exposure can be extrapolated to PCE, would the co-occurrence of those two contaminants be a limitation?

MR. MICELI: Object to the form. Foundation.

A So realistically, like, the co-occurrence studies are going to be more in the toxicology realm; right? So in terms of understanding the co-occurrence and how that could be a joint effect or not.

When we are doing our epidemiologic studies, having co-occurrence isn't necessarily a bad thing. We would expect to see differences in

our co-occurring population and if necessary, we could tease out people who had one exposure or the other or those who have co-occurrence.

And, again, it's going to be very dependent on the study design, the study itself, and how you are defining your exposures.

BY MS. MIRSKY:

Q If you are trying to extrapolate from TCE to PCE, and TCE and PCE are co-occurring in exposures, would that be a limitation to understanding whether that extrapolation could occur?

MR. MICELI: Object to the form. Foundation.

A It depends. It really does depend.

BY MS. MIRSKY:

Q Okay. Are you aware of any mechanistic studies that only focus on PCE and Parkinson's?

A Again, I'm not a toxicologist.

You know, I've summarized some of the mechanisms here. And, again, that's more in support of the Bradford Hill consideration.

Q Okay. So are you aware of any mechanistic studies that only focus on PCE and

Parkinson's?

MR. MICELI: Object to the form.

A That's not my area of expertise.

BY MS. MIRSKY:

Q So --

A I should -- I should not be speaking about any of the toxicology studies as it's not my area of expertise.

Q But they are included in -- as support for your report?

A I have --

MR. MICELI: Object to the form.

A I have referenced studies in terms of referencing the mechanism of action. Again, it's more that they are -- it's information about mechanism of action in the literature to support biologic plausibility.

BY MS. MIRSKY:

Q Did you review any studies that do not support mechanism of action as described in your report?

A Could you elaborate on that question?

MS. MIRSKY: Well, could I have Tab 18, please?

I need a new sticker.
MR. MICELI: But you are on 19 now.
MS. MIRSKY: Yes.
MR. MICELI: Just to be -- okay.
MS. MIRSKY: I'm going to mark this as Exhibit 19.
(Whereupon, Boehme's Exhibit 19, Analysis Titled "Comparative Analysis of Metabolism of Trichlorethylene and Tetrachlorethylene Among Mouse Tissues and Strains," was marked for identification.)
MS. MIRSKY: It is an analysis titled "Comparative analysis of metabolism of trichlorethylene and tetrachlorethylene among mouse tissues and strains."
BY MS. MIRSKY:
Q Have you seen this before?
A This looks familiar, yes.
Q So you believe you've reviewed it before?
A Yes.
Q Would you turn to page 11, please?
In the conclusion section it states, In

summary, this study provides a comparative analysis for the metabolisms between TCE and PCE. We show that one atom replacement of chlorine -- excuse me -- of chlorine can substantially affect the metabolism via both oxidative and glutathione conjugation pathways. The qualitative and quantitative differences between TCE and PCE metabolites as well as the tissue-specific distribution of metabolites, can shed light on the differences between TCE- and PCE-induced toxicities.

Did I read that correct?

A Yes.

Q Okay. Let's go back to the -- in this report to the abstract. About two-thirds of the way down it says, quote, As expected, the flux through oxidation metabolism pathway predominated over that through conjugation across all mouse strains examined. It is 1200 to 3800 fold higher for TCE and 26 to 34 fold higher for PCE. However, the flux through glutathione conjugation, albeit a minor metabolic pathway, was 21-fold higher for PCE as compared to TCE.

Did I read that correctly?

A Yes.

Q And under section four, page eight, under "Discussion" it states, The most notable finding of this comparative study of toxicokinetics of TCE and PCE in mice is the observation of considerable differences in metabolism between these structurally similar compounds. A larger portion of the parent compound undergoes metabolism in TCE-treated mice (15.7 percent to 38.3 percent) as compared to PCE-treated mice (6.6 percent to 9.7 percent), a finding that provides empirical data in strong support of the estimates from PBPK models. The more efficient oxidative metabolism of TCE as compared to PCE is also concordant with previous animal studies and PBPK modeling estimates.

Did I read that correctly?

A Yes.

Q And is it your understanding that PBPK stands for physiologically-based pharmacokinectic modeling?

MR. MICELI: Object to the form. Foundation.

A That is not my area of expertise.

BY MS. MIRSKY:

Q Are you able to interpret any of the

1 sections that were just read?
2 A That is not my area of expertise.
3 Q Okay. There are studies cited to in this
4 paragraph that support these statements, including
5 Cichocki, et al., 2017; Green and Prout, 1985; and
6 Larson and Bowl (phonetic), 1992.
7 Do you know whether you reviewed any of
8 these studies?
9 A I would have to look at them. I don't
10 know off the top of my head.
11 Q And you said that you had seen this study
12 previously; correct?
13 A Yes.
14 Q But it's not in your materials considered
15 list; correct?
16 A Correct.
17 Q Do you know whether this study came up in
18 your literature review?
19 A I don't remember where I saw this, but I
20 have seen this.
21 And, again, I'm not a toxicologist. This
22 is not my area of expertise.
23 Q How did you decide which toxicology
24 studies to cite to in your report?

A So, again, I was looking for toxicology studies that supported mechanism of action for biologic plausibility.

It is not my role as the epidemiologist to evaluate the mechanism of action studies to determine whether that body of literature is appropriate or not. It's more that in terms of identifying a body of literature for biologic mechanism ensuring that there is a body of literature.

Q But were you able to evaluate whether the literature that you included in your review in support of this report actually provided for a finding of biological plausibility?

MR. MICELI: Object to the form.

A So it's more that that's -- that it's out there.

If when I was doing my review there was no mechanism of action studies available, then I wouldn't be able to say that there is biologic plausibility.

And if you think about the term "biologic plausibility," it's that it's plausible, possible; right? It's not that it's definitive. It's that

there are potential explanations from a mechanistic perspective that would explain the epidemiologic findings. And it's not my role to review the mechanistic studies.

BY MS. MIRSKY:

Q So in your opinion, the existence of the studies that are cited in your report is sufficient to support biological plausibility?

A Again, I'm not going to speak on the mechanism of action.

But the fact that there are publications that support mechanism of action and indicate potential different mechanisms of action support that there's -- that the link is biologically plausible. That we are seeing this not just in our epidemiologic studies, but also in our bench science studies.

And I do want to take a moment to state that, you know, biologic plausibility, while it's one of the Hill considerations, whether you have it or not is not necessary to determine causality.

It is helpful if you have it in determining causality, but there are plenty of examples where we see epidemiologic evidence that we

1 would say is causal but we do not have biologic
2 plausibility.
3 However, in the case of this, there are
4 publications that support mechanisms of action,
5 which would support biologic plausibility.
6 Q Are toxilogi- -- toxicological studies
7 necessary in order to undertake a Bradford Hill
8 analysis?
9 MR. MICELI: Object to the form.
10 A It depends on the research question.
11 BY MS. MIRSKY:
12 Q In this instance, did you believe the
13 toxicological studies are -- were necessary to
14 perform your Bradford Hill analysis?
15 MR. MICELI: Object to the form.
16 A My Bradford Hill analysis could stand on
17 its own without the biologic plausibility. However,
18 having potential mechanisms of action do help in
19 explaining what we are seeing in the epidemiologic
20 literature.
21 BY MS. MIRSKY:
22 Q But as you testified, you haven't
23 analyzed the actual findings of the studies cited in
24 your report?

A For the mechanism of action, because that is not my expertise.

Q And we -- I asked about how you decided which studies to include in your report -- in your materials considered lists and which not to.

If you are not analyzing the reports themselves, how did you determine which ones to include and which ones not to include, like this Luo study, for example?

A So specifically for the mechanism of action, you know, I'm not looking at metabolism; right. This is metabolism.

I'm looking as the mechanism of action studies that are looking at neurotoxicity and, you know, what potential mechanisms are available.

And so it was summarizing the studies that have done -- that have evaluated mechanism of action that showed potential mechanisms.

Q On page 13 you do discuss that TCE and PCE break down, share a common metabolite such as TalCo, which is implicated in toxicity resulting in mitochondrial dysfunction; correct?

A Yes.

Q So you -- you did consider TalCo and --

or metabolites in the mechanism of action section; correct?

A Briefly. It's, again, part of the entirety of the possible mechanisms that could explain what we are seeing in the epidemiologic studies.

Q Okay. In the first paragraph on page -- excuse me.

In the first paragraph under "Mechanism of Action," the second sentence, it states, Many of the mechanisms relating TCE to PD are thought to be similar for PCE as PCE differs from TCE through an additional chlorine atom; correct?

A Yes.

Q Are you able to further analyze this statement?

MR. MICELI: Object to the form. Foundation.

A No. That is not my area of expertise.

BY MS. MIRSKY:

Q Do you know that -- excuse me -- are you aware that methanol and ethanol differ by one atom?

MR. MICELI: Object to the form. Foundation.

A That is not my area of expertise.
BY MS. MIRSKY:
Q But do you know whether methanol and ethanol can cause different health outcomes?
MR. MICELI: Object to form. Foundation.
A From an epidemiologic perspective, they are both considered toxic.
BY MS. MIRSKY:
Q But can they cause different health outcomes?
MR. MICELI: Object to the form. Foundation.
A That is not my area of expertise.
BY MS. MIRSKY:
Q Okay. You said earlier that you are not an expert in genetics; correct?
A Correct.
Q Is your section in your report on genetics similar to your report on mechanism of action?
A I don't have a section on genetics.
Q Excuse me.
Are the portions of your report that

speak to LRRK2 activity --

A LRRK2.

(Whereupon, the court reporter requests clarification.)

MS. MIRSKY: LR- -- it's LRRK2.

BY MS. MIRSKY:

Q Sorry, did you say LLRK2?

A Uh-huh.

(Whereupon, the court reporter requests clarification.)

MR. MICELI: Yeah. Start at the top.

COURT REPORTER: I interrupted you and then --

MS. MIRSKY: That's fine. That's fine.

BY MS. MIRSKY:

Q You do discuss LRRK2 in your report in a couple of sections; correct?

A Correct.

Q Is your discussion of LRRK2 in your report similar to your discussion of mechanism of action in your report in that you are not a geneticist and so you cannot provide opinions on the

statements in your report?
A So I --
MR. MICELI: Excuse me.
Object to the form.
Go ahead.
A I'm not a geneticist. What I'm speaking to in terms of LRRK2 is from an epidemiologic perspective as LRRK2 has been identified as a risk factor for Parkinson's.
And so in the context of understanding LRRK2's role in Parkinson's from an epidemiologic perspective, I can speak to that.
I cannot speak to LRRK2 from a genetic perspective.
BY MS. MIRSKY: Okay. Are you aware of any mutations that would prevent TCE "metabolation" -- metabolization?
A That is not my area of expertise.
Q Do you know whether if a mutation prevents the metabolism of TCE, that would result in a buildup of unmetabolized TCE?
MR. MICELI: Object to the form.
Foundation.
A That is not my area of expertise.

BY MS. MIRSKY:
Q Do you have an opinion as to whether any buildup of unmetabolized TCE increases the risk of Parkinson's?
A That is not my area of expertise.
Q So you do not have an opinion on that?
A That's not my area of expertise.
Q Is it -- do you know whether any LRRK2 -- and again, that's LRRK2 -- mutation could be caused by the parent TCE or PCE molecule?
MR. MICELI: Object to the form. Foundation.
A That's not my area of expertise.
BY MS. MIRSKY:
Q Do you know whether any LRRK2 mutation could be caused by a metabolite of TCE or PCE?
MR. MICELI: Same objection. Foundation.
A That is not my area of expertise.
BY MS. MIRSKY:
Q Okay. On page 12 of your report -- sorry. Let me just -- just give me one second.
Okay. Sorry.
On page four of your report, apologies,

you state that -- let me get the exact --

MR. MICELI: Just tell us where you are --

MS. MIRSKY: Yeah.

MR. MICELI: -- when you get there.

MS. MIRSKY: I will.

BY MS. MIRSKY:

Q It's halfway through the first full paragraph beginning with, Leucine-rich repeat kinase 2, or LRRK2, activity from LRRK2 mutations are known to cause PD.

Did I read that correctly?

A Yes.

Q Is LRRK2 an enzyme?

MR. MICELI: Object to the form. Foundation.

A I am not an expert in genetics. I should not be speaking to anything other than LRRK2 from an epidemiologic perspective.

BY MS. MIRSKY:

Q Okay. You cite to "Phenotype, genotype, and worldwide genetic penetrants of LLRK2-associated Parkinson's disease: A case control study" by Daniel Healy and others; correct?

A Yes.

MS. MIRSKY: That's 37 -- sorry.

MR. MICELI: This is a good sign. She's going to box number two.

MS. MIRSKY: For now. Okay. I'm going to mark this as Exhibit 20.

THE WITNESS: Okay.

(Whereupon, Boehme's Exhibit 20, Study Titled "Phenotype, Genotype, and Worldwide Genetic Penetrants of LLRK2-associated Parkinson's Disease: A Case Control Study" by Daniel Healy and others, was marked for identification.)

MR. MICELI: Thank you.

BY MS. MIRSKY:

Q This is that study; correct?

A Correct.

Q You state on page four of your report about halfway -- or two-thirds down this main paragraph, Elevated LRRK2 kinase activity alone is not enough to cause PD in people who have the LRRK2 mutation; correct?

A Yes.

Q And do you agree with that sentence?
MR. MICELI: Object to the form.
A So, again, within this, this is doing an overview of Parkinson's, so there are statements within this section that come from neurology textbooks. It's presenting -- it's setting the stage. This is not based on my epidemiologic expertise. This is all introducing other factors that may be relevant.
And so I am referencing this -- this particular Healy paper to, again, give an overview of LRRK2 and our understanding to date about LRRK2 and PD.
BY MS. MIRSKY:
Q And so the Healy study also does not find that elevated LRRK2 alone is enough to cause PD in people who have the LRRK2 mutations; correct?
A That was their finding.
Q Okay. Do you know whether in addition to any LRRK2 mutation, environmental exposure must also be present to cause Parkinson's?
MR. MICELI: Object to the form.
A Could you repeat your question?

BY MS. MIRSKY:
Q Do you know whether in addition to any LRRK2 mutation, environmental exposure must also be present to cause Parkinson's?
MR. MICELI: Object to the form.
A So what do you mean by that?
BY MS. MIRSKY:
Q Whether if someone has a LRRK2 mutation, environmental -- they must also be exposed to something in the environment in order to develop Parkinson's?
MR. MICELI: Same objection. Foundation.
A Again, I'm not a geneticist and so that's really outside of my area of expertise.
BY MS. MIRSKY:
Q Okay. Do you have any opinions or knowledge beyond what is written in the studies that you cited in your discussion of LRRK2?
A It's pretty much here. This is, again, an overview of the literature. This is not coming from my personal knowledge.
Q Okay. And -- and in addition to your personal knowledge, to your scientific knowledge as

well?

A Well, that's what I meant. Apologies. Yes. Scientific knowledge.

Q Thank you.

Do you know whether TCE continues to cause additional mutations of LRRK2 after exposure has ceased?

MR. MICELI: Object to the form.

A That is not my area of expertise.

BY MS. MIRSKY:

Q Okay. And in writing the discussion of LRRK2 in your report, how did you decide which studies to rely on and put in your materials considered list?

A So it was partially from, you know, reviews that have been done, especially if we are talking about, like, the overarching risk factors, understanding that, you know, the genetic factors are not the end-all cause for Parkinson's, and so knowing that I would have to look at LRRK2, and recognizing that LRRK2, again, does not fully explain Parkinson's, and so it was through literature review -- reviews.

And, again, this is to give a broad

overview to set the stage as to why we are interested in evaluating TCE and/or PCE with Parkinson's.

Q There are some instances in which genetic factors can cause Parkinson's; correct?

A Correct.

MR. MICELI: Object -- hold -- object to the form. Foundation.

A Correct. However, they are only present in a very small percentage of people with PD.

BY MS. MIRSKY:

Q Okay. On page 14 of your report at the bottom of the first main paragraph in a separate discussion of -- of LRRK2 mutations, you state that, Gene environment interactions are why only some individuals exposed to TCE or PCE develop PD; correct?

A Where is this --

Q It's the bottom of the main paragraph on page 14.

A Main paragraph.

MR. MICELI: Oh, 14.

A Okay. The second to last sentence?

BY MS. MIRSKY:
Q The last sentence. I can read it again.
A Yes, please.
Q These gene environment interactions are why only some individuals exposed to TCE or PCE develop PD; correct?
A Correct.
Q Is it your opinion that a gene environment interaction is necessary for an individual exposed to TCE or PCE to develop Parkinson's?
MR. MICELI: Object to the form. Foundation.
A Again, this is within the mechanism of action section and that is outside my area of expertise.
BY MS. MIRSKY:
Q Okay. On page 12 -- I know we're flipping around a little bit.
On page 12 you state, It is highly improbable the difference in the risk of PD between Camp Pendleton and Camp Lejeune in closely matched cohorts of -- of both military personnel and civilians is due to genetic factors. This is

1 illustrated by calculating the excess attributable
2 risk percent using data from this study. The
3 attributable risk percent for developing PD due to
4 TCE/PCE exposure in this population is 44.7 percent.
5 Meaning 44.7 percent of the PD risk is explained by
6 exposure to TCE or PCE at Camp Lejeune, the only
7 major difference between the two populations;
8 correct?
9 A Correct.
10 Q Do you have a citation for the assertion
11 that exposure to TCE or PCE is the only major
12 difference between the two populations?
13 A I do not. However, in thinking through
14 populations, you know, these are both large bases
15 and whether someone ends up at Camp Lejeune or Camp
16 Pendleton would not be based on their genetics or
17 factors such as that.
18 So realistically speaking, you know, when
19 we are thinking about a -- an appropriate comparator
20 group -- so we know that Camp Lejeune had the higher
21 levels of TCE and PCE -- it would not be appropriate
22 to compare the members of Camp Lejeune to the
23 general U.S. population because there are inherent
24 differences between being on a military base as

opposed to living in New York City, for instance.

And so to have an appropriate comparator group, in this case, an unexposed group, we would need another military base. And so therefore, the authors chose Camp Pendleton.

And the downstream effects that would drive someone to be at Camp Pendleton or Camp Lejeune and potential other risk factors for PD should not be different between the two.

Q Okay. How did you calculate the attributable risk percent?

A So an attributable -- attributable risk percent is really another way of looking at your risk ratios; right. So we already looked at our risk ratios, which have indicated that people who were exposed, people who were at Camp Lejeune, had a higher incidence of PD than those who were at Camp Pendleton. This attributable risk percent is another way of showing that.

And so what that means is we're taking Camp Pendleton and we're assuming that Camp Pendleton being unexposed would show us what Camp Lejeune would look like had they not been exposed.

1 So it would give us an estimate as to the
2 proportion of people who should develop PD from the
3 other factors that we've discussed: genetics,
4 potential head injury, age, pesticides; right.
5 So knowing that the causes of PD are
6 multifactorial, we have to have this unexposed group
7 so that we can have an understanding of what our
8 base disease rate would look like.
9 Now, with Camp Pendleton -- sorry, Camp
10 Lejeune, because there was a higher proportion of
11 people with Parkinson's, we are looking at what's --
12 called this -- it's sometimes referred to as excess
13 risk.
14 But essentially we are trying to drill
15 down on, hey, we know that this exposure is
16 different. This is the theoretically only
17 difference between Camp Pendleton and Camp Lejeune.
18 So how much more Parkinson's are we seeing because
19 of this difference?
20 And so that's what the attributable risk
21 percent does. It's another -- again, it's another
22 way of showing exactly what our measures of
23 association have shown in this publication. It's
24 just a different way of presenting it.

It's using the same information. You're looking at risk in exposed and risk in unexposed and determining how much more of the risk is due to your exposure of interest, in this case, Camp Lejeune.

Q So in -- in this paragraph, you list a 44.7 percent number.

How did you calculate that number?

A So within the publication, they had the number of people who developed Parkinson's, they had the number of people who didn't for both Camp Lejeune and Camp Pendleton. And so you take the risk in the exposed minus the risk in unexposed, divided by risk in exposed, times a hundred percent.

So it's, again, another way of presenting very similar information that their measure of association did.

So more often than not, we are used to seeing measures of association. However, sometimes it's helpful to have additional measures that we can say, all right, we know that Camp Pendleton, there are people who developed Parkinson's there. They're are going to be representative of the other causes of Parkinson's.

How much more Parkinson's would we see

due to this additional exposure?

Q Would you agree that, as you described it, an attributable -- attributable risk percent can also be known as a population attributable fraction?

A I'd have to go back and look at the equations.

Q Okay. In calculating the attributable risk percent here, is it your understanding that there are several assumptions that must be met for this calculation to be valid?

MR. MICELI: Object to the form. Foundation.

A So, again, there's assumptions for everything; right?

There's assumptions for measures of association. There's assumptions for a study design. There's assumptions for attributable risk percent. And, again, this is just another way of highlighting what our measure of association has already shown.

BY MS. MIRSKY:

Q Would you agree that three assumptions for a -- excuse me -- attributable risk percent are whether the exposure can cause the condition if the

exposure is removed -- well, let me just start with that, the exposure can cause the condition?

A So all of what we are doing is making the assumption the exposure can cause the condition.

Q Second, if the exposure is removed, the risk of the condition in those who were exposed immediately becomes the same as those who were never exposed?

MR. MICELI: Object to the form.

A I'd have to look back at the exact definition.

But what it's getting at is if those who had been exposed were never exposed, what would their underlying risk be.

BY MS. MIRSKY:

Q Okay. And three, that removing the exposure does not affect other risk factors for that condition?

A So that's really building on what I was just explaining. Like, in our unexposed group, we understand that there are other risk factors that's giving us our baseline rate.

And what we are interested in, and this is what we're doing with our measure of association

as well, we're interested in, okay, with this specific exposure, is it the same as what we see generally, indicating that that would not be an exposure, or is it heightened. And if so, you know, by how much.

And that's what we are doing when we calculate our measures of association. We're really attempting different ways of quantifying whether or not our exposure contributes and is on the causal pathway to our disease.

Q So with your framing of those assumptions, do you -- do you agree that those are part of an analysis of any -- I'm sorry, I keep losing -- attributable risk percent calculation?

MR. MICELI: Object to the form.

A So, again, you know, when we're doing our epidemiologic studies, we are already making the assumption -- we -- when we're conducting an epidemiologic study, one of the first things we do is draw a DAG or a structural framework to indicate why we think something might be on a causal pathway.

And so we're making those assumptions when we set up our epidemiologic studies. This is in line with the assumptions made to set up the

epidemiologic studies.
BY MS. MIRSKY:
Q Do any of the studies that you reviewed for this report demonstrate that if exposure to TCE were removed, the risk of Parkinson's in those exposed to TCE would immediately become the same as those who were never exposed?
MR. MICELI: Object to the form.
A So you're -- you're misconstruing what the counterfactual actually is. It's not so much that it's removed.
It's that if it were never present. So the idea of having an unexposed group is that we are trying to emulate what the exposed group would have looked like had they never been exposed.
It's not about removing the risk factor. It's about it not being there and what the risk of Parkinson's would be in the absence of that factor.
BY MS. MIRSKY:
Q Do any of the studies that you cite demonstrate that if there had not been exposure to TCE, the risk of those in the exposed to TCE group would immediately become the same as those who were never exposed?

MR. MICELI: Object to the form.

A So, again, this is why Camp Pendleton is used because it's an appropriate comparator group. It's an unexposed group that is representative of what Camp Lejeune would have looked like had they not been exposed to TCE or PCE.

And so if Camp Lejeune had never had TCE or PCE exposure, we would expect that their incidence of Parkinson's would be the same as Camp Pendleton.

And that's why we take a lot of care when we are setting up our studies in terms of selection to ensure that our unexposed group really does reflect, like I said, the counterfactual experience.

And the counterfactual experience is this idea of what would have happened to this group had they never been exposed.

BY MS. MIRSKY:

Q Do any of the studies that you cite demonstrate that removing or never having exposure to TCE would not affect other risk factors for Parkinson's?

MR. MICELI: Object to the form.

A So, again, in the counterfactual

experience of, again, Camp Pendleton, it's not about removing. It's if TCE is not present, which at Camp Pendleton it's not.

And so we still see that there are people who develop PD at Camp Pendleton, likely because of other factors that we've discussed, and what we are showing is that due to this increased risk or due to this exposure of interest, this population by being exposed doesn't have the same experience as the unexposed.

We would expect had they never been exposed, they would have the same experience as the unexposed.

MS. MIRSKY: Okay. We can take a break now.

THE WITNESS: For lunch?

MS. MIRSKY: Yes.

THE VIDEOGRAPHER: The time right now is 1:24 p.m. and we are off the record.

(Whereupon, there was a lunch recess taken from 1:24 p.m. to 2:13 p.m.)

THE VIDEOGRAPHER: The time right now is 2:13 p.m. and we are back on the

record.
BY MS. MIRSKY:
Q Good afternoon, Dr. Boehme.
If you could turn to page five in your report, at the bottom there's a section titled "TCE, PCE, and PD"; correct?
A Yes.
Q And the second sentence states, TCE has long been implicated as a cause of several poor health outcomes, including numerous cancers, neural tube defects, and PD; correct?
A Yes.
Q And --
MS. MIRSKY: If I can have Tab 19, please?
BY MS. MIRSKY:
Q And you site to "Human Health Effects of Trichlorethylene: Key Findings and Scientific Issues" by W.A. Chiu and others; correct?
A Yes.
MS. MIRSKY: I'll mark this as Exhibit 21.
(Whereupon, Boehme's Exhibit 21, Study Titled "Human Health Effects of

Trichlorethylene: Key Findings and Scientific Issues" by W.A. Chiu and others, was marked for identification.)

BY MS. MIRSKY:

Q Is this that document?

A Yes.

Q Okay.

MR. MICELI: This is 21, just so I'm --

MS. MIRSKY: Yes.

MR. MICELI: Okay. Thank you.

BY MS. MIRSKY:

Q Are you aware that this does not reference Parkinson's or neural tube defects specifically?

A I would need to go back and reread this paper to see where I pulled this from.

Q Okay. That's okay.

If you go to page 308, please, towards the top of the third column, and this statement is in reference to ATSDR and Dr. Bove studies that are not related to Camp Lejeune.

The publication states, Interpretation of

these data has been controversial because many of the studies are limited by small numbers of cases, insufficient exposure characterization, chemical co-exposures, and other methodological deficiencies; do you see that, correct?

A I see that.

Q Would you agree that proper interpretation of data may be difficult if studies are limited by a small number of cases?

MR. MICELI: Object to the form.

A It depends. I have not read those papers and so I would need to read them.

And, you know, again, like I was talking about earlier, when it comes to small numbers, it is dependent on what you are researching; right. So small numbers in something that's highly prevalent, like hypertension, is very different than small numbers in Parkinson's, especially when we are taking into consideration rare exposures. And so it's going to be dependent on a number of factors.

BY MS. MIRSKY:

Q But does the -- what you are studying, does that impact whether or not the small number of cases in a study can make it difficult to interpret

the data?

MR. MICELI: Object to the form.

A It really depends, like I said, on a number of factors: on the exposure, on the outcome, on the study design; you know, small is relative.

And so it depends on what they are referring to as small.

Q Do you have a definition under which -- you said earlier I think that -- let me start over.

I think you said earlier that you tried to look at studies with more than a hundred people for purposes of this report; correct?

A I didn't limit but, you know, the preference is to studies that have larger populations because this is a rare exposure, this is a relatively rare outcome. I did include information on studies that had under a hundred.

And, again, it's all about the context of the entirety of the literature and -- and small is relative, so it really does kind of depend.

Q Do you have a definition for what would constitute a small study?

A Again --

Q -- in this --

A -- that's going to be --
Q -- context?
A -- very dependent on the exposure, the outcome, the population, how the study is conducted.
Q Okay. Would you agree that proper interpretation of data may be difficult if studies are limited by insufficient exposure information?
MR. MICELI: Object to the form.
A Again, it depends. Like, it depends on what they are talking about with insufficient exposure characterization and it depends on what the study is doing. If the study acknowledges that how the exposure is being defined may not be as accurate as some other definition.
There are things that can be done in the analysis section to explore the extent that that could have on the measure of association.
So it's -- again, it's very dependent on what you are looking at.
BY MS. MIRSKY:
Q Would you agree that certain types of exposure characterization can impact the quality of a study?
MR. MICELI: Object to the form.

A So our exposure definition is often limited to what we can define by our data when and how. And even in more broad terms for exposure characterization, that still provides us information. And, again, we can always do quantitative bias techniques to assess, you know, how -- whether any misclassification would have an impact on our measure of association.

And so it is very dependent on how the exposure is being defined and, you know, the study of interest and the research question of interest.

BY MS. MIRSKY:

Q We talked about co-exposures earlier in both the mechanism of action section and a bit in respect to epidemiology.

This article that you cited to is stating that chemical co-exposures could make -- could be a limitation of a study; do you agree with that?

MR. MICELI: Object to the form.

A It depends.

MR. MICELI: Are you referring to -- are you still working on 21?

MS. MIRSKY: Yes.

MR. MICELI: Okay.

A It really depends. And it depends especially, like, how the epidemiologic study is set up, how the exposures are defined, whether there's the ability to tease out co-exposures and your overarching research question of interest.

BY MS. MIRSKY:

Q Would you agree that proper interpretation of data may be difficult if studies are limited by methodological deficiencies?

MR. MICELI: Object to the form.

A It depends on what they're -- what they're saying is a methodologic deficiency.

So every single study out there has limitations. If they're saying that, you know, any limitation is a methodologic deficiency, that's problematic; right.

We interpret our findings within the context of our limitations. You know, if there are gross methodologic deficiencies, inappropriate study design use, for instance, that could make things harder to interpret.

However, there's more leeway in robustness to deviations from what you should be doing in terms of epi- -- epidemiologic methods.

BY MS. MIRSKY:

Q What are some other examples of gross deficiencies that you can think of?

MR. MICELI: Excuse me. Object to the form.

A It depends. It depends on the research question, it depends on the intent of the research question, what you are setting out to do.

And, again, as long as the results are being interpreted within the context of the limitations, then, you know, you are recognizing that there are limitations.

BY MS. MIRSKY:

Q So is -- do you believe that it's important in analyzing an epidemiological study to discuss any of the limitations that we just talked about? And I can -- I can list them individually.

Do you think that it's important to when analyzing an epidemiological study discuss whether a study has a small population?

A So most epidemiologic studies if they have a small population will list that in there and will discuss that it's a small population. And, again, small population is relative to the research

1 question of interest.
2 Q And so do you think that it is important
3 to -- when you're analyzing an epidemiological study
4 that's been reported on, is it important to consider
5 whether the population was small in your analysis of
6 the study overall?
7 A So when reading and interpreting a study
8 that's been published, you know, it's one factor
9 that you include with everything else; right? So
10 it's the entirety of the publication as a whole, not
11 just was it small, was it not small, what did the
12 authors do. You know, it's thinking about
13 everything in the context.
14 And, again, it's very dependent on the
15 research question and -- and the ultimate goal of
16 the study.
17 Q Aside from any ultimate conclusion that
18 it may impact, do you believe that it is important
19 to note whether there is a stall -- small population
20 when analyzing a study?
21 MR. MICELI: Object to the form.
22 A It depends; right? I mean, if you are
23 publishing a study on 30 people, we are all going to
24 acknowledge that that's a smaller sample size.

But if it's a very, very, very rare genetic disorder, that might be the largest sample size you can obtain.

And whether someone states it or not, you know, as an informed reader, as an epidemiologic expert, I'm able to look at publications and think about additional limitations that may or may not have been listed.

BY MS. MIRSKY:

Q So I'm thinking about this more from your perspective as a reader rather than the writer of the study.

As the reader and interpreter of the study, regardless of whether it impacts your ultimate analysis of the -- the study itself, do you think that it is important to consider whether the study has a small population?

A Again, it depends. It really is going to depend on what's available on the study question, how the exposures and outcomes are being defined.

Q As a reader, do you think that it's important to note whether a study that you are analyzing has insufficient exposure characterization?

A It depends; right?

So often in epi, we try to -- to evaluate multiple studies that have used different exposure definitions. And the reason for that is there's no one perfect exposure definition.

And so by looking at different ways of quantifying the exposure, this helps us understand if -- overarching, what is this relationship from the exposure to the outcome.

Q Okay. On page six of your report -- you can put that to the side for now.

MR. MICELI: Mr. Videographer, what time did we start this session?

MS. MIRSKY: 2:16.

THE VIDEOGRAPHER: 2:13 p.m.

MR. MICELI: 2:13? Thank you.

BY MS. MIRSKY:

Q On page six of your report --

A Uh-huh.

Q -- you state towards the top, The root of exposure for TCE includes inhalation, dermal exposure, and ingestion; correct?

A Yes.

Q And you cite to that number 30

"Trichlorethylene and Parkinson's Disease" by Fariha Zaheer and John Slevin; correct?

A Yes.

Q Okay.

MS. MIRSKY: And I'll mark this as Exhibit 22.

(Whereupon, Boehme's Exhibit 22, Study Titled "Trichlorethylene and Parkinson's Disease" by Fariha Zaheer and John Slevin, was marked for identification.)

MR. MICELI: This is -- you said we're at page six of her report?

MS. MIRSKY: Yes.

BY MS. MIRSKY:

Q Is Exhibit 22 that document?

A Yes.

Q Okay. If you could turn to page 659, which is the third page?

The -- under "Trichlorethylene Exposure," the last sentence of the first paragraph states, Among these solvents, trichlorethylene, or TCE, has gained interest recently as a possible risk factor for IPD, or idiopathic Parkinson's disease, among

1 those chronically exposed.
2 Is that correct?
3 A Yes.
4 Q What does "chronically" mean in this
5 study?
6 A I would have to go back and read the
7 study to determine what their use of "chronically"
8 means.
9 Q Okay. That's okay.
10 On page 660 in the second paragraph under
11 "human cases and epidemiologic studies," it says --
12 there's a reference to a 1999 Guehl, G-u-e-h-l,
13 study; correct?
14 A I'm looking for that.
15 Q Starts with --
16 A Oh.
17 Q -- in 1999?
18 A Yes.
19 Q Okay. And the article describes the
20 study as reporting, quote, a case of a 47-year-old
21 woman who was professionally exposed to TCE for
22 several months in an uncontrolled and unprotected
23 environment. She was a house cleaner and worked
24 with TCE in poorly ventilated rooms. Later she

1 worked in the plastics industry where she was
2 exposed to multiple solvents including TCE. She was
3 diagnosed with Parkinsonism in 1987 after
4 seven years of exposure.
5 Did I read that correctly?
6 A Yes.
7 Q And have you heard of the 1999 Guehl
8 study before?
9 A I have.
10 Q And does this comport with your
11 understanding of this study?
12 A I would have to go back and re-review,
13 but this seems an overarching overview of it.
14 Q Okay. And understanding the amount and
15 duration of exposure is important to analyze the
16 findings of this kind of study; would you agree?
17 MR. MICELI: Object to the form.
18 A This is a case study.
19 BY MS. MIRSKY:
20 Q Uh-huh.
21 A And so this sets the stage for additional
22 studies.
23 Q But my question was whether understanding
24 the amount and duration of exposure is important to

analyze the findings of this kind of case study?

MR. MICELI: Object to the form.

A I'd have go back and look at the case study to see, you know, how they discussed the exposure.

And, again, when we are setting up epidemiologic studies, we look at different definitions of exposures.

BY MS. MIRSKY:

Q Case studies are very limited in their scope of analysis; correct?

MR. MICELI: Object to the form.

A What do you mean by "scope of analysis"?

BY MS. MIRSKY:

Q Well, usually has an incredibly small population; correct?

A Correct.

Q And when you are focused on a small population such as one in this case study, which is one person, would you agree that the amount of and duration of exposure would be important to analyze any related findings?

MR. MICELI: Object to the form.

A Not necessarily because, again, this is

setting the stage for additional research. This is one person. We don't necessarily know what this means for a population level.

BY MS. MIRSKY:

Q So is it your opinion that the findings of this study would not demonstrate causation between TCE and Parkinson's?

MR. MICELI: Object to the form.

A So we generally do not build causation off of a case study. It's off of the entirety of what has been done.

And, you know, the entirety of what has been done includes case reports and case studies that are then built upon with epidemiologic studies, mechanistic studies.

You are not going to find someone who publishes a case study and then says yes, based on this one -- this one case study, this is the causal relationship.

We need to see the bigger picture.

BY MS. MIRSKY:

Q And so would you agree that the findings of this one study would not be sufficient to demonstrate causation between TCE and Parkinson's?

A It sets the stage and it sets, you know, some ideas around how one would want to conceptualize exposure in their follow-up studies.

And it definitely puts that exposure-disease relationship on the horizon of people who are evaluating these factors, especially people within PD risk factor analysis.

Q But my question was whether this study on its own is sufficient to demonstrate causation between TCE exposure and Parkinson's?

MR. MICELI: Same objection.

A It's because it's part of the entire body. When we are looking at causation, we are looking at the entire body of what is out there, not the specific studies.

BY MS. MIRSKY:

Q So this specific study on its own would not demonstrate causation between TCE and Parkinson's?

A It's a piece of the pie, but it's not the whole pie.

Q Okay. On page 662 of the Zaheer publication, the article states that, TCE exposure appears to be a risk factor based on recent

observations, associated chronic TCE exposure, and development of IPD or Parkinsonian features. Clearly, TCE exposure alone is insufficient as some individuals appear to have developed frank IPD while others with similar exposure have been asymptomatic or have presented with mild motor abnormalities that, although insufficient to make a diagnosis of IPD, include tremors or rigidity.

Did I read that correctly?

A Yes.

Q Would you agree with this publication that you cited that TCE exposure alone is insufficient to cause Parkinson's?

A So this publication was published in 2011 and we've had a lot of information come out into the field since then. And so the opinions and the relationships have been updated with the more information.

Q Would you agree -- and we talked -- we talked about this a bit before.

Would you agree that Parkinson's disease is a multifactorial disease?

MR. MICELI: Object to the form. Foundation.

A Yes. There are different causal pathways, not just a singular causal pathway.

BY MS. MIRSKY:

Q Okay. Back to your report on page six, you state -- and this is about -- this is in the first paragraph about a third of the way down -- Inhalation of TCE is particular -- is a particularly potent route of exposure that results in highly powerful dopaminergic neurodegeneration.

Did I read that correctly?

A Could you reread it?

Q Sure.

A I'm still trying to find it.

Q It's -- it's the sentence that starts with, "All exposure routes" --

A Oh, got it.

Q -- "pose health risks. However, inhalation of TCE is a particularly potent route of exposure that results in highly powerful dopaminergic neurodegeneration."

A Correct.

Q And do you agree with that sentence?

A So, again, I'm not a toxicologist. I -- you know, the route in which TCE works is not my

area of expertise.

Q So do you agree with that sentence as written in your report?

A That sentence is based off of the reference from others who have expertise in terms of exposure routes and health risks.

Q You cite number 31, which is Ashley Adamson and others' 2023 study titled "Low-dose inhalation exposure to trichlorethylene induces dopaminergic neurodegeneration in rodents"; correct?

A Yes.

Q Okay.

MS. MIRSKY: And that's Tab 21. We'll mark this as Exhibit 23.

(Whereupon, Boehme's Exhibit 23, Study Titled "Low-dose Inhalation Exposure to Trichlorethylene Induces Dopaminergic Neurodegeneration in Rodents" by Ashley Adamson and Others, was marked for identification.)

BY MS. MIRSKY:

Q Is this that publication?

A Yes.

Q Do you know whether Adamson -- Adamson 2023 tested mice and rats exposed to doses of TCE via inhalation?

A I would have to reread this publication.

Q Okay. I can represent to you that it did.

Are you aware of the calculations that Adamson 2023 used to extrapolate inhalation exposure concentration from rodents to animals -- excuse me, from rodents to humans?

MR. MICELI: Object to the form. Foundation.

A I see it in here, but I am not an expert in this.

BY MS. MIRSKY:

Q Would you be able to answer any questions about the Adamson 2023 study?

MR. MICELI: Object to the form.

A Do you mean in terms of --

BY MS. MIRSKY:

Q In terms of its findings?

A I am not an expert in this.

Q So you would not be able to answer any questions about it?

A Correct.
Q Would you be able to answer any questions about the methodologies used in Adamson 2023?
A That is outside of my area of expertise.
Q So you would not be able to answer any questions about that.
Would you be able to answer any questions about the calculations used in Adamson 2023?
A That's outside my area of expertise.
Q So you would not be able to --
A No.
Q -- answer any questions about that?
MR. MICELI: Object to form. Foundation.
A No.
BY MS. MIRSKY:
Q Okay. Thank you.
On fig- -- I'm not done quite yet.
On page 222 --
MR. MICELI: 222?
MS. MIRSKY: Yes.
MR. MICELI: This -- the copy that you've --
MS. MIRSKY: Oh, I'm sorry, 224.

1 MR. MICELI: Of --
2 MS. MIRSKY: That --
3 MR. MICELI: Actually, you have -- I
4 think we've used a different version of
5 this before because I have pages one
6 through 11 on this one.
7 MS. MIRSKY: On the one that I --
8 that you --
9 MR. MICELI: Ashley -- yeah,
10 "Low-dose inhalation exposure to
11 trichlorethylene induces dopaminergic
12 neurodegeneration" -- this is -- well,
13 you know what, let me do this -- you
14 just --
15 MS. MIRSKY: Here.
16 MS. PLATT: Yeah.
17 MS. MIRSKY: We can take Liz's copy.
18 MR. MICELI: Yeah, that's -- you
19 know, I actually have Liz's copy. The --
20 MS. MIRSKY: Okay.
21 MR. MICELI: I apologize. We've
22 used it in other depositions.
23 MS. MIRSKY: Oh.
24 MR. MICELI: But it's the same exact

one. But the printout -- this one actually has the cite to --

MS. MIRSKY: It's okay.

MR. MICELI: Okay.

MS. MIRSKY: Can you turn to page 224, please?

MR. MICELI: Yeah, I just want to make sure it's the same one that we're -- singing out of the same hymnal, so to speak.

Okay. Yeah. Okay.

MS. MIRSKY: Okay.

MR. MICELI: I apologize for the interruption.

MS. MIRSKY: That's okay.

BY MS. MIRSKY:

Q Okay. So on page 224 -- do you have page 224?

A I do have a page 224.

Q Great.

Figure D -- oh wait, I'm sorry. Let me start this over.

It is page 222 and it is Figure D on page 222. Okay.

Figure two, it says in the description below the -- the graphics that, The quantification of dopaminergic terminal loss from the striatum in Figure C and dopaminergic loss from the SNpc, which is the substantia nigra, correct, in D?

A This is not my area of expertise, so I don't know what SNpc stands for.

Q Okay.

A But what you read was correct.

Q Okay. Thank you.

Do you see in -- in D that the average dopaminergic neuron loss from the substantia nigra in the control group is about 23,000?

MR. MICELI: Object to the form. Foundation.

A This is not my area of expertise.

BY MS. MIRSKY:

Q Okay.

A This is -- no.

Q Then going back to your report --

A Okay.

Q -- on page six -- and you can put that to the side.

So the sentence that we started with is

that, Inhalation of TCE is a particularly potent route of exposure that results in highly powerful dopaminergic neurodegeneration; correct?

A Correct.

Q And the Adam -- Adamson study that we just looked at is footnote thirty- -- or citation 31; correct?

A Correct.

Q But you can't expound on the findings of the Adamson study; correct?

MR. MICELI: Object to form. Foundation.

A Correct.

BY MS. MIRSKY:

Q And that is the only citation that you have for that statement; correct?

A I also at the beginning of this statement referenced 27.

Q Okay. Okay. On page six of your report, you then state that, Oral consumption of TCE leads to toxic metabolites accumulating in the body resulting in dopamine neuron degeneration; correct?

A Yes.

Q And that is citation 32, which is to

"Trichlorethylene and Parkinson's Disease: Risk Assessment" by M. Liu and others; correct?

A Yes.

Q Okay.

MS. MIRSKY: And I will mark this as Exhibit 24.

(Whereupon, Boehme's Exhibit 24, Study Titled "Trichlorethylene and Parkinson's Disease: Risk Assessment" by M. Liu and Others, was marked for identification.)

BY MS. MIRSKY:

Q Is this that document?

A Yes.

Q Okay. In the abstract, about two-thirds of the way down, there's a sentence that says, Following eight months of TCE treatment there was a progressive and selective loss of 50 percent of dopaminergic neurons in mouse substantia nigra; correct?

A Correct.

Q And this is less than the 60 to 80 percent loss you testified was necessary to result in common PD symptoms; correct?

A So as a reminder, that reference is setting the stage for what I'm discussing. And my understanding is that reference comes from autopsy studies and not mouse models.

Q But previously you testified that you agreed that 60 to 80 percent of neuron loss is necessary before common PD symptoms manifest; correct?

MR. MICELI: Object to the form.

A I actually didn't state that. I agreed that what I had written was reflective of what was in the publications.

We don't -- you know, unfortunately when it comes to neuronal loss and Parkinson's, there's no way to assess that unless there's an autopsy. And, you know, neuron loss is not part of the diagnosis for Parkinson's either.

And so those studies are evaluating different extents of neuron loss in people who have agreed to an autopsy post mortem.

BY MS. MIRSKY:

Q Did you reference any of those caveats in your report when you stated that 60 to 80 percent neuron loss was necessary before motor function

issues would manifest with PD?

A So I did not reference any of the additional reports where that comes from.

Those -- those discussions are kind of around the layman's explanations as to what is happening and why we are seeing the motor changes.

Again, I'm not a neurologist, nor a toxicologist, so, you know, evaluating neuron loss is outside of my expertise.

Q Okay. But -- let me find the exact page.

A It's on page three.

Q Thank you.

Do you disagree with your -- with the statement in your report that the most common PD symptoms result from death or damage of neurons -- but to neurons in the substantia nigra, an area near the base of the brain, resulting from the loss of 60 to 80 percent of dopamine-producing cells in this area?

MR. MICELI: Object to the form.

A So, again, this statement comes from my references where I'm describing Parkinson's disease, Parkinson's symptoms, what we understand about Parkinson's to date.

I'm not a neurologist, so, you know, weighing in from an expert perspective on this is outside of my realm of expertise.

BY MS. MIRSKY:

Q So do you agree or disagree with that statement as written?

MR. MICELI: Object to the form.

A I mean, this -- this statement is consistent with what is published and what we see in textbooks and what is referenced in other publications around PD.

BY MS. MIRSKY:

Q Okay. So then turning back to the Liu study, which shows that there was a selective loss of 50 percent of the dopaminergic neurons in mouse substantia nigra following eight months of TCE treatment, do you agree that that is less than the 60 to 80 percent loss you wrote in your report was necessary to result in common PD symptoms?

MR. MICELI: Object to the form.

A So, again, what I was discussing in -- in that specific sentence in my report is referring to humans.

This is one study on mouse models, so

it's not exactly comparable.
BY MS. MIRSKY:
Q The last sentence of the abstract states, Even prolonged administration of TCE was insufficient for producing a greater than 50 percent loss of nigral dopamine neurons indicating that additional comorbid factors would need -- would be needed for mimicking the profound loss of dopamine neurons seen in Parkinson's disease; correct?
A Correct.
MR. MICELI: Object to the form.
BY MS. MIRSKY:
Q And does that also fail to hit the 60 to 80 percent loss that you wrote about in your report?
A I --
MR. MICELI: Object to --
THE WITNESS: Oh, sorry.
MR. MICELI: Object to the form.
A Again, what I was writing about was in reference to human studies.
This is a single study that is on mice. This particular study is outside my realm of expertise. So, you know, critiquing the study is outside of my expertise.

But comparing mice to humans in terms of neuronal loss is not exactly appropriate.

BY MS. MIRSKY:

Q Okay. But you did cite this study in your report; correct?

A I did, yes.

Q And so does that indicate that you believe that it is a -- a worthwhile study to consider in the context of your report?

MR. MICELI: Object to the form.

A So this study is helping, again, to set the stage for understanding of consumption routes, mechanisms, what could be happening so that we have a better understanding when evaluating the epidemiologic studies.

BY MS. MIRSKY:

Q Okay. Going back to your report again, this is on page six now -- and you can put that study to the side -- you state that -- right after the sentence I just read, which had citation 32 -- Because PCE is more lipophilic than TCE, it has greater bioaccumulation resulting in greater toxic accumulation.

Do you see that?

A Yes.
Q Did I read that correctly?
A Yes.
Q Are you -- you have citations to 33 to 35 there.
Are you aware that none of the sources you cite -- you cite in support of this statement reference TCE specifically?
A I would have to go back and look at those references.
Q Do you know whether any of the sources you cite in support of that statement reference PCE specifically?
A I'd have to go back and look at them.
Q Are you able to offer any independent opinions about the lipophilic nature of PCE as compared to TCE?
A I'm not an expert in that area, so no.
Q Okay. In the next paragraph you state that, TCE as a cause of PD was first described in a 1999 case report describing the case of a woman who developed PD after seven years of TCE exposure.
And this is in reference to the Guehl study that we discussed earlier; correct?

A Correct.

MS. MIRSKY: Tab 23, please.

And I'll mark this as Exhibit 25.

(Whereupon, Boehme's Exhibit 25, Study Titled "Trichloroethylene and Parkinsonism: A Human and Experimental Observation" by Guehl and Others, was marked for identification.)

MR. MICELI: Thanks.

BY MS. MIRSKY:

Q Is this --

MR. MICELI: Is this -- have we already gone over this one?

MS. MIRSKY: We discussed it --

MR. MICELI: Okay.

MS. MIRSKY: Broadly.

MR. MICELI: Okay. I didn't know if you'd marked it already.

MS. MIRSKY: No.

MR. MICELI: Okay. Thank you.

BY MS. MIRSKY:

Q But this is that study that we --

A Yes.

Q -- discussed earlier; correct?
Okay. Can you please turn to page 610?
So the first -- first paragraph says, The patient was first professionally exposed to TCE for several months in an uncontrolled and unprotected environment when she was a house cleaner. The principle solvent was TCE and the small rooms, 30 met- cubed meters, were poorly ventilated. After this intensive exposure she worked for six years, between 1978 and 1984, in the plastics industry in a very small 20 cubed meters, an unventilated office which was exposed to the diffusion of many volatile components, particularly trichlorethylene.
Did I read that correctly?
A Yes.
Q So given the conversation we had before and the specifics that you see here involved in this study, this was a case study; correct?
A Correct.
Q And therefore there was no comparison group in this study; correct?
A There's only a single person.
Q Does that weaken the impact of the study?

MR. MICELI: Object to the form.
A What do you mean?
BY MS. MIRSKY:
Q Would you consider case studies to have a more limited impact than other types of epidemiological studies?
A Again, it depends.
Q In --
A It --
Q Sorry.
A It depends on, you know, what kind of evidence is out there and the rarity of the exposure or the outcome.
Q In this specific context, would you --
A In the --
Q -- consider --
A In this context, this study really set the stage for the future epidemiologic studies.
Q But looking at this study on its own merits, would you agree that the fact that it's a case study is a limitation of the impact that its findings could have?
A So in terms of a case study, the findings are relevant in terms of a case study. And they're,

again, important to set the stage for future epidemiologic studies and, you know, we build off of this.

We then look to see, okay, in the future epidemiologic studies, is what is being described in this study consistent.

Q So then this case study standing on its own would have less of an impact than the other studies that you're referencing it set the stage for?

MR. MICELI: Object to form.

A Again, when we are looking at causality, again, we are looking at the body of the literature and not just a specific study. And so it's this within the context of everything else.

And, again, this set the stage for the future studies to evaluate different solvents on PD risk.

BY MS. MIRSKY:

Q Would you give this study the same weight as a broader epidemiological cohort study?

A Could you clarify the question?

Q When evaluating the totality of the evidence --

A Uh-huh.
Q -- as you were discussing, would you give this case study the same weight as a cohort study?
A I think it's important to set the stage as to why the cohort studies are being done. It's important to understand the historical perspective of, you know, how these evaluations came to be.
Q But my question was whether they would have the same weight in your analysis of the evidence overall?
A So in looking at the evidence overall, we are going to place more of, you know, the measures of association where we can look at people's counterfactual experience -- or estimate people's counterfactual experience and that's what really helps to illuminate whether or not something could be considered on the causal pathway.
Q Okay. And so for this study, how does that fit into your overall analysis?
A Again, this study sets the stage for why the cohort studies were done and it sets the stage for how hypotheses were generated to conduct the epidemiologic studies.
Q Okay. There was no control for

confounding variables in this study; correct?

A It's a single person so there's no -- nothing.

Q Is that a limitation of the study?

A Well, it's a case study and that's just an inherent aspect of a case study. You are describing something that you've seen and setting the stage for future explorations.

Q Would you agree that the lack of confounding variables in case studies is an inherent limitation in case studies?

A Well, case studies by design don't consider confounding variables because they are just presenting a unique case. They are not evaluating an exposure on an outcome relationship. They are presenting, Hey, this is something interesting that we are seeing; more research needs to be done.

Q Okay. Case studies cannot control for temporality; correct?

A In this --

MR. MICELI: Object to -- excuse me. Object to the form.

A In this case we have temporality. So we understand when the exposure happened and we

1 understand when PD diagnosis occurred.
2 BY MS. MIRSKY:
3 Q But generally speaking, case studies
4 can't control for whether the exposure happens
5 before or after a diagnosis; correct?
6 MR. MICELI: Object to the form.
7 A So there's no controlling for
8 temporality; there either is or there isn't.
9 And actually, most of the time in case
10 reports and case studies -- depending on the
11 exposure, of course -- there's a description of when
12 the exposure occurred and when the outcome occurred,
13 and so the temporality is set.
14 BY MS. MIRSKY:
15 Q We previously talked about whether it's
16 important to understand the extent and duration of
17 exposure when analyzing a study's results; correct?
18 A Yes.
19 Q And do you believe that the extent and
20 duration of exposure in the Guehl study is important
21 to analyzing its findings?
22 A Again, I believe it sets the stage for
23 future studies in how they're going to define their
24 exposure, including duration.

Q My question was whether understanding the extent and duration of exposure in this study is important context for analyzing its findings?

A Well, it presents what's happened and so understanding the exposure, understanding what has happened then leads to the question, you know, do we see this in a population and do we see this with different exposure definitions and different exposure durations.

Q When you discuss this report in your -- excuse me.

When you discuss this study in your report, you say that TCE as a cause of PD was first described in a 1999 case study -- case report describing the case of a woman who developed PD after seven years of TCE exposure; correct?

A Yes.

Q But you do not talk about the extent of that exposure in your report; correct?

A Correct.

Q And you do not discuss the duration or sort of the -- the nature of the exposure in your report; correct?

A Correct.

Q Okay. You then state that, The authors expanded on this case by evaluating TCE exposure on neuronal death in mice findings that mice exposed to TCE presented significant dopaminergic neuronal death compared to the control group; correct?

A Yes.

Q Are you able to answer any questions on this part of the Guehl study?

MR. MICELI: Object to the form.

A This is not my area of expertise.

BY MS. MIRSKY:

Q So you would not be able to answer any questions on that?

A Correct.

MR. MICELI: Object to the form.

BY MS. MIRSKY:

Q On page 610, which we were just looking at in the Guehl study, it states at the top of the second column, The trichlorethylene-treated mice presented no Parkinsonian motor abnormalities as it is generally reported after MPTP treatment; correct?

A Yes.

Q And are you able to provide any analysis of this sentence?

A This is outside of my expertise.
Q And so you're not able to provide any analysis of this sentence?
A Correct.
Q Okay. In the discussion section at the top of the -- or the first sentence in the second paragraph, it says, The nigral degeneration that trichlorethylene produces in mice was 50 percent; correct?
A Correct.
Q And, again, this is less than the 60 to 80 percent you testified to previously; correct?
A So, again, the 60 to 80 percent is referencing the human studies and not mouse studies.
Q Okay. In the -- going back to your report, in the next paragraph, you discuss a 2012 study evaluating the risk of PD in twins -- excuse me.
That's referring to "Solvent Exposures and Parkinson's Disease Risk in Twins" by Goldman; correct?
A Let me check --
Q That's citation 43.
A Correct.

Q Okay.
MS. MIRSKY: Twenty-four.
THE WITNESS: Oh, sorry.
MR. MICELI: Quite all right.
Can I take just one second? We don't need to go off the record. I'm just going to go over there and get some paper clips.
MS. MIRSKY: Okay.
BY MS. MIRSKY:
Q This is the Goldman 2012 --
MS. MIRSKY: Oh, I'm sorry. This is -- I'm marking in as Exhibit 26.
(Whereupon, Boehme's Exhibit 26, Study Titled "Solvent Exposures and Parkinson's Disease Risk in Twins" by Goldman, was marked for identification.)
BY MS. MIRSKY:
Q That is the Goldman 2012 article -- study; correct?
A Yes.
Q And the sample for this study included 99 pairs of twins; correct?

A Correct.
Q The study subjects were World War II veterans; correct?
A Yes.
Q And the study relied on self-reporting of these veterans; correct?
A Yes.
Q If the informants were unavailable, then there was proxy reporting; correct?
A Yes.
Q And on page 12 at the very bottom -- oh, no -- sorry, yes, at the very bottom there's a chart that says, Characteristics and pairs completing interview, 46 percent of the PD-affected twins responded by proxy; correct?
A Correct.
Q And 18 percent of the unaffected twin responded by proxy; correct?
A Correct.
Q Solvent exposures were inferred in this study based on occupational and hobby history; correct?
A Correct.
Q And for deceased and cognitively impaired

subjects, a proxy informant would have provided the occupational history and hobby history; correct?

A Correct.

Q The authors did not have exposure samples for the population; correct?

A Correct.

Q Is inferring exposure based on job and hobby histories collected through self- or proxy-reported histories a way that exposure misclassification could occur?

MR. MICELI: Object to the form.

A So there's two questions in there it feels like.

BY MS. MIRSKY:

Q Okay. Is inferring exposure based on job and hobby history a way to lead to exposure misclassification?

MR. MICELI: Object to the form.

A So inherently any time we measure an exposure, there's risk of exposure misclassification.

BY MS. MIRSKY:

Q Okay. And does inferring exposure based on job and hobby history increase the possibility

for exposure misclassification?

MR. MICELI: Same objection.

A Not necessarily, because, you know, you are using a proxy -- you're using a proxy essentially, occupation for exposure.

Recognizing that that's not quantifying the direct exposure, you know, will inform additional studies that more work needs to be done outside of using occupation as a proxy for TCE exposure.

BY MS. MIRSKY:

Q Within the context of this study, does inferring exposure based on job and history indicate that there could be exposure misclassification?

MR. MICELI: Object to the form.

A So, again, there could be exposure misclassification any way that we are measuring exposure. So whether it's self-report, whether it's a biomarker, you know, this is an issue we run into with epidemiologic studies.

And generally speaking, as long as the misclassification is non-differential between those with PD and those without PD, if anything, it would be likely to lead our results closer to the null

than anything.
BY MS. MIRSKY:
Q In this study, did inferring exposure based on job and hobby histories lead to exposure misclassification?
MR. MICELI: Object to the form.
A There is no way to accurately measure whether there was exposure misclassification; right.
So they're using occupation. And, again, every epidemiologic study likely has exposure misclassification.
You know, running joke is when -- in terms of misclassification, the only thing we are really good at identifying is death; everything else is likely going to have misclassification. And that's no different here than any other epidemiologic study.
BY MS. MIRSKY:
Q The fact that the job and hobby histories were collected through self- or proxy-reported histories, would that lead to a higher likelihood of increased exposure misclassification?
MR. MICELI: Object to the form.
A Not necessarily. They use structured

survey questionnaires and occupation is not something that is often misclassified. It's not stigmatizing. It's something most people remember.

BY MS. MIRSKY:

Q Okay. Data on head injury was also provided through interviews; correct?

A Correct.

Q And for deceased and cognitively impaired subjects, a proxy informant provided data on head injury; correct?

A Correct.

Q Head injury was almost three times as great in the twins with PD than those without; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:

Q And as we discussed earlier, head injury is a risk factor for PD; correct?

A Severe head injury is a risk factor for PD.

Q Twins do not have identical exposures, particularly in adulthood; correct?

A Depends. We don't -- it depends; right?

It depends on how the twins live, it depends on where they live.

We do know that with twin studies, you know, this accounts for a lot of the early life factors and adolescent factors. There is some evidence that twins and even triplets have very similar hobbies, approaches, places of living, etc.

Q But twins may live in different residential locations; correct?

A It's possible, yes.

Q And twins may have different exposures over the course of their lifetime; correct?

MR. MICELI: Object to the form.

A It's possible, yes.

BY MS. MIRSKY:

Q Goldman 2012 did not access other exposure information aside from what was reported; correct?

A Correct.

Q And the authors did not have information on residential history for each twin; correct?

A Correct.

Q And adult residential history could have impacted their overall exposures; correct?

1 MR. MICELI: Object to the form.
2 A So not necessarily. It's -- again, it's
3 dependent; right.
4 So we have our exposed group, we have our
5 unexposed group. The unexposed group through other
6 occupations likely has similar residential
7 experiences to the exposed group.
8 You know, again, more studies would need
9 to be done to, like, tease that out, and more
10 studies were done after 2012 to start evaluating
11 this more explicitly.
12 But, again, the -- the idea is that the
13 unexposed group shows what the association with PD
14 would be had the exposed group not been exposed.
15 BY MS. MIRSKY:
16 Q What is your definition of exposure?
17 A So, again, it depends.
18 Generally speaking, like in this case,
19 they used employment history to define their
20 exposure of interest.
21 Exposure is generally in most
22 epidemiology studies the factor that we're
23 interested in exploring. The factor that we are
24 trying to determine is on a causal pathway with our

outcome. It could be causal a protective factor, it could be a risk factor.

Whether it's a continuous variable, categorical, dichotomous really depends on the nature of the exposure of interest. How we define it is going to depend on the nature of the exposure of interest.

But, again, it's -- it's the characteristic or the variable that we are seeking to determine whether that characteristic or variable is implicated in the cause of a disease.

Q Is the route of exposure important to analyze?

A It depends.

Q In the context of this study, would the route of exposure be important?

A So since this study is using proxy by -- through occupation, route of exposure would not be of interest because you're already proxying exposure.

Q When would route of exposure be important?

MR. MICELI: Object to the form.

A It depends; right? You know, because TCE

can be inhaled, ingested, or through skin, you know, that's where some of the case series help us understand the route of exposure, that's where some of the mechanism models help us understand, you know, where should we be focusing. It depends.

BY MS. MIRSKY:

Q Okay. So in Goldman 2012, ultimately the authors inferred exposure information for each twin; correct?

A Correct.

Q And this study found a relative risk ratio for TCE to be 6.1 with a 95 percent confidence interval of 1.2 to 33; correct?

A Hold on. I'm looking for it. It is --

Q On the --

MR. MICELI: Page 14.

BY MS. MIRSKY:

Q -- first page.

And the first page in the results section?

A I was just going to go to the table, page 14.

Yes.

Q You are on page 14?

MR. MICELI: I'm sorry, I didn't mean to --

MS. MIRSKY: That's okay.

MR. MICELI: -- interrupt you.

BY MS. MIRSKY:

Q 1.2 to 33 is a wide confidence interval; correct?

A So in terms of evaluating confidence intervals, this is all relative. And we recognize that this is a smaller sample size.

You know, we still have an extremely strong effect here between TCE and Parkinson's and, you know, compared to some other confidence intervals I've seen before, it's not the widest I've ever seen -- I've seen significantly wider -- but it's not the narrowest.

Q Do you think that 1.2 to 33 indicates that the finding is imprecise?

A I would not say that. I would say that, you know, we see a very strong magnitude of effect here. Our confidence interval is basically telling us if we resampled and redid this study that our true parameter estimate would land somewhere in that range at some point.

And, again, it's a range. It could be that it's much higher. It could be that it's much lower.

Regardless, that's a very strong magnitude of effect and the point estimates within the confidence interval are strong as well.

Q But you don't know if the study were to be rerun whether it would be the same or higher?

MR. MICELI: Object to the form.

A We would have to see when it's rerun.

BY MS. MIRSKY:

Q But you don't know whether that would be the case; correct?

MR. MICELI: Same objection.

A Not necessarily, yeah.

BY MS. MIRSKY:

Q Okay. Are you familiar with confidence interval ratios?

A What do you mean by confidence interval ratios?

Q Have you heard of a confidence interval ratio before?

A I've heard of confidence interval ratios and I've heard of them used a couple different ways

and so that's why I was asking for clarification.

Q Have you ever used a confidence interval ratio to measure the precision of results?

A I have personally never used a confidence interval ratio.

Q What are the ways in which you have seen confidence interval used?

A I'd have to go back and look. I mean, sometimes they are used in publications. They are not frequently used.

Q Is there a reason why they are not frequently used?

A Because generally, again, when we are reading and evaluating a publication, we are thinking about the entirety of the publication.

You know, in this case, this has small sample sizes in terms of people who were exposed. We've got really small numbers. And so it's expected that you would see a confidence interval that's not particularly narrow because of small sample sizes.

The -- the precision within a confidence interval is driven by sample size. You also want to interpret it within the context of the limitations,

the context of the study design, the timeframe.

You know, this is one of the first epidemiologic studies really starting to explore these relationships and by using a twin study, that really helps account for a lot of the early lifestyle factors and adolescent factors that could contribute to Parkinson's.

And so that's generally why you don't see confidence ratios.

Q What is the reason why you would use a confidence interval ratio?

A Well, it depends on what definition you are using of confidence interval ratio. Like there's -- I've seen a couple different definitions floated around. I don't know all of them off the top of my head.

But in reviewing papers for journals, especially for environmental health journals, I've seen different uses. But each time they explain what they're using it for.

Q And do you believe that it's an appropriate way to analyze a confidence interval?

A We don't generally analyze confidence intervals.

Q Is an acceptable way to interpret the precision of a confidence interval?

MR. MICELI: Object to -- excuse me. Object to the form.

A Not necessarily, because precision is more than just the confidence interval. It's about the magnitude of effect, it's about decreasing bias. Again, you know, it's the entirety of the study, not just one piece.

You know, we see more focus on bias and magnitude of association than confidence intervals and discussion of precision around confidence intervals.

And all of this really builds into why the Bradford Hill consideration really leans on what we see broadly to determine causality. You know, single studies in and of themselves, we need more than just a single study. We need to see this replicated over and over and over again.

And we are going to see varying effect estimates, we are going see varying confidence intervals, we are going to see studies that are statistically significant and studies that are not statistically significant.

But we're looking at the overarching picture and looking for the theme, so to speak. Do we see that it's consistently a risk factor or do we see something different.

And, I mean, if we think about relationships that are definitively causal, that we discuss at length -- smoking and lung cancer, for instance -- not a single cancer epidemiologist that I know can pinpoint to specific magnitude of effect or confidence interval. It's about the body of work between smoking and lung cancer that really drove home this is a causal relationship.

And you would see a wide away of magnitude of effect and a wide array of confidence interval; some that are wide, some that are narrow.

BY MS. MIRSKY:

Q Is a small sample size the only reason that you would have a wide confidence interval?

A It's going to be a big reason.

Q But are there other reasons?

A The magnitude of effect will play a role, too.

Q In what way?

A So when you're calculating your

confidence interval, it incorporates your sample size, your magnitude of effect in determining your upper bound and lower bound.

Q Okay. With the 6.1 relative risk ratio that we were just discussing from Goldman 2012, that was in Goldman's attempt to consider TCE exposure on its own with this data; correct?

MR. MICELI: Object to the form.

I think you might have misstated something. I don't --

MS. MIRSKY: Did I?

MR. MICELI: -- need to correct you, but you said risk ratio -- I'm sorry --

MS. MIRSKY: She said odds ratio.

MR. MICELI: Okay.

MS. MIRSKY: Excuse me.

A Well, you were calling it relative risk.

BY MS. MIRSKY:

Q Oh.

A And an odds ratio is a relative risk.

Q Okay.

A So it's fine.

Q So is that - is it correct that that was his -- that's what the study's interpretation was of

TCE -- TCE exposure on its own?

A So this is not controlling for PCE exposure; right. So it is not TCE by itself, is my understanding.

Because if we look at TCE or PERC, we see that the categories for PERC and TCE are not mutually exclusive and exhaustive.

Q But TCE is listed by itself in this chart; correct?

A It's listed by itself.

Q And the association and confidence interval that we were just discussing is what is listed for TCE on its own; correct?

A For TCE by itself, yes.

Q Okay. And Goldman found that the ratio for PCE is 10.5 with a 95 percent confidence interval of 0.97 to 113; correct?

A Yes.

Q When the lower end of the confidence interval is less than one, the increased risk is not statistically significant; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:

Q And so this finding is not statistically significant; correct?

A It is not statistically significant, but it is extremely relevant from a public health perspective.

Q 0.97 to 113 is a wide confidence interval; correct?

A Again, how wide a confidence interval is, is relative.

Q But you don't know whether if there were more people added to this study, whether the confidence interval would become more narrow; correct?

A It is likely it would. One of the problems with this is that there is a zero in one of the cells which really causes problems when we are running these models.

And because there are zero pairs that had PERC for the case positive, control positive, you know, it really distorts our findings in that regard.

Q And my question was whether you know if additional individuals were added to this study,

1 whether the confidence interval would narrow?
2 A It's very likely it would narrow.
3 Q But you don't know?
4 MR. MICELI: Object to the form.
5 A It's very likely it would narrow.
6 BY MS. MIRSKY:
7 Q Okay. And this is the study's attempt to
8 consider PCE exposure on its own; correct?
9 A Correct.
10 Q The sample in this study included ten
11 cases of individuals exposed to TCE; correct?
12 A I have to look.
13 Where are you seeing that?
14 Q Oh, sorry. Nine, in the chart?
15 A So in the chart, that's nine pairs. So
16 that's where the case is positive and the control is
17 negative.
18 Q So then it would only be half that for --
19 I'm sorry, so it would be 18 people that --
20 A For -- who -- so 18 people who are case
21 positive, control negative were exposed to TCE; four
22 people who part of the case negative, control
23 positive were exposed to TCE. One case positive,
24 control positive pair, so two people were exposed to

TCE; and 87 case negative, control negative pairs were exposed to TCE.

Q Okay. And for PCE, it would be five pairs case positive, control negative; one pair case negative, control positive and --

A Zero pairs --

Q -- zero case positive --

A -- uh-huh.

Q -- control positive; correct?

A Correct.

Q Do you believe that the small sample size in this study is a limitation?

A It's an inherent aspect of PD studies and a rare exposure.

Q But do you -- so do you believe that the small sample size in this study is a limitation of the study?

MR. MICELI: Object to the form.

A I mean, we have about 200 people in the study total, 99 pairs. Again, it's a rare exposure. If we had more people, that would be really helpful.

But there's a lot of information that is gleaned from this study.

BY MS. MIRSKY:

Q Okay. Can you turn to page six, please, of the study?

At the top of the page it says, The major limitations of the study are its small sample size which yielded imprecise risk estimates and exposure inferences based on retrospective recall; correct?

A That's what it says, yes.

Q So do you agree that the small sample size was a limitation of the study?

MR. MICELI: Object to the form.

A It's always nice when we have bigger sample sizes. If we had had larger sample sizes for this study, you would have seen more precision around the confidence intervals.

However, the results of the study are still important in and of itself.

BY MS. MIRSKY:

Q But do you agree with the authors of this study that a major limitation is its small sample size?

MR. MICELI: Object to form.

A Even though it's a small sample size, the results are still important.

BY MS. MIRSKY:
Q But it is a limitation?
MR. MICELI: Object to the form.
A Yes. But, again, with our limitations we interpret our results within the context of the limitations and recognize that having a larger sample size is always something that we want but may not be possible.
BY MS. MIRSKY:
Q And you don't know whether the odds ratio for PCE would be statistically significant if the sample size were larger; correct?
MR. MICELI: Object to the form.
A Based on the magnitude of effect, it is very likely it would be.
BY MS. MIRSKY:
Q You can't predict how the odds ratio would change if the sample size were larger; correct?
MR. MICELI: Object to the form.
A To an extent. But that large of an effect is likely going to be significant with a larger sample size.

BY MS. MIRSKY:
Q Is it plausible that the odds ratio could decrease with a larger sample size?
MR. MICELI: Object to the form.
A There are always things that are possible.
However, it is likely that the effect estimate would remain a risk factor and would likely be statistically significant.
BY MS. MIRSKY:
Q But it is possible that the odds ratio could decrease with a larger sample size; correct?
MR. MICELI: Object to the form.
A Even if it decreases, it's still a significant risk.
BY MS. MIRSKY:
Q Is it --
(Whereupon, there was an interruption.)
(Whereupon, there was a discussion off the record.)
BY MS. MIRSKY:
Q Is it possible that because of the small sample size an underlying bias in the sample

population could be magnified in the resulting finding?

MR. MICELI: Object to the form.

A What do you mean by "underlying bias"?

BY MS. MIRSKY:

Q If there is a bias in the underlying sample population, if the underlying sample population is small, would the impact of any bias be magnified in the results?

MR. MICELI: Object to the form.

A What type of bias?

BY MS. MIRSKY:

Q For -- for any type of bias?

A Well, it would depend.

Q Okay. Is -- can you think of a type of bias in which that would be the case?

MR. MICELI: Object to the form.

A Likely, any type of bias is driving our results closer to the null. That's what we see more often than not in terms of, you know, exploring the extent of bias and how that shapes our magnitude of effect.

It would be very dependent on the type of bias and how that bias is creeping in, so to speak.

BY MS. MIRSKY:
Q Okay. Does the table that we were looking at in Goldman 2012, table three, does that --
(Whereupon, the court reporter requests clarification.)
MS. MIRSKY: Yes.
BY MS. MIRSKY:
Q -- does that indicate whether there was any adjustment for head injury?
A It did not adjust for head injury.
Q Okay. On page six of your report, you state that Goldman 2012 -- this is at the bottom in the last paragraph -- found that any exposure of TCE as evaluated through occupational exposure assessment methods was associated with a 500 percent increased risk of PD and was statistically significant; correct?
A Correct.
Q How do you define any exposure in this sentence?
A So what they defined as any TCE exposure.
Q Okay. Would a -- would exposure to a single molecule of TCE constitute exposure in this

1 context?
2 MR. MICELI: Object to the form.
3 A So in terms of TCE exposure, they are
4 using the occupation as a proxy and it's not a
5 single molecule. I don't know a single person who
6 would be exposed to a single molecule of anything.
7 And so generally it's more than a single
8 molecule. But because of occupational proxy, it's
9 not able to be estimated.
10 BY MS. MIRSKY:
11 Q So the study doesn't state what a minimum
12 exposure to TCE for increased risk to PD would be;
13 correct?
14 A Correct.
15 Q And the study doesn't characterize the
16 purported increased risk as 500 percent increased
17 risk; correct?
18 A But that's an easy statement. So when
19 we're -- when we're talking about odds ratios, risk
20 ratios, hazard ratios, we can present and say the
21 study had a 6.1 increased odds.
22 When we turn it to percent, we do one
23 minus -- or the absolute value of one minus a
24 relative risk measure, whether that's odds ratio,

hazard ratio, risk ratio, and we can present it as 500 percent in that case.

So the -- me saying it was associated with a 500 percent increased risk of PD is the exact same as me saying was associated with a 6.1 increased odds ratio of PD.

Q So that's how you did the calculation, you subtracted one and then you multiplied it by a hundred?

A Correct.

Q Okay. And is the 500 percent increased risk the same for everyone included in the Goldman study?

MR. MICELI: Object to the form.

A So epidemiology is based on population; right? And so this is looking at the population.

We don't then take our population and put it on an individual.

BY MS. MIRSKY:

Q Okay. But the -- because the individuals in this study were just relating their general exposure history by proxy; correct?

MR. MICELI: Object to the form.

A No. So what it is, is it's because we

are looking at an exposed group compared to an unexposed group.

So our exposed group is at 500 percent increased risk of PD. But we're looking at the group. We're not looking at individuals. And that's compared to our unexposed.

BY MS. MIRSKY:

Q Okay. You then state in your report that the risk of PD after PCE exposure was even stronger with a 950 percent increased risk of PD after PCE exposure, but not statistically significant; correct?

A Correct.

Q And did you calculate this in the same manner?

A Exactly.

Q And your opinion is that this study demonstrates that there's a 950 percent increased risk of PD after PCE exposure, even though this finding was not statistically significant; correct?

A That magnitude of effect is very large so -- and it's, again, driven by the small sample size and the fact that we've got a zero in our case positive, control positive group.

So it is very likely that with increased sample size, we would see a statistically significant result and still see an increased magnitude of effect.

Q And, again, this study doesn't state what a minimum exposure to PCE would be to have --

MR. MICELI: Object to form.

BY MS. MIRSKY:

Q -- an increased risk; correct?

A Correct.

Q You then state that in evaluating the combined risk of TCE and PCE (labeled as PERC in their study) they found a 790 percent increased risk of PD after exposure to TCE or PCE; correct?

A Correct.

Q And did you calculate this number in the same manner?

A The exact same manner.

Q And the study doesn't state what a minimum exposure to TCE and PCE would be for an increased risk of PD; correct?

A Correct.

Q You include footnote one here?

A Uh-huh.

Q Correct?

The footnote states that, The authors noted their combined category of TCE or PERC was included, quote, because the compounds are thought to share toxic properties, end quote; correct?

A Correct.

Q And this is quoting from Goldman 2012; correct?

A Correct.

Q And so the citations that follow are the citations that were in Goldman 2012; correct?

A Correct.

Q Did you review these studies?

A I glanced at them but not in depth because that is outside of my area of expertise.

Q So you did not come to any independent opinions about them; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:

Q And you did not assess whether they support the statement for which they are cited in Goldman 2012; correct?

A Correct.

Q And are you able to independently analyze any of the findings in these cited studies?
A You mean, specifically the Bringmann study?
Q The --
A Or the Lash?
Q The -- the -- yeah. The Bringmann study, the Lash study --
A Oh, the Lash. Right.
Q -- or the other Lash study.
A That's outside of my expertise.
Q So you couldn't answer any questions about those three studies?
A Correct.
Q Okay. On page eight of your report, you state at the top that, The combined category of TCE and PCE statistically significant finding is telling in that by combining TCE and PCE, the authors were able to increase the sample size sufficiently to result in findings that are statistically significant in combination with a strong measure of effect; correct?
A Correct.
Q And this further supports or in fact

demonstrates that the number of people in the study, study power or lack thereof, was driving whether the relationship between PCE and PD is demonstrated with statistical significance or not when PCE was evaluated on its own; correct?

A Correct.

Q What is the number of -- what is the difference between the PCE-only category and the TCE and PCE combined category?

A What do you mean?

Q How many people -- like, what is the number -- what is the difference in the number of people between the two?

A Like the number of people who are in PERC only versus the TCE or PERC?

Q Yes.

A I would have to calculate that. I don't know it off the top of my head.

But, you know, just reminder that the PERC category and the TCE category are not mutually exclusive and exhaustive, and so some of the people in PERC also have TCE exposure.

Q Okay. Do you agree that the proxy -- the fact that proxies responded for a majority of the

PD-affected sample is a limitation of the study?
MR. MICELI: Object to the form.
A I don't. Use of proxy is a standard way of evaluating an exposure or an outcome in epidemiologic studies and occupation is not a stigmatizing aspect. It's something that people would understand and would know.
You know, so in terms of using a proxy, it should be fairly reliable.
BY MS. MIRSKY:
Q Do you agree that the difficulty in isolating specific effects of single agents because many work settings involve exposure to multiple agents is a limitation of the study?
A Could you repeat that?
BY MS. MIRSKY:
Q Do you agree that the difficulty isolating specific effects of single agents because many work settings involve exposure to multiple agents is a limitation of the study?
MR. MICELI: Object to the form.
A Yes. So using occupation as a proxy for exposure is a bit of a limitation. But, again, if anything, it's probably underestimating exposure.

BY MS. MIRSKY:

Q On page eight of your report you discuss the study "Solvent exposed occupations and risk of Parkinson's Disease in Finland" by Susan Nielsen and others; correct?

A Let me double-check. Twenty-seven... Correct.

Q Okay.

MS. MIRSKY: Mark this as Exhibit 27.

(Whereupon, Boehme's Exhibit 27, Study Titled "Solvent Exposed Occupations and Risk of Parkinson's Disease in Finland" by Susan Nielsen and Others, was marked for identification.)

MR. MICELI: Just out of curiosity, we've been going about an hour and ten minutes since we started.

Do you want to take a break or do you want to go through this one?

MS. MIRSKY: I'm fine either way.

Do you want to take a break? I'm happy to if you'd like to.

THE WITNESS: Let's go through this one. I'll get through my water and then I'll refill.

MS. MIRSKY: Okay. That's fine.

BY MS. MIRSKY:

Q So this is the Nielsen study; correct?

A Yes.

Q On page one of the report --

A The report or the publication?

Q Sorry, the publication -- or -- yes, the publication -- one second.

-- it states that there were 11,757 cases and 23,236 controls with an occupation in the 1990 census corresponding to age 40 to 60; correct?

A Yes.

Q And they focused on 28 occupations with a greater than or equal to 5 percent probability -- probability of solvent exposure according to the Finnish Job-Exposure Matrix; correct?

A Correct.

Q And on page five of the publication in the table, the odds ratio for TCE is 5.92 with a confidence interval of 0.56 to 62.2; correct?

MR. MICELI: Where -- I'm sorry, you

are on page five?

MS. MIRSKY: Yes.

MR. MICELI: And you are referring to which one? I apologize.

MS. MIRSKY: TCE. Right at the top.

MR. MICELI: Oh.

BY MS. MIRSKY:

Q Is 5.92 with a confidence interval of 0.56 to 62.2 correct?

A Correct.

Q And for PCE, the odds ratio is 1.67 with a confidence interval of 0.09 to 30.8; correct?

A Correct.

Q Neither of these are statistically significant; correct?

A Correct.

Q And would you agree that these confidence intervals are wide?

A Again, that's all relative. You know, we see a very wide confidence interval for methylene chloride. We see a very strong effect, though.

Q But the confidence intervals are wide in these findings; correct?

A Again, it's relative and it's likely

driven by the small numbers of people with trichlorethylene exposure, etc.

Q And on page eight of your report, you state that, Occupations with a potential exposure to chlorinated hydrocarbon solvents, including TCE, had an increased risk of PD. The measures of association found in this study are likely a conservative estimate of the association between solvent and risk of PD as the use of occupation as proxy to solvent exposure overestimates actual exposure, thereby driving the association closer to the null; correct?

A Correct.

Q And I think we've discussed that second sentence previously; correct?

A Correct.

Q Do you have a citation for the assertion that use of occupation as a proxy to solve an exposure overestimates actual exposure in the Nielsen study?

A I don't. That's based off of my scientific expertise and being an epidemiologist and evaluating, you know, whether or not something over- or underestimates something.

Q So do you have any specific citations to the idea that using occupation likely overestimated actual exposure?

A I don't have a citation in my report.

Q Okay. Would that also apply to the Goldman study then?

A Yes. So using occupation is likely -- again, like I was saying earlier, not an exact estimate of TCE or PCE exposure. However, it's likely that what is being measured is lower than what is actually being seen in terms of true exposure.

Q Can you explain what you mean by that?

A Yeah. So whenever we are making -- when -- whenever we are driving an association closer to the null, it means that we are having a more conservative estimate.

And if we were to have measured our exposure more appropriately, then our magnitude of effect would be stronger.

Q So in terms of the Goldman study, would the number of individuals exposed to TCE and/or PCE also have been overestimated?

A So it's likely that using occupation was

not an exact estimate. And so it is very likely that the measures of association are more conservative because of that.

And if we had had a more -- if we were not using a proxy for TCE and PCE exposure, we likely would have had a stronger relationship.

Q Okay. Exposure in this study was based on an occupational job title reporting on a single census; correct?

A Correct.

Q Could that have led to exposure misclassification?

A Again, all epidemiologic studies likely have some form of exposure misclassification.

You know, the use of the survey in the Goldman study is going to be more appropriate because it captures more of an entirety of someone's occupation and hobby exposure, whereas the census is limited.

But, again, this is within the context of the entirety of the literature.

Q And you considered the findings of this study to be worthwhile to include in your report; correct?

A Yeah. It's part of the body of literature.

Q Despite the limitations that we were discussing?

MR. MICELI: Object to the form.

A Well, there's -- again, there's limitations in every single study out there. There's no perfect study, including RCTs.

And so it's one of the reasons why we want to look at the whole body as a whole and not just single publications. Because often what you'll see is one publication will address some of the limitations of another publication.

And so if we are seeing consistent effects where the limitations differ across the board, then you're seeing what we would say is the true effect.

BY MS. MIRSKY:

Q You didn't note what these limitations to the Nielsen study were in your report; correct?

MR. MICELI: Object to the form.

A Correct. I did not note them.

BY MS. MIRSKY:

Q And did you note the limitations to the

Goldman study in your report?

A I did not note them. And, again, it's not necessary because all studies have limitations.

And so in evaluating these studies, in looking at them, in looking at the body of work as a whole, you see where different studies come in and address the limitations of specific studies.

Q But the limitations of a specific study are important to consider when analyzing what the study's findings are; correct?

MR. MICELI: Object to the form.

A Could you repeat that?

BY MS. MIRSKY:

Q Understanding what a study's limitations are, are important when analyzing the study's overall findings; correct?

MR. MICELI: Object to the form.

A Yes. So we interpret our findings within the context of our limitations. But, again, within this report, we are really focusing on the broader picture and what we're seeing overarching.

And, you know, as we see these studies progress from case control to cohort studies, we start to see the different limitations that we're --

that we're talking about now being addressed in future studies.

BY MS. MIRSKY:

Q But you present the findings from Goldman 2012 and Nielsen 2021 without any discussion of those limitations; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:

Q Okay. You write in your report that the measure of association in Nielsen 2021 was likely a conservative estimate of the association between solvent and the risk of PD; correct?

A Correct.

Q What are you basing this assertion on?

A So this is based on the assertion that this idea that there is potential exposure misclassification is likely resulting in a more conservative estimate of our measure of association.

And if we did not have exposure misclassification, if we were measuring the true exposure, our true measure of association would likely be much larger in terms of magnitude of effect.

Q And as we previously discussed, you did not include the Sallmen 2024 study in your materials considered list; correct?

A Correct.

Q And to confirm, did you testify whether you considered this report in forming your opinions in this case?

A So I looked at the Sallmen paper. It's again another case control study based in Finland.

When you look at it, it actually, you know, again, fits in with the body as a whole. And I really wanted to start focusing on U.S.-based publications, so referencing another Finnish case control was not needed.

Q Okay.

MS. MIRSKY: Could I have Tab 39, please?

MR. MICELI: We -- are you going to Sallmen?

MS. MIRSKY: I'm just going to do this --

MR. MICELI: Okay.

MS. MIRSKY: -- and then it will be a good stopping point --

MR. MICELI: Okay.
MS. MIRSKY: -- if that's okay.
MR. MICELI: Yeah.
MS. MIRSKY: So I'll mark this as Exhibit 28.
(Whereupon, Boehme's Exhibit 28, Study Titled "Parkinson's Disease and Occupational Exposure to Organic Solvents in Finland: A Nationwide Case-control Study" by Sallmen, was marked for identification.)
BY MS. MIRSKY:
Q And this is the Sallmen 2024 study; correct?
A Yes. Do you have the supplementary material to this?
Q I don't have that with me, I'm sorry.
A Okay.
Q So this is an expanded case control study by some of the same scientists who worked on the Nielsen study that we just discussed; correct?
A Correct.
Q And like the Nielsen study and Goldman 2012, this study used occupational data to

extract exposure data; correct?
A Correct.
Q And this study included 17,187 individuals with PD or comparative movement disorders; correct?
A Correct.
Q And there were 35,738 controls; correct?
A Correct.
Q So this is a larger population than in the Nielsen study; correct?
A Correct.
Q And just for clarity, there were 11,757 individuals with PD in the Nielsen study, and 23,236 controls in the Nielsen study; correct?
A Correct.
Q And the results of this study demonstrated no association between Parkinson's and TCE exposure; correct?
A Not correct. If you look in the supplementary material, there is a distinct relationship.
Q In the main materials on page 43, the findings represented in this chart do not demonstrate an association between PD and TCE

exposure; correct?

A So what --

MR. MICELI: Object to the form.

A What the authors are doing in this table is they have categorized their continuous exposure, which is different than what the Nielsen paper did.

And in their supplementary analysis, they have the continuous TCE exposure. And you start to see that there's similar results in this paper to this paper when using the exposure as a continuous variable.

Any time we categorize something, we start to lose power. And because we are already looking at a rare exposure and a rare outcome, we really can't lose power with the categorization.

And so that's what we are seeing here in this table two. But in the supplementary material, you do see that there is a relationship between TCE and PD.

BY MS. MIRSKY:

Q Is -- are the associations in the Sallmen study weaker than those reported in Nielsen?

A I would have to look at the supplementary table. I don't remember off the top of my head.

Q Okay. And you said that you wanted to focus on U.S.-based studies and so therefore you decided not to include the Nie- -- the Sallmen study; correct?

A That, and it had similar results to the Nielsen study and -- and, you know, the idea of moving into really evaluating cohort studies to give a broad picture of, you know, what's happening when we see things over time as well.

Q But even if you hadn't included it as a citation within your report, it could have been in the materials considered list; correct?

MR. MICELI: Object -- object to the form.

A Sorry, what?

BY MS. MIRSKY:

Q Even if it hadn't been directly cited in your report, it could have been in your supple- -- in your materials considered list; correct?

MR. MICELI: Object to the form.

A Well, so when I was looking at this, as I was with other papers where I was reading the abstracts and the papers and determining whether I wanted to include them or not, you know, I did not

include all of those in the materials considered because that's part of the literature review in evaluating, using my scientific expertise to determine what to include in the report.

Including this would not have changed any of the opinions in my report. And, again, it's very similar to what the Nielsen study found.

BY MS. MIRSKY:

Q Other than your scientific expertise, were there any criteria that you employed to decide whether you included something in your materials considered list or not?

MR. MICELI: Object to the form.

A So, again, it's looking at the study design, the location, how they're measuring the exposure and the outcomes. I mean, we already have one where the exposure is measured the same way, the outcomes are measured the same way, it's in Finland.

You know, using occupation as a proxy has its own limitations, and so including more papers where occupation is the proxy for TCE exposure I felt was not necessary.

BY MS. MIRSKY:

Q Then why did you decide to supplement

your materials considered list with this report now?
A Because, again, this is just another piece that had been raised and discussed and it is the same as this.
Q So then why did you decide now to include it?
MR. MICELI: Object to the form.
A Because it was something I had considered, it was something that's been discussed, and so I was advised to include it.
BY MS. MIRSKY:
Q Okay. So it wasn't your independent decision to include it in the supplemental list?
MR. MICELI: Object to the form.
A No.
BY MS. MIRSKY:
Q In preparing this report, did you review a study called "Retrospective Cohort Study of Microelectronics and Business Machine Facility" by Sharon Silver?
MS. MIRSKY: It's just two questions.
MR. MICELI: Okay.
A I would have to look at that. I don't

1 know.
2 MS. MIRSKY: Okay. Okay. We can go
3 off the record.
4 THE VIDEOGRAPHER: The time right
5 now is 3:55 p.m. and we are off the
6 record.
7 (Whereupon, there was a recess taken
8 from 3:55 p.m. to 4:12 p.m.)
9 THE VIDEOGRAPHER: The time right
10 now is 4:12 p.m. and we are back on the
11 record.
12 BY MS. MIRSKY:
13 Q Dr. Boehme, you have on page eight of
14 your report a section titled "TCE, PCE exposure from
15 Camp Lejeune and PD mortality"; correct?
16 A Correct.
17 MR. MICELI: Are we done with both
18 Sallmen and --
19 MS. MIRSKY: Nielsen, yes.
20 MR. MICELI: -- Nielsen?
21 BY MS. MIRSKY:
22 Q You state that the levels of TCE, PCE,
23 and VOCs was up to 3,000 times the permitted safety
24 standards; correct?

A Where do you see that? Sorry.
Q It is --
A What page?
Q I think it's on page eight, sorry.
It's on the fifth --
A Oh, yes.
Q -- beginning on the fourth line.
Do you see that?
A Yes.
Q What safety guidelines are you referring to for TCE and PCE?
A Forty-eight, let's see.
So this references to the ATSDR position on the water contamination at Camp Lejeune.
Q So do you know which specific safety standards are referenced in that report?
A I would have to go back to the report.
Q Okay. And did you calculate the 3,000 number in your report?
A I did not. I referenced from the ATSDR.
Q Did you run any calculations to confirm whether the number reported in the ATSDR report was correct?
A That is outside of my expertise.

Q So you did not run any calculations on that issue?

A I did not run any calculations.

Q Okay. And -- sorry, so that's citing to number 48.

And that's the ATSDR's background website on the position on the water contamination at Camp Lejeune; correct?

A Yes.

Q Okay. And you also state that you will be assuming the median levels of TCE and PCE as set out in Goldman 2024; correct?

A Goldman 2024 and ATSDR.

Q And ATSDR. Okay.

And there is a number listed for Hadnot Point and a number for TCE and PCE and a number listed for Tarawa Terrace for TCE and PCE; correct?

MR. MICELI: Where are you looking?

MS. MIRSKY: On page eight of her report.

MR. MICELI: Okay. I just want to --

A Correct.

BY MS. MIRSKY:
Q Are you aware that Camp Lejeune had eight water systems?
A Yes.
Q And are you aware that individuals at Camp Lejeune may not have come into contact with the Hadnot Point or Tarawa Terrace water systems?
A Yes.
Q Did you review ATSDR's water modeling reports?
A I be- -- I believe I did. I don't know. Are they listed in my materials referenced?
Q I don't believe so.
A Okay.
Q Are you aware that the numbers listed in your report are estimates?
MR. MICELI: Object to the form.
A This is not my area of expertise. I don't do water modeling.
BY MS. MIRSKY:
Q So you can't answer questions on these numbers listed in your report?
A Correct.

Q And you don't know whether these numbers were based on assumptions?

MR. MICELI: Object to the form.

A Correct. Outside my expertise.

BY MS. MIRSKY:

Q Okay. Did you use any of these estimates in forming your opinions?

A No. I'm really looking at the epidemiologic results and recognizing that these are the thresholds that are referenced.

Q And by thresholds you -- they are median estimates of the levels of TCE and PCE in the water at Tarawa Terrace and Hadnot Point; correct?

A That's what they reference, yes.

Q Okay. You cite to a 2014 cohort study by Frank Bove comparing civilian co- -- civilian workers at Camp Lejeune to a group of people at Camp Pendleton in California; correct?

A Yes.

Q Okay.

MS. MIRSKY: And this is marked as Exhibit 29.

(Whereupon, Boehme's Exhibit 29, Bove 2014 Civilian Study, was marked for

identification.)
BY MS. MIRSKY:
Q Is this the Bove 2014 civilian study?
A Yes.
Q This study did not find any statistically significant increased risk of PD mortality in those with exposure greater than the median amounts compared to those with exposure less than the median amounts; correct?
MR. MICELI: Object to the form.
A Could you point me to where you are referencing this from? Which table?
BY MS. MIRSKY:
Q It's on page seven. There's -- in the -- in the -- in the paragraph below the table. And it lists the hazard ratio of 3.13 with a confidence interval of 0.76 to 12.86; correct?
A Correct.
Q And this study only included five individuals who died of Parkinson's; correct?
A From Camp Lejeune.
Q From Camp Lejeune?
A Correct.
Q And there were four people at Camp

Pendleton who died of Parkinson's; correct?

A Correct.

Q And so that's a difference of one person; correct?

A Yes. So when we're looking at Parkinson's, people with Parkinson's disease have a relatively normal lifespan. Parkinson's is not something that is generally considered a disorder that kills people. People die with Parkinson's; it doesn't kill them.

And so, you know, looking at Parkinson's mortality is a bit hard because that means someone would have to have not died from other causes.

So when we think about -- you know, I mentioned earlier, we're really good at saying when someone died. We're really bad in some ways at saying what the cause of death is from.

And so when we're looking at cause of death, you know, it's very likely -- very possible, not necessarily likely, but it's absolutely possible that when looking at the underlying cause of death there are other factors that are happening; right.

So people could easily have Parkinson's but die from cardiovascular disease, COPD, etc. And

so, you know, dying from Parkinson's disease is fairly rare.

Q Okay. And so you don't know whether more people were -- would be added to this study whether a stronger association would be seen in comparing Parkinson's death between Camp Lejeune and Camp Pendleton; correct?

A See --

MR. MICELI: Object to the form.

A You would need to understand how many other people in the -- in this mortality assessment had Parkinson's disease as well.

You know, again, dying from Parkinson's is not very common. People die of other causes when they have Parkinson's disease, and so studying Parkinson's mortality is a little tricky.

BY MS. MIRSKY:

Q And -- but the focus of this study was studying mortality; correct?

A Correct.

Q On page nine of your report, you state that -- let me find exactly where it is. It's about halfway down after citation 51.

The length of exposure was influenced by

length of employment (median length of employment at Camp Lejeune. This study was 2.5 years) indicating that two years on base is sufficient to increase the risk of PD; correct?

A I did state that, yes.

Q And do you have any support beyond the Bove 2014 study for the assertion that two years on base is sufficient to increase risk of Parkinson's disease?

A So in the other Camp Lejeune studies, I believe they look at the duration and so I'd have to look at those publications to see what the average duration was there in order to, you know, further assess that statement.

Q But your statement that two years on base is sufficient to increase the risk of Parkinson's disease is only citing to the Bove 2014 study; correct?

MR. MICELI: Object to the form.

A It is only saving [sic] to the Bove study.

BY MS. MIRSKY:

Q And the median length of employment in Bove 2014 was 2.5 years; correct?

A Yes.
Q So how did you determine that two years on base would be sufficient to increase the risk of PD just based on the 2014 study?
A So recognizing that the median is two and a half years, just under the median should be sufficient.
Q And you consider two years instead of 2.5 years to be just under the median?
A Yes.
Q Is it your opinion that if an individual spends less than two years on base, they would have no increased risk of PD?
A Not necessarily. We would need to further study that.
But the less time someone spends on base, the -- you know, there's -- it depends on what they're doing. And because they were looking at the median time spent on base trying to approximate TCE exposure, you know, it's very possible that shorter amounts of time on base could still confer an increased risk of Parkinson's.
Q Do you know whether any of the Camp Lejeune studies evaluate what the individuals in

the -- in the studies were doing on base?

A Could you repeat that?

Q Do you know whether any of the -- you said that understanding whether there would be risk under two years depends in part on what individuals would be doing on base.

Do you know whether any of the Camp Lejeune studies have information related to what the individuals were doing on base?

A I should clarify. So especially in the civilian population, you know, the evaluation -- no other studies look into what people are doing on base. But in understanding the exposure to water sources, exposure to a number of different factors may be helpful.

But it is definitely likely that, you know, a shorter term on base still confers an increased risk in Parkinson's.

Q You reviewed Dr. Bove's deposition transcripts; correct?

A I reviewed them in the context for evaluating -- for establishing that the ATSDR is peer reviewed.

Q So do you recall that Dr. Bove testified

that the study suffered from exposure misclassification issues?

MR. MICELI: Object to the form.

A I do not recall.

BY MS. MIRSKY:

Q Okay. Bove published another study regarding the mortality of Marines and Navy personnel in 2014; correct?

A Correct.

Q And the Bove 2014 mortality study of Marines and Navy personnel could not analyze Parkinson's because of the lack of Parkinson's data from the Camp Lejeune and Camp Pendleton cohort; correct?

A My understanding is that the military personnel were younger and on average, people aren't diagnosed with Parkinson's until after 65, whereas the civilian personnel were older.

And so, you know, again, Parkinson's is a disease that you live with for quite some time if you do not die from a different competing risk.

And so with the military personnel being younger, not only have they not necessarily had the opportunity to be diagnosed with PD but they also

1 don't have the opportunity to live long enough with
2 PD to then have any mortality associated with PD.
3 Q But therefore, the Bove 2014 mortality
4 study of Marines and Navy personnel could not
5 analyze Parkinson's because of the lack of
6 Parkinson's data from the Camp Lejeune and Camp
7 Pendleton cohort; correct?
8 A Yes.
9 Q Okay. You also relied on Bove's 2024
10 study titled "Evaluation of mortality among Marines,
11 Navy personnel, and civilian workers exposed to
12 contaminated drinking water at USMC base Camp
13 Lejeune: A cohort study"; correct?
14 A Let me look and see if it's --
15 MR. MICELI: Where -- where are you
16 reading from, just so I can keep up with
17 you?
18 A Yes. That's from page ten of my report,
19 reference 54.
20 BY MS. MIRSKY:
21 Q Thank you.
22 MS. MIRSKY: And I'll mark this as
23 Exhibit 30.
24 (Whereupon, Boehme's Exhibit 30, Bove

2024 Study Titled "Evaluation of Mortality Among Marines, Navy Personnel, and Civilian Workers Exposed to Contaminated Drinking Water at USMC Base Camp Lejeune: A Cohort Study," was marked for identification.)

A Okay. Thanks.

BY MS. MIRSKY:

Q And this is the Bove 2024 study; correct?

A Yes.

Q And this study compared a Camp Lejeune cohort to a Camp Pendleton cohort; correct?

A Yes.

Q This was the same cohort that was used in Bove 2014; correct?

A But published -- now they have more data. They have ten years more data.

Q But it is the same cohort; correct?

A Yes.

Q And as you were alluding to, the only change to the cohort was the age at the end of follow-up; correct?

A Correct.

Q The exposures for those at Camp Lejeune were not directly estimated in this study; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:

Q Instead, exposures were assumed based on assignment and employment tertiles of duration of assignment and employment at Camp Lejeune; correct?

A Correct.

Q And on page five in -- towards the bottom of the second column, the second to last paragraph starts with, Results of the duration analysis should be interpreted with caution because monthly contaminated levels fluctuated considerably between 1972 and 1985; correct?

A Correct.

Q Did you note this in your report?

A I did not note this.

Q Bove 2024 did not have data on other risk factors for the health outcomes considered; correct?

A Correct.

Q Would you agree that this is a weakness of this study?

MR. MICELI: Object to the form.

A It's a limitation.
BY MS. MIRSKY:
Q And you did not cite this limitation in your report; correct?
A Correct.
Q Bove 2024 observed 15 Parkinson's death among the Marines and Navy personnel; correct? If you look on page nine?
A Page nine, sorry.
Q So there are 15 Parkinson's death at Camp Lejeune; correct?
A Correct.
Q And it is correct that that was not statistically significantly different from the general U.S. population; correct?
A So they are comparing to Camp Pendleton.
And, you know, in looking at our hazard ratio here, it's a very large hazard ratio, but we do have very small numbers. We have 15 people in Camp Lejeune who died from Parkinson's and eight in Camp Pendleton who died from Parkinson's.
And so these small numbers are driving the statistical significance. However, it is a very large magnitude of effect.

Q On page six, table two, this has standardized mortality ratios; correct?

A Correct.

Q And what does the column "Camp Lejeune observed SMR" reflect?

A So that reflects the standardized -- hold on. Let me read this.

I see.

MR. MICELI: You are on page six.

MS. MIRSKY: Yes.

MR. MICELI: Okay.

A Let me triple-check before I say anything.

Okay. So the standardized mortality ratios here are looking -- are standardizing the mortality ratios using age, sex, race, and calendar period-specific U.S. mortality rates for underlying causes of death using a life table.

BY MS. MIRSKY:

Q So does table two then reflect the difference between the population at Camp Lejeune and the U.S. population?

A Yes.

Q And Parkinson's disease is listed towards

the bottom and the observed SMR is 1.47 with an odds -- excuse me, with a confidence interval of 0.73 to 2.21; correct?

A Correct. But the standardized mortality ratio is a little bit different than an odds ratio or a risk ratio. And we really look at it as less than one, one, or greater than one.

It's not necessarily interpreted exactly like we would a risk ratio. It's not a relative risk measure.

Q But nevertheless, this finding is not statistically significant; correct?

A But, again, we have a very small sample size here with 15 Parkinson's deaths from Camp Lejeune. And we do see that there is an increased risk of Parkinson-related death in the Camp Lejeune population.

Q But there is not a statistically significantly different risk from the general U.S. population; correct?

MR. MICELI: Object to the form.

A It's not statistically significant, but it is of public health relevance.

BY MS. MIRSKY:

Q Okay. And back on page nine, comparing -- table four compares Camp Lejeune to Camp Pendleton; correct?

A Yes.

Q And for Parkinson's disease, that is also not statistically significant; correct?

A Again, we have very small numbers in terms of our Parkinson's mortality of 15 and eight.

And while this adjusted and unadjusted hazard ratio is not statistically significant, it's very relevant from a public health perspective.

Q On page 11, there were 30 Parkinson's deaths among civilian workers; correct?

A Correct.

Q And the finding there was not statistically significant either; correct?

A Correct. But, again, we see an increased risk hazard ratio of 1.21, which should show us public health significance.

And this is particularly interesting because, as I was saying earlier, Parkinson's death generally underscores the burden of Parkinson's disease.

Q But even with the increased number of individuals, there is still not a statistically significant finding; correct?

A Correct. But there's still a magnitude of effect that is relevant from a public health perspective.

Q And the adjusted HR is lower for the civilian outcome than for the Marine outcome; correct?

A Correct.

Q And that's true even though there are more individuals in the civilian --

A We still --

Q -- studies --

MR. MICELI: Object -- excuse me.

A Sorry.

MS. MIRSKY: Wait. Let me finish my question first. We'll start over. We'll start over.

MR. MICELI: Yeah. All three of us were going.

Go ahead.

MS. MIRSKY: Okay. So can you read back my question, please? Sorry.

(Whereupon, the referred-to text was read back by the court reporter.)

BY MS. MIRSKY:

Q Okay. So there -- there is a decreased hazard ratio in the civilian study as compared to the Marine study even though there are more individuals in the civilian study; correct?

MR. MICELI: Object to the form.

A Correct. But with 30 deaths and 31 deaths, we are still looking at small numbers.

BY MS. MIRSKY:

Q But you still cited to this report within your -- or to this study within your report; correct?

A Yes. Because it's part of the entirety of the body of evidence in terms of, you know, the relationship between these compounds and Parkinson's disease and in this case, Parkinson's mortality.

Q Bove 2024, this study uses confidence interval ratios, which we discussed earlier; correct?

A Correct.

Q But you cite to the statistical significance; correct?

A I cite to the magnitude of effect.
Q I'm sorry. You cite -- yes, you cite to the ratios and the confidence intervals; correct?
A Correct.
Q So you do not use the confidence interval ratios; correct?
A Correct.
Q On page ten of your report, about halfway through the main paragraph, you state -- it starts with, The strong measure of association indicates that it is reasonable to postulate that if the sample size were increased, the associations would likely be statistically significant; correct?
A Correct.
Q That did not happen between the Marines and the civilians that were studied in this; correct?
MR. MICELI: Object to the form.
A There's a level that it must meet; right. And so we are still working with small sample sizes here with 30 and 31. And so when I say this, I'm talking about even more of an increase in -- in sample size.

BY MS. MIRSKY:
Q So what would be an appropriate increase?
A I would have to actually back calculate that.
Q Okay. And you have not done that for this report; correct?
A Correct.
Q And you don't know whether the ratio would go up if you added more people; correct?
MR. MICELI: Object to the form.
A The hazard ratios have been consistent in that they are showing a magnitude of effect for increased risk, in this case, of Parkinson's mortality.
It is likely that it would continue to show an increased risk. How that magnitude of effect would change would be dependent on what happens as the sample size increases.
BY MS. MIRSKY:
Q And in Bove 2024, the hazard risk decreased as the number of people were added to the study; correct?
A It's a different population.
Q But with a larger sample size, the hazard

1 risk went down; correct?
2 A The -- it's not really comparable because
3 you are looking at the military population and the
4 civilian population. They are different
5 populations.
6 Q Is -- okay.
7 You then state that -- and this is on
8 page ten -- that the authors explored whether base
9 duration explained some of the findings between
10 contaminant exposure and PD mortality and found that
11 regardless of whether duration was low, mediate --
12 medium, or high, the strong association remains
13 between Camp Lejeune and risk of PD mortality;
14 correct?
15 A Correct.
16 Q And you have these numbers listed on page
17 ten of your report. For ease of reference, we can
18 look there.
19 These findings were all not statistically
20 significant; correct?
21 A Correct.
22 Q And the adjusted hazard ratios that you
23 list here demonstrate a non-monotonic dose response;
24 correct?

A Correct.
Q Because the numbers do not increase with the increasing doses each --
MR. MICELI: Object to the --
BY MS. MIRSKY:
Q -- each time; correct?
A Correct.
Q Okay. On ten, you also note that the authors explored whether exposure misclassification contributed to the findings; correct?
A Correct.
Q And then you state that for both the civilian and military populations, exposure misclassification did not contribute to the findings; correct?
A Correct.
Q We talked about exposure misclassification before; correct?
A Yes.
Q And you haven't cited exposure misclassification as a limitation with respect to other studies that we discussed; correct?
A So, again, exposure misclassification is going to happen in every single study, including

RCTs; right. So whether or not we can truly define someone as exposed or unexposed is generally not something that we're able to do.

And so what we really focus on is ensuring that exposure is measured in the same way for everyone in our study because if we have what's called non-differential misclassification, so we expect that there's misclassification of exposure, as long as it's non-differential, we are not going to inappropriately influence our measure of association.

And what I mean by that is whenever we have a bias in our study, exposure misclassification being a potential bias, you run the risk of moving your magnitude of effect towards the null or away from the null.

When you have non-differential exposure misclassification, you move towards the null. So you have a more conservative estimate.

And so what that tells us is as long as we have non-differential misclassification for exposure, that the measure of association that we are calculating is likely an underestimate of our true measure of association.

And, again, we've been looking at our exposure measured in the same way across all studies.

And so Bove really did a great job of showing using the -- the quantitative bias analysis that, you know, our exposure is measured in the same manner. We only see differential misclassification if our exposure is measured in different ways across different populations within our sample.

And so when they conducted their quantitative bias analysis to -- to assess the extent to which the exposure misclassification may have had an impact on their measure of association, they actually saw that the measure of association got stronger after assessing the exposure misclassification.

So exposure misclassification, again, we really see it in a lot of epidemiologic studies, in all of our studies, and if there was concern about differential exposure misclassification, that is when I would have raised it in my report as non-differential exposure misclassification is ubiquitous in epi studies.

Q Okay. You then cite to the 2018 ATSDR

morbidity study; correct?

A Correct.

MS. MIRSKY: And -- thank you.

COURT REPORTER: Just a second.

MS. MIRSKY: Sure.

MR. MICELI: I'm going to have to carry this home.

MS. MIRSKY: I'll mark this as Exhibit 31.

(Whereupon, Boehme's Exhibit 31, 2018 ATSDR Morbidity Study, was marked for identification.)

BY MS. MIRSKY:

Q And this is 2018 morbidity study; correct?

A Correct.

Q And this study was the result of surveys that were sent to people who had lived and worked at Camp Lejeune and Camp Pendleton; correct?

A Correct.

Q And if you turn to page five, please?

MR. MICELI: Of the exhibit or of the report?

BY MS. MIRSKY:
Q Oh, I'm sorry. Table five, page 73 of the report.
MR. MICELI: Table five, page 73?
MS. MIRSKY: Yes.
MR. MICELI: Okay.
BY MS. MIRSKY:
Q This shows that for Parkinson's disease there is not a statistical significant or association shown between Camp Lejeune and Camp Pendleton; correct?
MR. MICELI: I -- I apologize, but I'm on page 73 of the Exhibit 31.
Is it -- you -- is it this?
MS. MIRSKY: Am I not looking at the right one?
Oh. Sorry. Want to look at this?
MS. PLATT: Yeah.
MS. MIRSKY: Sorry.
MR. MICELI: Does the first line start under digestive organs colon? Am I looking --
MS. MIRSKY: I'm looking for the right thing.

So it would be on page 70 -- yeah, it's page 73. It has multiple sclerosis as the first outcome.

MR. MICELI: Yes. Okay.

MS. MIRSKY: And Parkinson's disease is the second.

MR. MICELI: Yes. I apologize.

MS. MIRSKY: Okay.

MR. MICELI: Yup.

MS. MIRSKY: Okay.

MR. MICELI: Thank you.

MS. MIRSKY: Great. Thank you.

BY MS. MIRSKY:

Q So my question was: There's not a finding of association here compare -- comparing Camp Lejeune to Camp Pendleton; correct?

A So one of the things to note of this, so this is a survey, 31 percent response rate, and 93 percent of the military population was younger than 65.

Again, Parkinson's is generally diagnosed at 65 or older. And so, you know, this younger military population included in the survey is going to influence what you're seeing in terms of

Parkinson's.

Q But you still cited this as part of your evaluation of the overall literature on Parkinson's; correct?

A Correct.

Q And on page 74, the odds ratios comparing cancers and other diseases among Camp Lejeune Marines with those of Camp Pendleton, there again is -- the odds ratio is 0.89; correct?

A Correct. But, again, this population is much younger and hasn't had the opportunity to develop symptoms and be diagnosed.

Q And on page 76, odds ratio for cumulative TCE exposure in Marines at Camp Lejeune compared with those at Camp Pendleton, again, the -- all of the odds ratios are below one; correct?

A Correct. But, again, these are young people who haven't had the opportunity to develop disease yet.

Q And for low exposure, the odds ratio is 0.86; for a medium exposure the odds ratio is 0.87, and for high exposure the odds ratio is 0.29; correct?

A Correct.

Q And you say that on page 11 of your report, The authors found the results of this study were in support of the prior ATSDR report concluding evidence of causality for TCE and PD was equipoise and above; correct?

A Yes. And that's also referring -- because if we look back at page 73, you know, they see in the workers who were older and who did have the opportunity to develop Parkinson's that there's a relationship between TCE, PCE, and Parkinson disease.

Q Although this finding would be non-monotonic, correct, comparing CL versus CP to CL versus CP high exposure?

A Well, the CL versus CP is just Camp Lejeune to Camp Pendleton.

Q But the next one is for high exposure; right?

A For looking at just high exposure, which is not exactly looking at monotonic because it's looking at high versus low, medium, and so you would need to look at more of the categories.

Q Okay. You also relied on Goldman's 2023 study titled "Risk of Parkinson's disease among

service members at Marine Corps Base Camp Lejeune"; correct?

A Which one is that one? Let's see.

Q That is --

A Fifty-six?

Q Yes.

A Yes.

Q Sorry.

MR. MICELI: Good toss that time.

MS. MIRSKY: And I'll mark this as Exhibit 32.

(Whereupon, Boehme's Exhibit 32, Goldman 2023 Study Titled "Risk of Parkinson's Disease Among Service Members at Marine Corps Base Camp Lejeune," was marked for identification.)

A Thank you.

BY MS. MIRSKY:

Q This is the Goldman 2023 study; correct?

A Correct.

Q And the sample population for Goldman 2023 was the same population in Bove 2014; correct?

MR. MICELI: Object to the form.

A Yes.
BY MS. MIRSKY:
Q And this population was limited to April 1975 through December 1985; correct?
A Correct.
Q And this is also the same population used in Bove 2024; correct?
A Correct.
Q Within this cohort, Goldman only identified the analytic cohort to be used as those that used the Veteran's Health Administration or Medicare health services; correct?
A Correct.
Q And this excluded individuals that have never received VHA or Medicare services; correct?
A Correct.
Q This limits the study population; correct?
A Not necessarily. I mean, in order for them to conduct the study, they need to ensure that they are able to follow them over time, which is why they need the linkage, the VHA and Medicare.
Q But by having that limitation, it limits the size of the study; correct?

A Correct.
Q And this is the same Goldman that published Goldman 2012; correct? The twin study?
A I would have to look at his name.
Q It is.
A Okay.
MR. MICELI: We'll stipulate to that.
MS. MIRSKY: Thank you very much. . Samuel Goldman.
BY MS. MIRSKY:
Q The exposures in Goldman 2023 were based solely on being stationed at Camp Lejeune or Camp Pendleton; correct?
A The 2023?
Q Yes.
A Yes.
Q And there was no data on direct exposures; correct?
A In the 2023?
Q Yes, I'm just --
A Oh.
Q -- talking about 2023?
A Correct.

Q And on page 679, in discussing the -- some of the limitations of the study, it states that, quote, We cannot -- this is in the second column in the last full paragraph -- quote, We cannot be certain that everyone who resided at Camp Lejeune between 1975 and 1985 was in fact exposed to biologically meaningful levels of contaminants and we are unable to account for other environmental exposures that individuals from either camp may have sustained before, during, or after military service; correct?

A Correct. However, you know, that's okay in this circumstance because in terms of Camp Lejeune, that's going to bias the results towards the null.

So by having more people who you classify as exposed when they are not really exposed in your denominator is going to make the relationship appear weaker than it really is.

Q Are you aware whether Goldman did any type of dose response analysis in his Parkinson's study?

A In this 2024?

Q Yes.

Or 2023.

A Oh, in the 2023?

Is it 2023? Yeah, 2023.

Let me look.

It looks -- this one did not do dose response.

Q Do you believe that doing a dose response analysis would have better informed any causation analysis?

MR. MICELI: Object to the form.

A Not necessarily. Dose response is another one of the Hills considerations that's really not necessary in terms of establishing causality.

We know that there are many factors out there that need just a threshold effect and don't need a dose response.

BY MS. MIRSKY:

Q How did you determine that dose response is not necessary in conducting a Bradford Hill analysis?

A Oh, that's wide --

MR. MICELI: Excuse me.

THE WITNESS: Sorry.

MR. MICELI: Object to the form.

A That's widely discussed in epidemiology. So in terms of Bradford -- Bradford Hill consideration, the only one that really is truly necessary is temporality, followed by what we see in the literature, the entirety of the body.

Dose response in and of itself is not necessary because there are some factors where just a single exposure is all you need to then be set on the causal pathway to disease.

All epi textbooks discuss that whether dose response is met or not does not negate whether something is a causal factor.

BY MS. MIRSKY:

Q It -- are you aware of any studies that demonstrate that single exposure to TCE is causative for Parkinson's?

A What do you mean by "single exposure"?

Q Well, you just used the term "single exposure."

A Well, I was using that as an example in talking about dose response.

There could be also threshold effects, there could be minimum effects. There's a lot of

things that could occur with an exposure and, you know, that's one of the reasons why dose response is not a -- not necessary in order to discuss causality.

Q But are you aware of any -- of any studies that demonstrate that a single exposure of TCE is causative for Parkinson's?

A What do you mean by a single exposure?

Q In the way that you just used it?

A No, but I am aware that there are other studies that look at dose response and have shown that, you know, a higher dose at earlier ages leads to more severe symptoms and more severe disease and earlier onset of disease.

Q Goldman 2023 considered the association between TCE and PD; correct?

A Correct.

Q And Goldman noted that the water was contaminated with other VOCs, such as PCE; correct?

A Correct.

Q But Goldman's conclusions only consider the connection between TCE and PD; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:
Q Do you consider that ignoring the potential presence of other contaminants like PCE is a limitation of this study?
MR. MICELI: Object to the form.
A Not necessarily, because they are just using Camp Lejeune -- being at Camp Lejeune as a proxy to the exposure.
BY MS. MIRSKY:
Q At the bottom of 679, the last sen- -- the last couple sentences before six seven -- before conclusions, excuse me, it says, Finally, although TCE was the VOC present in the Camp Lejeune water supply at the highest concentrations --
(Whereupon, the court reporter requests clarification.)
MS. MIRSKY: I'm sorry.
MR. MICELI: Where are you reading that, please?
MS. MIRSKY: It's right above "Conclusions."
BY MS. MIRSKY:
Q Finally, although TCE was the VOC present in Camp Lejeune water supply at the highest

concentrations, the water also contained high levels of PCE, vinyl chloride, and benzene. These other compounds or mixtures of compounds could have contributed to the associations we observed.

Did I read that correctly?

A Yes.

Q And so do you agree that a limitation of the study was the fact that Goldman did not consider the impact of these other contaminants?

MR. MICELI: Object to the form.

A Not necessarily.

BY MS. MIRSKY:

Q On page 677, table three, there is an OR of 1.70 with a confidence interval of 1.39 to 2.07 when comparing the Camp Lejeune cohort to the Camp Pendleton cohort; correct?

A Correct.

Q But when Goldman limited the cohort to Parkinson's diagnosed before January 13th, 2020 -- excuse me, 2017, he found an OR of 1.28 with a confidence interval of 1 to 1.64; correct?

A Correct. However, again, you know, it's really important to extend that amount of time because Parkinson's symptoms generally exhibit after

the age of 65.

Q And January 13th, 2017 was when the VA designated Parkinson's as a presumptive service connected condition for veterans who served at Camp Lejeune between August 1st, 1953 and December 31st, 1987; correct?

MR. MICELI: Object to the form.

A Yes.

BY MS. MIRSKY:

Q So the fact that statistical significance did not persist when taking out those who were diagnosed prior to January -- excuse me -- after January 13th, 2017, shows potential selection bias occurred due to the available information in the press at that time; correct?

MR. MICELI: Object to the form.

A There would be no selection bias there. That would not -- that would not constitute selection bias.

So one of the things -- there's a couple of things here from an epidemiologic perspective. So the PD ascertained before January 13th, 2017. Again, these are younger people, so they need more time to develop their PD.

Additionally, an odds ratio of 1.28 still shows an increased risk. So there's still a relationship between Camp Lejeune and PD, even when not accounting for enough time for people to develop the disease.

And, you know, they're looking at Camp Lejeune and Camp Pendleton. Selection bias determines who would be sent to Camp Pendleton in the 1950s. So by removing any PD diagnosis after January 13th, 2017 would not contribute to selection bias.

And the other thing is PD has a very distinct clinical feature. And so when we think about potential misclassification of a movement disorder, we very rarely see misclassification where we would say someone has PD when they really don't.

Generally, in terms of misclassification of an outcome, we're categorizing people as non-diseased when they're really diseased.

BY MS. MIRSKY:

Q But the January 13th, 2017 date is there because that is when the VA designated PD as presumptive service connection; correct?

MR. MICELI: Object to the form.

A Correct. But that still does not -- that's not going to necessarily influence who is diagnosed with PD after 2017.

BY MS. MIRSKY:

Q But is it --

A You could go to a neurologist and say, Hey, I was at Camp Lejeune. There are risks from being, you know, exposed to this.

But that's not going to exhibit Parkinsonian symptoms. You can't fake Parkinsonian symptoms.

Q On page 11 of your report, you state in talking about this Goldman 2023 that it's significant that overall PD and these prodromal pre-PD diagnoses all demonstrated clear association and all demonstrated effect with statistical significance.

For the studies that did not show a statistical significance, you did not note whether statistical significance was found or not; correct?

A In the studies that didn't find statistical significance, I said it was not statistically significant in my report.

Q What is -- if -- if you were -- sorry,

excuse me.

Earlier in your report you stated that statistical significance was not important to your analysis; correct?

A Correct. But regardless, I still state whether something is statistically significant or not when I'm discussing them.

I'm really focusing on the magnitude of effect and, again, the overarching understanding of the literature. And we still see these very large magnitudes of effect, we see these increased risks in these populations.

I'm not basing this on statistical significance, but I do call out whether something is statistically significant or not during my interpretations.

Q Okay. On -- on page 12 --

MR. MICELI: Are you done with --

MS. MIRSKY: No, not yet.

MR. MICELI: Okay.

BY MS. MIRSKY:

Q -- at the top you state that the study found three months of exposure to TCE and PCE was sufficient for developing Parkinson's; correct?

A Correct.
Q And are you basing that on the -- on the first page it notes that the population was at Camp Lejeune or Camp Pendleton for at least three months?
A So I'm basing it off of what the authors state in their publication.
Q So you are basing it off of this statement?
A And they have other statements about it within the cohort assembly, I believe, as well. Hold on. Let me look and see where it is.
Yeah, so I'm basing it off of the study design.
Q Okay. Are you an expert in risk assessment?
A No.
Q Are you an exposure expert?
A What do you mean by that?
Q Are you qualified to opine as an expert on exposure as it relates to increased risk?
MR. MICELI: Object to the form.
A Are you talking about exposure from an epidemiologic perspective or exposure from a toxicology perspective?

BY MS. MIRSKY:
Q From a toxicology perspective?
A So I can discuss exposures from an epidemiologic perspective, but in terms of toxicology, I'm not an expert in that area.
Q You previously testified that one epidemiology study alone is not sufficient to prove causation; correct?
A Yeah. We look at the entirety of what we are seeing.
Q But in your report you state that based on this report that three months' exposure to TCE and PCE was sufficient for developing PD; correct?
MR. MICELI: Object to the form.
A Correct.
BY MS. MIRSKY:
Q And do you have an opinion beyond that statement about the study's finding?
A Well, so the study has found that people at Camp Lejeune have a higher incidence of Parkinson's than Camp Pendleton. You know, the big difference between Camp Lejeune and Camp Pendleton is exposure to TCE and these VOCs.
And so, you know, having at least

three months' exposure as per the study design here is sufficient for an exposed group to be -- to experience a higher incidence of PD.

Q You also relied on Goldman -- you can put the 2023 study to the side.

A Okay.

Q You relied on Goldman's 2024 study titled "Parkinson's disease progression and exposure to contaminated water at Camp Lejeune"; correct?

A Let's see. Where is that?

MR. MICELI: Sara, I want to note you have broken the record now for exhibits in one of these.

MS. MIRSKY: Thank you so much.

BY MS. MIRSKY:

Q Sorry, I think that it's num- --

A Correct. It's number 50.

THE WITNESS: Thank you.

BY MS. MIRSKY:

Q And I'll mark that as Exhibit 33 and that is the Goldman 2024 study; correct?

A Correct.

(Whereupon, Boehme's Exhibit 33, Goldman 2024 Study Titled

"Parkinson's Disease Progression and Exposure to Contaminated Water at Camp Lejeune," was marked for identification.)

BY MS. MIRSKY:

Q This used the same population analyzed in Goldman 2023; correct?

A Correct.

Q And the same population analyzed in Bove 2024; correct?

A Correct.

Q The Goldman 2024 study is limited to --

A Well, I should actually clarify.

So from that study population, this study is specifically looking at people with Parkinson's disease. So it's taking from the Camp Lejeune population, the 270 people with Parkinson's.

Q Okay. And this also limited the study to only those that received health care through the VHA or Medicare; correct?

A Same as the other one, yes.

Q But Goldman 2024 did not limit the population to diagnosis before 2017 as he did in Goldman 2023; correct?

A He did not do any -- actually, is -- there's supplementary information with this. I'd have to look at the supplemental information.

Q But as reported in the main section of the report?

A Supplemental is just as important as the main.

Q Okay. This study did not consider individual exposure to contaminants; correct?

A What do you mean by that?

Q The study inferred exposure based on residential location and time on base; correct?

A Correct.

Q And the study did not have any data on other lifetime exposures; correct?

A Correct.

Q On page 12 of your report, you state that -- and this is halfway in the bottom paragraph -- the authors found people who were exposed to VOCs at Camp Lejeune had faster progression of markers of PD progression; correct?

A Correct.

Q And VOCs includes TCE and PCE here; correct?

A Correct.

Q But Goldman 2024 did not observe an earlier age at Parkinson's diagnosis in exposed individuals; correct?

You can look at page 1734. Under "Results," halfway down it says, Age at PD diagnosis averaged 55.3 years and was similar in exposed and unexposed individuals?

A So can you repeat your question? Sorry.

Q That -- just that Goldman 2024 did not observe an earlier age at PD diagnosis in exposed individuals.

A So here we are not talking about ex- -- exposure changes because everyone is exposed. And so age at PD diagnosis among all the people is 55.3 in this. So that's all the PD participants at Camp Lejeune.

What are you comparing that to?

Q It says it was similar in exposed and unexposed individuals?

A Oh. Yeah, we need the supplementary information because that has --

Q Okay. If you look at the next page, 1735, where it says, Age at PD diagnosis, almost

towards the bottom of the chart? It says --
A Yes.
Q -- VOC --
A Exposed.
Q -- exposed 55.8 --
A Uh-huh.
Q -- VOC unexposed 54.5 and so the total is 55.3; correct?
A Correct.
Q And so would you agree that this is similar between the exposed and unexposed individuals as the authors state in their report?
A Yes.
Q Okay. Do you believe that if a chemical increased the rate of progression of Parkinson's, then the latency period would also be sped up?
MR. MICELI: Object to the form.
A Not necessarily.
BY MS. MIRSKY:
Q Would the age of diagnosis be lower?
A Age of --
MR. MICELI: Excuse me.
THE WITNESS: Sorry.
MR. MICELI: Object to the form.

1 Lack of foundation.
2 A Age of diagnosis is very dependent not
3 just on symptoms but on access to care and on other
4 factors. So not necessarily.
5 BY MS. MIRSKY:
6 Q But in discussing the other studies, you
7 were testifying that the small numbers were related
8 to the age at which the individuals were studied;
9 correct?
10 A So what I was saying was a lot of the
11 people haven't had the opportunity to be diagnosed
12 with Parkinson's.
13 These people have already been diagnosed
14 with Parkinson's and they are younger than what we
15 see on average; right.
16 So on average we see that diagnosis for
17 Parkinson's happens at 65 or older. This is
18 happening at 55 in this population, which is
19 younger. And so we still need more time for more of
20 this cohort to become older in order to develop
21 Parkinson's.
22 We have the other factor of competing
23 risk. If these people die from other disorders or
24 other effects, they won't have the opportunity to

develop Parkinson's.

Q But the -- this study is demonstrating that there is a younger than average, according to your testimony, diagnosis of Parkinson's for both the exposed and unexposed; correct?

A Could you repeat that? Sorry.

Q This study demonstrates that there's a younger average age of diagnosis at -- younger than what you were testifying to for both the exposed and unexposed; correct?

A Yes.

Q And so wouldn't that indicate that the populations studied in the other reports that we went through would have also included younger -- the ages of diagnosis for the exposed and unexposed?

A So --

MR. MICELI: Object to the form.

A -- this is including the younger people, but what I'm saying is in the people who -- so in this paper, who are non-PD just yet, they haven't had the time or the opportunity to be diagnosed with it, whether that's because they're still in the middle of their latency period or they're not going to develop PD is unknown.

1 This is a little funny in that all of
2 these people have PD, so it's hard to compare to
3 what we would see elsewhere.
4 BY MS. MIRSKY:
5 Q Okay. So moving onto your Bradford Hill
6 analysis on page 15 --
7 MR. MICELI: Before you get there
8 and -- let's go off the record just for a
9 second.
10 THE VIDEOGRAPHER: The time right
11 now is 5:11 p.m. We are off the record.
12 (Whereupon, there was a recess taken
13 from 5:11 p.m. to 5:14 p.m.)
14 THE VIDEOGRAPHER: The time right
15 now is 5:14 p.m. We are back on the
16 record.
17 BY MS. MIRSKY:
18 Q On page 14 you cited the Camp Lejeune
19 Justice Act of 2022, which includes that --
20 A Oh, page 15, you mean.
21 Q Page 15, yes.
22 A Okay.
23 Q Sorry.
24 -- that part of the standard is whether a

party produces evidence showing that the relationship between exposure to the water at Camp Lejeune and the harm is sufficient to conclude that a causal relationship is at least as likely as not.

Is that where the language in your three opinions came from?

A Yes.

Q Okay. And you apply the Bradford Hill criteria to TCE and PCE together; correct?

A Correct.

Q Are you aware of any other reports that have applied Bradford Hill to multiple contaminants at once?

A Could you elaborate on that question?

Q Well, you have analyzed all of the criteria for TCE and PCE together; correct?

A Correct.

Q Are you aware -- have you ever applied the Bradford Hill criteria to multiple contaminants at once before?

A I would have to go back and look.

Q Are you aware of any time when you've done that?

A I would have to go back and look at my

records.

However, you know, this is done based off of the expertise of the neurotoxicologist indicating that the mechanism of TCE and PCE is the same and so therefore can be considered in the same manner.

Q So your opinions as to all of the Bradford Hill criteria evaluating TCE and PCE together are based on your reliance on Dr. Cannon's report; correct?

MR. MICELI: Object to the form.

A It's based on my reliance on neurotoxicologists indicating that TCE and PCE are similar enough to be considered together.

BY MS. MIRSKY:

Q And that includes Dr. Cannon's report; correct?

A Correct.

Q And is it your opinion that the majority of Bradford Hill criteria are met when evaluating the relationship between TCE and PD, so TCE alone?

A Could you elaborate on that a little bit more?

Q If you were to conduct this analysis just based on whether there's a relationship between TCE

and PD, would you come to the same conclusion?

A So because TCE and PCE are similar in terms of their mechanism of action, it is relevant to apply epidemiologic studies that look at TCE and PCE as similar; right?

So we do have some epidemiologic studies that tease out PCE separately and that has shown that there is an increased risk of Parkinson's after PCE exposure.

There is more epidemiologic evidence for TCE than PCE, but it doesn't negate the evidence that is out there for PCE.

Q Is your evaluation for whether PCE and -- the relationship between PCE and PD meets the majority of the Bradford Hill criteria contingent on the TCE analyses and the neurotoxicology -- in your estimation the neurotoxicology findings that TCE and PCE are similar?

MR. MICELI: Object to the foun- -- form and foundation.

A So could you repeat that question? Sorry. I started getting lost.

BY MS. MIRSKY:

Q Nevermind.

Okay. On page 16 you list the Bradford Hill criteria; correct?

A Correct.

Q And this is the criteria you applied in your report; correct?

A Correct.

Q Did you perform a weight of evidence approach to your Bradford Hill analysis?

A So, you know, in terms of, again, evaluating the entirety of the body of evidence, you know, one thing in particular did not stand out more than another.

As shown here, temporality is consistently met, there's a consistent strong association. We are seeing from study to study very similar results. You know, the biologic plausibility in terms of mechanism of actions is met.

And so it's not really necessary to weight something more so than the other. And I was doing this based off of my scientific expertise and also recognizing that, you know, these considerations, the Bradford Hill criteria, came about in 1965. A lot has changed since then.

1 Particularly with regard to epidemiologic methods
2 and ways to evaluate biologic plausibility.
3 As we have moved from 1965 to now, we see
4 that there's a number of these considerations that
5 are not exactly necessary in order for something to
6 be on the causal pathway.
7 And so with all of that being said, you
8 know, it's more of a overarching broad overview.
9 Q And this analysis that you undertook for
10 these criteria is how you reached your ultimate
11 opinions in the report; correct?
12 A Correct.
13 Q And you formulated your own
14 interpretations of the criteria; correct?
15 A Correct.
16 Q You state that temporality is arguably
17 the only Bradford Hill consideration that's
18 necessary for concluding causation; correct?
19 A So, again, this is -- we are looking at a
20 lot of things all at the same time. So if we have
21 temporality and we have consistency, biologic
22 plausibility, experiment and analogy, then not
23 having some of the others is okay.
24 In terms of a consideration that

absolutely must be met to state causality, temporality is the only one.

Q Okay. You state that strength of association is met because the strong measure of association found in epidemiologic studies is consistently shown over and over in the above assessment; correct?

A Correct.

Q And is this conclusion based on your belief that if a study does not have statistical significance, it's likely because of the sample size?

A So this is based off of looking at the magnitude of effect. Strength of association is speaking strictly to the magnitude of effect. What is the risk ratio, odds ratio, hazard ratio that you are seeing.

And knowing that sample size is very dependent -- or sorry, knowing that statistical significance is very dependent on sample size and knowing that, you know, TCE, PCE exposure is rare, PD is relatively rare, we are working with small sample sizes that influence our statistical significance.

Q Okay. And is this opinion related to both TCE and PCE contingent on applying studies that only related to TCE to PCE?

A It is --

MR. MICELI: Object -- excuse me. Object to the form.

A Could you repeat the question?

BY MS. MIRSKY:

Q Is -- this analysis is related to both TCE and PCE together; correct?

A Correct.

Q And is your analysis of TCE and PCE together for strength of association contingent on studies focused on TCE applying to PCE?

MR. MICELI: Object to the form.

A That is part of it. But there -- it's also incorporating the studies on PCE and PD.

BY MS. MIRSKY:

Q Consistency refers to the reproducibility of findings within and across studies; correct?

A Correct.

Q And you state on page 18 that the -- the last -- next to last sentence of consistency, the different proxies, techniques, and populations did

not affect the consistent associations between TCE, PCE, and PD in the literature; correct?

A Wait, I'm trying to find that on this --

Q I'm sorry.

A You said second to last --

Q -- sentence in consistency.

The different proxies, techniques, and populations did not effect the consistent association between TCE, PCE, and PD in the literature?

A Oh, there we go. Sorry.

Okay. Sorry. What was the question?

Q Your report says that; correct?

A Correct.

Q Okay. And Goldman 2023, 2024, and Bove 2024 considered the same underlying populations; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:

Q And this would not demonstrate findings across different populations; correct?

MR. MICELI: Object to the form.

A But it's consistency within a population

and they do allow for additional time, which in a sense. makes it a different cohort.

BY MS. MIRSKY:

Q But it's not actually a different cohort; correct?

MR. MICELI: Object to the form.

A Correct.

BY MS. MIRSKY:

Q And you cite to Goldman 20- -- 2012, 2023, and 2024 in your report; correct?

A Correct.

(Whereupon, the court reporter requests clarification.)

BY MS. MIRSKY:

Q Goldman 2012, 2023, and 2024.

And as we discussed, those were all written by the same lead scientist; correct?

A Correct.

Q You state that the findings of these studies were always in the same direction with a strong magnitude of association; correct?

A Oh, and that's beyond the studies you were just referencing.

Q Yes.

A Yes.
Q But you do not discuss the lack of statistical significance in this analysis; correct?
A That's not part of this; right?
So statistical significance, again, is so dependent on sample size, it's why it's not a piece of the Bradford Hill criterion.
Q Okay. Regarding specificity, you conclude that that criterion is partially met; correct?
A Correct.
Q And you state that the more specific an association between an exposure and outcome or a population exposed and outcomes, the more likely the association is causal; correct?
A Where do you see that?
Q It's -- it starts at the very last sentence on 18.
A The more -- okay.
Q The more specific an association between an exposure and an outcome or a population exposed and outcomes, the more likely the association is causal?
A Correct.

Q Is that from --

COURT REPORTER: Excuse me. We lost the Zoom folks, I believe.

MS. MIRSKY: Can we go off the record, please?

THE VIDEOGRAPHER: The time is 5:25 p.m. and we are off the record.

(Whereupon, there was a recess taken from 5:25 p.m. to 5:27 p.m.)

THE VIDEOGRAPHER: The time right now is 5:27 p.m. and we are back on the record.

BY MS. MIRSKY:

Q Okay. Let's actually move onto dose response, please?

A Okay.

Q You found that dose response was partially met; correct?

A Correct.

Q And you state that there is consistent evidence of a dose response relationship; correct?

A Correct.

Q And you do not cite any specific studies for this; correct?

A I was not citing any specific studies in -- like, when I discussed the consistency -- well, I -- sorry, I did there.

Q Yeah, there are some.

A There are some. But I was not specifically culling out which studies would have dose response and which ones would not.

Q Aside from the 2024 civilian mortality study, there is no dose response for Parkinson's in any of the ATSDR studies with three or more categories; correct?

A Hold on.

So in the disease progression, there was tertiles: None, median, less than or equal to median, greater than median.

Q Okay. The 2024 civilian mortality study was based on duration and not direct exposure values; correct?

A Which exhibit was that?

I'd have to look.

MS. MIRSKY: Do you -- 2024, the exhibit that -- what exhibit was that? Thirty. Sorry.

BY MS. MIRSKY:
Q Twenty-nine.
A Twenty-nine?
Q Yeah.
A Oh. May you repeat the question, please?
Q The 2024 civilian mortality study was based on duration and not direct exposure values; correct?
A Correct.
Q Okay. Is it your opinion that any dose of TEC or PCE would be sufficient to cause Parkinson's?
MR. MICELI: Object to the form.
A I am not a neurotoxicologist so I don't necessarily know exactly what would be necessary.
BY MS. MIRSKY:
Q So you don't have an opinion on that?
A What has been shown in the cohort studies -- you know, we've seen that, for instance, here in the Goldman 2023 study, they estimated that it was -- said, 336 parts per billion. It is likely that based off of the exposure disease measures of association here, that exposure at a lower concentration would still lead to Parkinson's

because we see such a strong effect here that a lower exposure would still confer increased Parkinson's risk.

Q You previously testified that you didn't have any opinions regarding that 360 --

A I'm saying it from an epidemiologic perspective.

I don't -- I'm not a neurotoxicologist. I -- you know, but when thinking about our exposures in terms of epidemiologic perspectives, we understand that this is how this Goldman paper defined exposure and we see this very strong relationship. So it is likely that exposure at a lower concentration would still increase the risk of Parkinson's.

Now, again, I'm not a neurotoxicologist. This is, strictly speaking, from how we -- how we classify exposures in epidemiologic studies, how we think about them, how we think about their relationship to the outcome of interest and the magnitude of association.

Q On biological mechanisms you cite to Dr. Cannon's report; correct?

A Correct.

Q Are you familiar with Dr. Cannon's discussion of this criterion?

A I separated out my criterion from him. I was really looking to the mechanism of action and ensuring that I was appropriately discussing the neurotoxicology literature considering I am not a neurotoxicologist.

Q And you don't have an opinion, one way or the other, on the findings of those mechanism of action reports that we discussed earlier today; correct?

MR. MICELI: Object to the form.

From a toxicology standpoint?

BY MS. MIRSKY:

Q From a toxicology --

A From a toxicology standpoint, I do not.

Q Okay. On coherence you write, the third sentence, Both the laboratory studies evaluating the association between TCE and PCE and PD in basic science in animal studies and epidemiological studies evaluating the relationship in humans are entirely in line with the known mechanistic and toxicological effects of TEC and PCE.

Correct?

1 MR. MICELI: You may want to slow
2 down.
3 MS. MIRSKY: Okay.
4 BY MS. MIRSKY:
5 Q Did I read that correctly?
6 A Correct.
7 Q Okay. Do you know whether there are any
8 animal studies focused on whether TCE can cause PD?
9 A That is not my area of expertise.
10 Q So do you have any opinions on any of the
11 animal studies that are referenced in this sentence?
12 A That is not my area of expertise.
13 Q Okay. You also state that, PD is, by and
14 large, described as idiopathic or related to
15 genetics or environmental exposures; correct?
16 A Correct.
17 Q And this list does not include age;
18 correct?
19 A Age kind of falls under idiopathic. Age,
20 in and of itself, is not a risk factor.
21 Everyone who ages isn't -- you know,
22 it -- it's -- age is a little funny in that it
23 allows for the opportunity for the latency period to
24 progress far enough that symptoms develop. That is

what we mean when we say age is a risk factor for Parkinson's disease.

Q And this doesn't list head trauma as well; correct?

A I was not describing all potential risk factors.

Q Okay.

A And in general, you know, when we discuss Parkinson's, if someone is diagnosed with Parkinson's and there is no genetic factor, you know, idiopathic is often the diagnosis and that can include people who've had environmental exposures, head trauma, etc.

Q Okay. On page 20, you are talking about the comparisons between Camp Lejeune and Camp Pendleton. And you state, Their respected demographic characteristics were strikingly similar and should reflect similar rates and risks, however, the glaring difference is the contaminated water at Camp Lejeune; correct?

A Correct.

Q And you have no citations for these sentences; correct?

A I can provide citations.

Q Okay. Are you aware of any study that has analyzed the cohort's family history?

MR. MICELI: Object to the form.

A What do you mean by that?

BY MS. MIRSKY:

Q Are you aware of whether or not the cohort's family histories have ever been studied and compared?

A To my knowledge, no. But that should not be a factor as -- you know, as I described earlier when we are thinking about the counterfactual of Camp Pendleton to Camp Lejeune, we should see similar distributions in terms of Parkinsonian familial history for Camp Pendleton and Camp Lejeune.

So the incidence of PD captured in Camp Pendleton would include familial history, would include the other factors outside of what we've been discussing for TCE, PCE, in the Camp Lejeune exposures. And so accounting for that in the studies really wouldn't be necessary because of how they structured their unexposed group to most closely reflect the counterfactual experience of the exposed.

1 And, again, by that what I mean is Camp
2 Pendleton is really reflecting what we would have
3 seen in the Camp Lejeune population had they never
4 been exposed.
5 Q Do you know whether the activities at --
6 that -- that took place on base at Camp Lejeune
7 versus Camp Pendleton were the same during the
8 relevant time period?
9 MR. MICELI: Object to the form.
10 A That is not my expertise.
11 BY MS. MIRSKY:
12 Q Do you know whether the occupations of
13 those on Camp Pendleton were similar to those on
14 Camp Lejeune during the relevant time period?
15 MR. MICELI: Same objection.
16 A That's not my expertise.
17 BY MS. MIRSKY:
18 Q So you don't know the answer to either of
19 those --
20 A I --
21 Q -- questions?
22 A -- believe that occupation was discussed
23 in some of these publications. So I'd have to go
24 back and look and see when they discuss that there

are similarities in terms of appropriate unexposed groups. I would have to go back into those publications for those though.

Q Okay. In your conclusion, which is on page 21, you say that, The attributable risk proportion found 42 percent of the PD risk in the population at Camp Lejeune was due to TCE or PCE exposure; correct?

A That's what it says here, yes.

Q But on page 12 you stated it was 44.7.

A This is one of a few typos in here. My "5" is right above my "2."

I meant to type in 45 percent, which is 44.7 percent rounded up.

Q Okay. You state that, It is likely people with the longest exposure or highest exposure to TCE and PCE from Camp Lejeune die from other implicated health outcomes before being able to show symptoms of having PD.

Do you have any support for this assertion?

A So this is one of the basic epidemiologic concepts of competing risk. So this happens with diseases where we don't see symptoms until older

1 age; so Parkinson's is an example, dementia,
2 Alzheimer's.
3 And one of the factors is often we don't
4 have the full picture because people may die before
5 the age of 65, before the age of 70, whenever they
6 would start exhibiting their Parkinson's symptoms,
7 of other factors. So acknowledging that TCE and PCE
8 also increase the risk of other disorders, the idea
9 that people may have died from something else before
10 the opportunity to exhibit Parkinson's symptoms,
11 should not be ignored.
12 Competing risk is a big aspect of
13 epidemiologic studies.
14 Q Have you ever heard of Pearl's Ladder?
15 A Pearl's Ladder? It sounds familiar.
16 Is that -- can you give me the reference
17 to this or --
18 Q Or have you ever seen it applied in
19 scientific studies that you can recall?
20 A In what way?
21 Q It's a framework of analysis that's
22 somewhat similar to Bradford Hill.
23 MR. MICELI: Object --
24

BY MS. MIRSKY:
Q That's my -- that's my generalization of it.
MR. MICELI: Okay. Object to the form.
A In -- in -- okay.
So in terms of what context, I guess?
BY MS. MIRSKY:
Q Well, have -- do you know if you've ever --
A I've read --
Q -- encountered it?
A -- all of Pearl's work.
Q Okay. So are you familiar with Pearl's Ladder as a concept?
A I am. I've not used it.
And are you talking about using it in lieu of Hill's considerations?
Q No. I'm just curious whether you've ever used it?
A I'm -- I'm trying to -- so I know that when Pearl talks about causality I don't remember the phrase, "Ladder," explicitly but, again, he has a lot of work out there. And his causal

1 considerations are where the field of epidemiology
2 has been moving to, away from Hill criteria.
3 However, a lot of his causal
4 considerations really apply to the methodology of
5 publications -- or of our studies, and ways to
6 reduce bias, ways to ensure unmeasured confounding
7 has been met, etc.
8 In a lot of ways, what Pearl has
9 discussed in moving forward causality still fits
10 within the causal criteria, it's supplementary to
11 what this is.
12 Q Okay. Are you familiar with the National
13 Academies of Science?
14 A Yes.
15 Q Have you ever worked with -- if I call it
16 NAS, are you...
17 A That's fine.
18 Q Have you ever worked with NAS?
19 A I have no idea. Not directly.
20 Q Okay. Have you ever referred to or
21 relied on any studies by NAS?
22 A I have no idea.
23 Again, I have a lot of publications so
24 I'm not sure.

Q Are you familiar with Dr. David Savitz?
A Yes.
Q Would you consider him an authority on epidemiology?
A I would consider him an epidemiologic colleague.
Q Are you familiar with any of his texts on epidemiology?
A I am.
Q Are they authoritative treatises for epidemiology?
MR. MICELI: Object to the form.
A What do you mean by "authoritative treatises"?
BY MS. MIRSKY:
Q Are they texts that you would rely on in your own work?
A There are many texts. I would not call any of them -- I mean, like, again, it's an entire body of epidemiologic research.
But Dr. Savitz's work is highly regarded, his textbooks are often used in epi classes and are reliable.
Q Did -- are you familiar with the NRC's

study called, "Contaminated Water Supplies at Camp Lejeune, Assessing Potential Health Effects"?

A NRC?

Q I'm sorry.

NRC is the National Research Council.

A Okay. So -- and what's the title?

Q "Contaminated Water Supplies at Camp Lejeune, Assessing Potential Health Effects."

A Is that part of the CDC reports or ATSDR reports?

Q No.

A I don't know. I'd have to go back and look.

Q Did you review this report in preparing your report?

A I would have to look.

Q Are you aware that the report classified Parkinson's as inadequate, insufficient evidence to determine whether an associ- -- an association exists considering the literature on TCE, PCE, and solvent mixtures?

A I'd have to --

MR. MICELI: Object to the -- excuse me.

THE WITNESS: Sorry.
MR. MICELI: Object to the form.
A I would have to look at the report.
BY MS. MIRSKY:
Q Okay. Did you know that David Savitz chaired the NRC committee that authored this report?
MR. MICELI: Object to the form.
A Again, I'd have to look at the report.
MS. MIRSKY: Okay. Can we take a quick break?
MR. MICELI: Yeah.
MS. MIRSKY: Okay.
THE VIDEOGRAPHER: The time right now is 5:43 p.m. and we are off the record.
(Whereupon, there was a recess taken from 5:43 p.m. to 6:04 p.m.)
THE VIDEOGRAPHER: The time right now is 6:04 p.m. and we are back on the record.
BY MS. MIRSKY:
Q Dr. Boehme, earlier we discussed the epidemiology-related courses that you teach at Columbia and NYU; correct?

A Yes.
Q Do you create syllabi for these courses?
A Yes.
Q Do you maintain these syllabi after the course is completed?
A What do you mean by that?
Q Do you still have copies of the syllabi for your epidemiology courses, even after they're done?
A I don't generally go back to them, no.
Q But do you keep them?
A Not necessarily.
Q Okay.
MS. MIRSKY: We would request, to the extent that you have them, that those be produced?
MR. MICELI: Well, they were not part of the request for the deposition. We'll consider it and we'll evaluate it based upon the CMOs and consider it with the witness, but we can't say that we agree.
MS. MIRSKY: I understand.
MR. MICELI: Okay.

BY MS. MIRSKY:
Q Do you create PowerPoints for any courses in these lectures?
A Sometimes.
Q And do you keep these PowerPoints, after the course is completed?
A Sometimes. It depends on the course.
MS. MIRSKY: Okay. So we would make the same request and understand your position, Dave.
MR. MICELI: Okay.
MS. MIRSKY: Okay.
MR. MICELI: Thank you.
BY MS. MIRSKY:
Q Do you create reading lists for these courses?
A Sometimes.
Q And do you maintain these reading lists, after the course is completed?
A Generally, no, because the literature updates year to year.
Q Okay.
MS. MIRSKY: To the extent you still have those, we would request those.

MR. MICELI: Same.

BY MS. MIRSKY:

Q And do you create any handouts for these courses?

A No.

Q Okay. Do you have any staff supporting your work in this matter?

A On this? No.

Q Okay. Have you received any compensation in connection with your work in this case?

A Yes.

Q And approximately how much have you been paid to date?

A I believe it's right around 23,000.

Q And how do you charge for your services as an expert? Is it hourly or fixed?

A It's hourly.

Q How much do you charge per hour?

A It depends on what I'm doing. 350 for document review, drafting of report. I know it's 500 per hour for deposition, things like that.

Q Okay. And how do you keep track of your time?

A So I log it into my calendar and -- the

moment I start working on it, I put it in my calendar. And then I increase as I go along. And I do it within 15-minute increments.

Q Okay. Are you reimbursed for expenses or costs?

A I believe I have that on my fee schedule, but I've had no expenses or costs.

Q And do you have a fee schedule in this case?

A I provided it to --

MR. MICELI: This morning.

MS. MIRSKY: So that's related to the --

MR. MICELI: You have that, yeah.

MS. MIRSKY: Yeah.

I will mark this as -- sorry. Where did the -- here we go.

(Whereupon, there was a discussion off the record.)

MS. MIRSKY: I'll mark this as Exhibit 34.

(Whereupon, Boehme's Exhibit 34, Invoice, was marked for identification.)

THE WITNESS: Okay.
MR. MICELI: I've got a copy.
BY MS. MIRSKY:
Q So is this what you'd refer to as your fee schedule?
A This is not my fee schedule. This is my invoice.
Q Okay. So do you have a separate fee schedule?
A I do.
MS. MIRSKY: Okay. To the extent that hasn't already been provided, will you produce that, please?
MR. MICELI: We'll -- we'll take a look at it.
I don't know if it's in a letter, the retention, but we'll -- we'll take a look at it and get back with you.
MS. MIRSKY: Okay.
MR. MICELI: It may simply be an e-mail that says -- so I -- I don't -- I don't want to commit to something that may include privileged communications.
MS. MIRSKY: Understood.

BY MS. MIRSKY:
Q This invoice, I think at the top right, it says the date is October 12th, 2024.
Can you read that?
A No. That's --
Q Oh, or is it --
A It's ten, twelve --
Q -- European and it's --
A Yes.
Q -- December 10th --
A Yes.
Q -- 2024?
Got it. Okay.
Have you billed more to this matter since December 10th, 2024?
A No.
Q Okay. So this is up to date?
A That's my most recent invoice, yes.
Q Okay.
MR. MICELI: I don't mean to inter- -- but I think she's asking, have you done work since then?
THE WITNESS: Oh.
A I thought you meant, have I invoiced.

I have done work. I have not invoiced.
BY MS. MIRSKY:
Q Okay. Apologies.
MR. MICELI: I don't mean to interrupt --
THE WITNESS: I'm sorry.
MR. MICELI: -- and I don't want to get called out for speaking objections --
MS. MIRSKY: That's okay.
MR. MICELI: -- but I want to make sure you were fully informed.
MS. MIRSKY: Thank you for clarifying.
THE WITNESS: Yeah.
BY MS. MIRSKY:
Q Is your compensation in this matter in any way dependent on the outcome of this case?
A Absolutely not.
Q You -- your CV reflects that you have acted as an expert witness in the past; correct?
A I have no expert witness information on my CV, whatsoever.
Q Oh, then is it attached to the end of your CV --

MR. MICELI: Yeah.
BY MS. MIRSKY:
Q -- in your report?
MR. MICELI: Yeah.
A Oh, okay.
BY MS. MIRSKY:
Q You don't have to turn to it quite yet.
A Okay.
Q But generally speaking, what percentage of your annual income is earned from performing expert services?
A Oh, I have no idea.
It ranges year over year. And I'm very -- I tend to be very selective of which cases I take.
Q If we could turn to your CV, which -- your updated CV.
A Okay.
Q In the academic appointment section --
A Uh-huh.
Q -- it says that you were an assistant professor on tenure track at Columbia from 2015 to 2022; correct?
A Correct.

Q And are you a tenured professor at this point?
A I'm not a tenured professor.
Q Are you now an adjunct professor at Columbia University?
A Yes.
Q So does this mean that you are no longer a tenure-track professor?
A Correct.
Q And were you up for tenure and not selected?
A No. I was almost two years away from going up for tenure and decided not to go through with it.
Q And why did you decide not to go through with it?
A I mean, it's pretty personal, but around COVID and everything that happened with COVID, epidemiologists became very burned out and systematic changes within academia meant that a lot of us epidemiologists were going to struggle with tenure.
Q What do you mean by that?
A The way in which tenure is measured has

not caught up with how NIH funds grants.
Q Understood.
Returning to your work for Aetion --
A Uh-huh.
Q -- how are you compensated for that work?
A What do you mean?
Q Are you paid a salary? Are you --
A I'm paid a salary.
Q You are paid a salary?
A Uh-huh.
Q And what is your annual salary at Aetion?
A Do I have to disclose that?
MR. MICELI: That's not -- has nothing to do with what she does in this case.
MS. MIRSKY: So are you instructing not to answer?
MR. MICELI: I'm going to -- I'm going to instruct you not to answer if that's --
THE WITNESS: Okay.
MR. MICELI: Yeah. That...
Rule 26 says we have to provide her compensation for this case. That's

outside -- has nothing to do with this case and so we are going to instruct her not to answer.

If the -- if the Court orders us to do otherwise, we'll provide what that number is.

MS. MIRSKY: Okay.

BY MS. MIRSKY:

Q Does Aetion conduct work related to Parkinson's?

A Sometimes.

Q Has it done any work related to Camp Lejeune?

A No.

Q Will your compensation at Aetion be impacted by your work as an expert here?

A No.

Q Will your compensation at Aetion be impacted by the outcome of this litigation?

A No.

Q Okay. So in your report, could you turn to the list of your expert work since 2020, please?

It's all the way at the back --

A Okay.

Q Like, the last two pages.
A Oh, okay.
Q Or last three pages.
It's the blue tab --
A Okay. Thank you.
Q So this lists your expert work since 2020; correct?
A Correct.
Q Did you serve as an expert prior to 2020, as well?
A On one case -- two cases?
Two cases.
Q Okay. Are any of the cases on this list related to Parkinson's?
A I would have to look.
Q Okay.
A One other.
Q Which one would that be?
A That is the Steven Waltman (phonetic) vs. Travelers Insurance.
MR. MICELI: Which year?
THE WITNESS: That is 2021.
MR. MICELI: Okay.

BY MS. MIRSKY:
Q The attorneys Brach & Eichler --
A Yes.
Q -- is that correct?
Okay. And did you draft an expert report in that case?
A I did not.
Q Did you submit any affidavits or declarations to the court?
A No. It never made it that far.
Q What were your opinions in that case?
A My understanding is that that's confidential. And it wasn't just Parkinson's, it was multiple movement disorders.
Q Okay. Were your opinions subject to any Daubert or Rule 702 motions in that case?
A No.
Q Have any of your opinions or reports ever been subject to a Daubert or Rule 702 motion before?
A No.
Q Did any of your prior expert work relate to TCE?
A No.
Q Did any of your prior expert work relate

to PCE?
A No.
Q Did any of your prior expert work relate to VOCs?
A No.
Q Did any of your prior work relate to solvents?
A No.
Q Did any of your prior work relate to any of the opinions that you have put forward in this report?
A If you are referring to epidemiologic opinions, everything that I've done is based on epidemiologic opinions.
Specifically, to TCE and PCE and Parkinson's? No.
Q Okay. Other than this case, have you ever worked with any of the counsel representing you today?
A No.
Q Did you do anything in preparation of today's deposition?
A Yes.
Q What did you do?

A We reviewed some of the publications, we reviewed my report, and met with the lawyers.

Q And you mentioned that you reviewed Dr. Cannon's report -- excuse me -- deposition transcript; correct?

A Correct.

Q Did you disagree with any of the testimony that you read in that transcript?

A I was not reading it to determine whether I agreed or disagreed with anything.

Q But did you disagree with anything that I you read?

A I would have to re- -- go back through it and look to see.

Q Okay. How many times did you meet with counsel in preparation for this deposition?

A I don't know off the top of my head.

I think it's about three, not including this morning.

Q Okay. And, approximately, how long did each meeting last?

A Anywhere from an hour to a couple hours.

Q Okay. Do you have an approximation of how many hours you spent overall meeting with

counsel in preparation for --
A I have to go back to my calendar --
Q Let me just finish the --
A Sorry.
Q Do you have an estimate for how many hours overall you spent preparing for this deposition with counsel?
A I'd have to go back to my calendar.
Q Okay.
MR. MICELI: When we get the new invoice, we'll provide it.
BY MS. MIRSKY:
Q Were you provided with any documents in connection with this matter?
A You mean just before the deposition?
Q No. In -- in general?
A Other than the ATSDR, that was it.
Q Okay. Were you provided with any documents in connection with this deposition?
A No.
MS. MIRSKY: Okay. I have nothing further. Thank you for --
THE WITNESS: Wow.
MS. MIRSKY: -- your time,

Dr. Boehme.

THE WITNESS: Thank you.

MR. MICELI: All right. It's 6:15. I am going to have some other questions but since we took a break, I don't want to take a break. I'm just going to roll right in, if that's okay with you all?

MS. MIRSKY: That's up to Dr. Boehme.

THE WITNESS: I'm fine. I'm good.

MR. MICELI: Are you good to go?

THE WITNESS: Yes.

MR. MICELI: And I promise, I'm not going to take long.

I'm going to fold up some of this and so I can get to certain parts of it because, hopefully, it's all in order. I don't...

CROSS-EXAMINATION

BY MR. MICELI:

Q I'm going to try to, sort of, go back -- backwards more where we -- where we have been only

because I think that will be helpful. Hopefully, more recent stuff will be easier for you to recall.

When you were talking with Ms. Mirsky about the Goldman 2024 paper -- and that was Exhibit number 33.

And when you are looking at those, I want you -- if you've had things piled up, I want you to refer to Goldman 2024, Goldman 2023 -- I've got to make sure I have all of these correctly -- Goldman 2012, which was number 26.

A Yeah.

Q Are you there?

A Yes.

Q Okay. I'm going to have to --

A Oh, number 26 is the twins.

Q The twin study?

A Yes. Okay.

Q That's Goldman 2012; right?

A Yes.

Q Okay. I want to make sure I have that one as well. I'm just going to flip them out.

You were asked some questions about those three studies being the same population. Do you recall that?

A When I was asked about the same population, the twin pair was not part of this.

Q Okay.

A The 2023, 2024, and there is --

Q The Bove studies?

A Hold on. There -- I can tell you which exhibit it is in just a second.

MS. MIRSKY: I think it's 30.

MR. MICELI: Yeah. Thirty.

THE WITNESS: Thirty?

MR. MICELI: Yes. Thank you.

BY MR. MICELI:

Q Okay. We have 30, 32, and 33?

A Yes.

Q Do you have those all in front of you?

A Yes.

Q Okay. You were asked about -- several questions about the same population in these studies.

And I want to start off with -- with the -- with Bove evaluating mortality among marines, Exhibit 30. And when you look at table number one on page four of 14, do you see where that table is?

A Yes.

Q And it says, Camp Lejeune N?
A Uh-huh.
Q And it says, Total 159,128?
A Uh-huh.
Q Okay. Set that aside for just one second and go to the Goldman 2023, which is Exhibit 32; okay? And go to table number one there.
Do you see where it says, Camp Lejeune number of veterans, 172,128?
A Uh-huh.
Q Okay. Is that a yes?
A Yes.
Q Okay. So they may have been the same cohort but are they the same exact population?
A There are some differences. And again, you know, looking at the numbers, there's some slightly different numbers --
Q Okay.
A -- here but overall, they are trying to pull from Camp Lejeune and Camp Pendleton, and they are trying to get the most representative samples from both for these --
Q I understand.
A -- time periods.

Q I'm just trying to make clear that they are not the same exact population?
A Ah. There are some differences.
Q And if you look at Exhibit 33 --
A This is very different because this is just the PD patients so it's not including those without Parkinson's.
Q And does that include people -- is that people just from Camp Lejeune?
A Correct.
Q Okay. And the "N" there equals 177 -- or excuse me -- 177,000 -- excuse me -- 177 individuals -- correct? -- from Camp Lejeune?
A Who were exposed.
Q Okay.
A And then 93 who were unexposed.
Q Okay.
A So there's 270 people with PD --
Q Right.
A -- used in that population.
Q Clearly not the same population in all three studies; right?
MS. MIRSKY: Ob -- objection. Form.
A Correct. So the PD patients are pulled

from the population from the 2023 paper.
BY MR. MICELI:
Q Yup.
A But there are different -- they are not the same population.
Q Okay. And so is it uncommon to run statistical analyses or epidemiological studies off of a similar or same cohort?
MS. MIRSKY: Objection.
A Generally speaking, we are encouraged by NIH, the National Institute of Health, that when a large cohort is set up we do everything we can within that large cohort.
So you'll see examples of very large cohorts having hundreds of studies --
BY MR. MICELI:
Q Okay.
A -- run in those large cohorts.
Q And again, I'm just going to look at the -- and I'm going to paraphrase the headings or the titles of these.
The -- Exhibit 30 was the mortality -- the mortality comparison between the marines at Camp Lejeune and Camp Pendleton; correct?

A Correct.
Q Okay. So that's a mortality study comparing populations from two marine bases?
A Correct.
Q Okay. And Exhibit 32, "Risk of Parkinson's Disease Among Service Members at Marine Corps Base Camp Lejeune," is the title; correct?
A Correct.
Q And that's comparing the marines at Camp Lejeune to the marines at Camp Pendleton?
A Correct.
Q Is that the same study as Exhibit 30 or is that a different study?
A It's slightly different because we are looking at just marines whereas the other study had civilian workers. The 2023 study is looking at risk of Parkinson's whereas the evaluation of mortality is looking at mortality and called "Specific Mortality."
Q Okay. Not the same study?
MS. MIRSKY: Objection.
A Correct.
BY MR. MICELI:
Q Okay. And then, of course, the

Parkinson's -- or excuse me -- the Exhibit 33 is just the Parkinson's disease progression and exposure of the people at Camp Lejeune --

MS. MIRSKY: Object- --

BY MR. MICELI:

Q Correct?

MS. MIRSKY: Objection.

A Correct.

BY MR. MICELI:

Q All right. So are you familiar with the Framingham Heart Study?

A Yes.

Q How long has that study been going on?

A That study has been going on for decades.

Q And is that a single cohort?

A It started as a single cohort and has built and expanded.

Q Okay.

A And now there are child cohorts, etc.

Q And are they still running statistical analyses on this decades-old cohort?

A Yes.

Q Okay. So it's not uncommon for multiple analyses to be run on the same cohort?

MS. MIRSKY: Objection.

A Correct.

BY MR. MICELI:

Q Okay. You were asked about -- I need to -- what did I do with that? here we go.

You were asked some questions about Exhibit number 31, which is the morbidity study from ATSDR.

A Yes.

Q Okay. And you were asked to look at a number of different tables; correct?

A Yes.

Q Okay. You were -- if you could turn to page 73 for me?

A Yes.

Q Do you recall being asked questions about the Parkinson's disease findings among the workers on the far right side of the page, the last three columns?

A The focus was on the marines.

Q Okay. Do you recall being asked about those questions -- questions about the civilians, as well?

A I did discuss the civilians and the

relationship with the civilian workers who were at Camp Lejeune versus Camp Pendleton --

Q Okay.

A -- in terms of risk of Parkinson's disease.

Q Okay. Do you see in the, CL vs. CP, Camp Lejeune versus Camp Pendleton, high exposure?

A Yes.

Q There's a 2.9, and it has an asterisk after it?

A Yes.

Q What does that asterisk denote down at the bottom of the page?

A It says, "positive monotonic exposure response relationship."

Q Okay. And what is a positive monotonic exposure relationship -- exposure response relationship?

A So that is outside of my wheelhouse in terms of --

Q Okay.

A -- explaining it.

Q All right. Would you -- would -- who would -- who would be the expert to ask from --

1 categorically, is it a toxicologist?
2 A I would ask a toxicologist.
3 Q Okay. And since this is dealing with
4 neuro -- or Parkinson's disease, would you suggest a
5 neurotoxicologist?
6 A Agreed.
7 Q Okay. When you were discussing the
8 Goldman 2024 study with Ms. Mirsky, other than
9 asking about same population, she asked you some
10 questions about other lifetime exposures; do you
11 recall that?
12 A Yes.
13 Q Is there any reason to believe that other
14 lifetime exposures would only apply to the marines
15 at Camp Lejeune?
16 MS. MIRSKY: Objection.
17 A No, they should apply to both.
18 And this comes back to the discussions I
19 was having about counterfactuals in this regard.
20 And, you know, the -- that the Camp Pendleton is an
21 appropriate representation of what Camp Lejeune
22 would look like in terms of if they had not been
23 exposed to water contamination. And this would
24 include familial history, this would include other

exposures, etc.

We would expect that the distribution of those factors would be similar between the two.

BY MR. MICELI:

Q Okay. You wouldn't expect anything to be disproportionately skewed towards Camp Lejeune?

MS. MIRSKY: Objection.

A We would not expect anything to be different between the two populations.

BY MR. MICELI:

Q Now, when -- when you were also speaking with Ms. Mirsky about the Goldman 2024 study, you had asked about the supplemental data tables that went along with that study.

Do you recall that?

A So there were a couple of -- of the exhibits where I asked about the supplemental data and the supplemental information.

Q And I have, in my notes, this was one of those.

Is it necessary for you, when a study has supplemental data tables, to look at those in conjunction with the published literature?

A Absolutely.

So often journals will limit how many tables and figures you can publish and so more information is always provided in the supplemental material. This can also include methods that were used.

And just because something is in the supplemental information, doesn't mean it's not as important as what is in the full body.

Q Okay. Thank you.

If you could turn to Exhibit 32 with me, this is the Goldman 2023 publication.

A Yes.

Q Are you there with me?

A Uh-huh.

Q Okay. If you could turn with me to page 677?

A Okay.

Q Let me know when you are there.

A I'm there.

Q Okay. You were asked some questions about table three; do you recall that?

A Yes.

Q Okay. And you had mentioned something about for -- to have selection bias, you -- you

would -- you were mentioning something about having to have selection bias when people were selected for Camp Lejeune versus Camp Pendleton; correct?

MS. MIRSKY: Objection.

A Correct.

So, you know, the question around Parkinson's ascertained before or after January 13, 2017, does not pertain to selection bias.

BY MR. MICELI:

Q Okay. If you look -- and -- and you -- we don't need to go back over the -- the findings of the odds ratios and the confidence intervals, but one that was not discussed with you -- well, let me -- let me just ask this question. I apologize.

Do you see the probable -- possible or probable PD, total with PD, N equals 430?

A Yes.

Q Okay. And then if you drop down to the other one Ms. Mirsky discussed with you, the PD ascertained before January 13, 2017, N equals 257?

A Yes.

Q Okay. If you drop -- all other things being equal in a study, if you just "shrimply" -- "shrimply" -- simply reduce the number of people you

look at, what effect will that have on the power of your study?

A It's going to --

MS. MIRSKY: Objection.

A Sorry.

That's going to reduce --

BY MR. MICELI:

Q Let me ask -- let me ask it differently.

If all -- all other things being equal in a study, if you just decide to only look at a little more than half of the people, from an epidemiological standpoint, not talking about Goldman here, just from a -- from a epidemiological methodology principles point of view, shrinking the number of people you're studying will do what to the power, if you can say?

MS. MIRSKY: Objection.

A So that's going to decrease our power.

BY MR. MICELI:

Q Okay.

A And so we will likely not see statistical significance because we're decreasing our sample size.

Q Okay. Let's apply that now to the

1 Goldman table three -- Goldman 2023, table three.
2 When you shrink the size of people you are
3 investigating with PD from 430 to 257, is a lowering
4 of the odds ratio, and an effect on the confidence
5 interval lower bound, predictable?
6 MS. MIRSKY: Objection.
7 A It is.
8 BY MR. MICELI:
9 Q Okay. And why is that?
10 A So what's happening is the people who go
11 on to develop PD throughout the rest of the study
12 are classified as non-diseased, and so they are
13 moved into a different cell in our assessment. And
14 so we're artificially inflating our non-diseased in
15 that realm.
16 And so on top of that, we're also
17 decreasing the number of people with our outcome.
18 So we're going from 430 down to 257.
19 Q And so what does that do to power?
20 A That decreases our power.
21 Q Okay. And is power directly related to
22 statistical significance in this case?
23 MS. MIRSKY: Objection.
24 A So power and sample size are related to

statistical significance.
BY MR. MICELI:
Q Okay. Now, let's drop down. This is the other thing I wanted to go to wasn't discussed with you when -- when Ms. Mirsky was asking you questions.
But the probable PD, N equals 376, what is the odds ratio and confidence interval there?
A 1.81 with a confidence interval of 1.46 to 2.24.
Q Okay. And what does that tell us?
A So that tells us in the more strict definition of PD, that the relationship between Camp Lejeune and PD is stronger.
Q Okay. And is it statistically significant?
A It is.
Q And what does the P value of less than 0.001 mean?
A That it's statistically significant.
Q Okay. And the threshold for that is what, 0.05?
A Correct.
Q Okay.

A For this study is 0.05. It can range.
Q All right. We'll stay in this one for a second.
Turn to page 675 with me, if you would. The bottom of the left-hand column --
A Uh-huh.
Q -- there's a section called "Covariate Data"?
A Yes.
Q What is "covariate data" in this case?
A So these are additional variables that either are used in our table one to explain demographic characteristics between the two populations so that we can understand generalizability and, also, they might be used as covariates in our regression models to account for confounding.
Q Okay. And regression models accounting for confounding means what, for the benefit of the people who might read or see this deposition?
A So when we are doing our analyses of epidemiologic study, we do an unadjusted, where we are just looking at our exposure and our outcome, and then we move to what is called an adjusted

model. And this is where we acknowledge that with observational data there are often third factors, fourth factors -- other factors that can influence the relationship between our exposure and our disease.

And in order to most appropriately estimate the estimated true association, we need to account for confounding, these factors that can influence the exposure disease relationship.

Q Okay. If you go down underneath that section there's one that starts out, "Statistical analysis."

A Uh-huh.

Q Do you see that?

A Yes.

Q First sentence, tell me if I read this correctly, "We compared participant characteristics using Peter-" -- "Pearson...," and is that "chi-squared"?

A It is chi-squared.

Q Okay. "...statistic for categorical variables for student..." is that "T tests"?

A So the Pearson chi-squared is for the categorical variables and the student T-test is for

the continuous variables.

Q Okay. And those are statistical methods?

A Correct.

Q Used to do what?

A So these are used when we're looking at just comparisons between, in this case, two groups to determine if there is statistical significance.

Q Okay. And then if you go to the next sentence, it says, We evaluated association between Camp and PD using unconditional logistic regression to derive odds ratios and 95 percent confidence intervals.

Did I read that correctly?

A Yes.

Q What is logistic regression?

A So it is a type of regression model that we use when we have a dichotomous outcome, in this case, Parkinson's or no Parkinson's, and it allows us to calculate odds or odds ratios to determine our measure of association when we're evaluating our exposure/disease relationship.

Q Okay. Does it -- does it help refine the findings by decreasing the likelihood of bias affecting them?

MS. MIRSKY: Objection.

A Could you explain the question?

BY MR. MICELI:

Q Sure.

I mean, you do logistic regression to -- do you do logistic regression in order to refine the findings, the raw findings, of a study?

MS. MIRSKY: Objection.

A So we do logistic regression because, again, we start out with the unadjusted and then we need to account for confounding.

Now, whether someone considers confounding a bias or not is debatable, but confounding does influence our true measure of association and so we need to account for it in our regression models. And so that's why we moved to regression models, in this case logistic, because of our binary outcome.

And the authors state that they controlled for potential confounding in their regression models and those included age, sex, race, etc.

BY MR. MICELI:

Q Okay. All right.

1 And then at the bottom of page 679,
2 please, the conclusion say, This cohort's study's
3 findings suggest that the risk of PD is 70 percent
4 higher in veterans who were exposed to TCE and other
5 VOCs 40 years ago.
6 Did I read that correctly?
7 A Yes.
8 Q Okay. Trichlorethylene is a ubiquitous
9 environmental contaminant used in -- used throughout
10 the world since the 1920 [sic]. Many millions have
11 been continued -- have been and continued --
12 continue to be exposed.
13 Correct?
14 A Correct.
15 Q Okay. That's all I have on that one.
16 You've been asked a number of times today
17 whether or not you included, specifically,
18 mentioning limitations in your report. Do you
19 recall that?
20 A Yes.
21 Q Okay. And the -- and you've also said
22 throughout this deposition that every study has
23 limitations?
24 A Yes. Absolutely every study out there

has limitations.

Q Is that known?

A To people within the field of epidemiology, yes; to lay audiences, maybe not necessarily.

Q Okay. The -- what I wanted to ask you is that you cite to a number of studies and published epidemiological studies; correct?

A Yes.

Q Throughout -- we -- we have 33 -- we have 34 exhibits, one of them being your invoice, the vast majority of them have been peer-reviewed published medical literature -- or scientific literature.

MS. MIRSKY: Objection.

BY MR. MICELI:

Q Fair enough?

A I mean, I believe that --

Q Well, let me put it this way.

A Yeah.

Q Your depo -- aside from your depo notice, report, your updated CV, and your -- the -- the website from -- is it Anthion (phonetic)?

A Aetion.

The blog post from Aetion.
Q The blog post from Anthion -- Aetion.
What you've been shown in your supplemental disclosure, you've been shown a number of scientific journal articles; correct?
A Correct.
MS. MIRSKY: Objection.
BY MR. MICELI:
Q And an ATSDR report?
A Correct.
MS. MIRSKY: Objection.
BY MR. MICELI:
Q Okay.
A Yeah.
Q What is the purpose of the peer review process?
A So the peer review process -- when you are submitting a study to a journal, the idea behind the peer review process is for someone to come through and ensure that the way you've conducted your study -- and when I say conducted your study, I mean, your study design, how your exposure is defined, how your outcome is defined, covariates of interest, confounders, your research question of

interest -- is all done appropriately within the context of your study design and approach.

They also evaluate your statistical methods, your inference, your research, whether or not there's concerns that bias is going to grossly influence your measure of association and lead you to make an incorrect conclusion.

Q Okay. Does -- or -- is peer review a gatekeeper to make sure that what is published is reliable, scientific information?

MS. MIRSKY: Objection.

A Yes.

BY MR. MICELI:

Q Okay. I just wanted to put the cookies on a shelf I could reach them.

So I want to look with you at -- well, when you cite a study in your report, for instance -- well, we -- actually, we are going to get to that. We'll come back to that in a second and we'll talk about peer review again.

If you could turn with me to Exhibit 28, which is the Solomon article.

Are you there with me?

A Yes.

Q On the left-hand side of the page, there's a -- there is a --
A What page?
Q Excuse me. Page 45.
A Okay.
Q There's a -- there's a paragraph that begins, "It is difficult."
Do you see that?
A Yes.
Q Okay. Tell me if I read this correctly: It is difficult to undertake a detailed examination of potential selection bias related to both exposures and the outcome with respect to both outcomes and exposures because in case control design we did not access exposure for the entire parent cohort.
Did I read that correctly?
A Correct.
Q Okay. Such selection bias is always a concern and, therefore, our conclusions, strictly speaking, only apply to native born residents of Finland from specific era and age who were economically active, had census record, and had unambiguous diagnosis. Bias is unlikely to have

arisen from incidence, density, sampling, and controls.

Did I read that correctly?

A Correct.

Q Okay. Are you familiar with the fact that Camp Lejeune is in North Carolina?

MS. MIRSKY: Objection.

A Yes.

BY MR. MICELI:

Q Are you familiar with the fact that Camp Pendleton is in California?

A Yes.

Q So by the author's statement of limitations in Solomon, et al., Camp Lejeune and Camp Pendleton would not have application to residents of Finland from specific era and age -- native born residents of Finland from specific era and age; correct?

MS. MIRSKY: Objection.

A So extrapolating from this, it's listed as a limitation. Furthermore, they use occupation as a proxy and that is very Finland specific and era specific.

BY MR. MICELI:

Q Okay. Thank you.

I may have missed something on that.

Do you recall throughout -- I'm done with -- well, actually, keep that there.

Do you recall throughout this deposition being asked questions -- really throughout the entire day -- about risk factors for Parkinson's disease?

A Yes.

Q Okay. If -- if you'll turn back to that page 45, just under where we were reading a moment ago there's a sentence that begins, "These adjustments for confounding..."

Do you see that?

About midway down the paragraph that begins --

A Oh, yes.

Q -- we addressed.

We addressed potential confounding; do you see that?

A Yes.

Q They talk about that and then they have this sentence: These adjustments for confounding

may not have been complete because there are no well established risk factors for PD.

A Yes.

Q Do you see that?

A Yes.

Q Did I read that correctly?

A Yes.

Q Okay. Have we talked -- have you talked with Ms. Mirsky today about risk factors -- well established risk factors --

A I've been --

Q -- of Parkinson's?

A I've been discussing risk factors all day.

Q Okay. Thank you.

I'm going to go back, just for a moment, to the Goldman 2012 twin study.

Actually, I don't think we've been there yet. That would be number 26.

A Uh-huh.

Q Are you there?

You were asked a hypothetical question about not knowing when you were discussing table three on page seven -- well, 779 for me. It's

actually in the back of the exhibit that we used today. It's on page 14.

Do you recall table three?

A Yes.

Q And -- and you had testified that -- that with regard to PCE or PERC, that if -- it was your opinion that if the population size were increased that you'd expect to see -- it would be likely to see statistical significance achieved?

MS. MIRSKY: Objection.

A Correct.

BY MR. MICELI:

Q Is that an accurate summary of what you said earlier today?

A So that's a summary of what I said, yes.

Q Okay. Now, Ms. Mirsky asked you -- I counted about four times it may have been more -- that you don't know what would happen if we increased the sample size; right?

MS. MIRSKY: Objection.

BY MR. MICELI:

Q That's what she asked you?

A And that's why I was saying it would likely --

Q Right.
A -- reach statistical significance --
Q Right.
A -- particularly because of the magnitude of effect.
Q Okay. Because of magnitude of effect, you believe as an epidemiologist with your experience, education, and training, you think that it is -- do you believe that it is more likely than not that the -- a larger sample size would reflect a statistically significant finding?
MS. MIRSKY: Objection.
A Likely.
BY MR. MICELI:
Q Okay. And then can you say that with -- to a reasonable degree of scientific certainty?
MS. MIRSKY: Objection.
A Yes.
BY MR. MICELI:
Q Okay. And if you look down -- keeping that in mind and you look down at the effect of the TCE or PCE category that resulted in the 8.9 odds ratio and a confidence interval of 1.7 to 47, the fact that that number is an increase from what is

found in the TCE line, and that it is statistically significant, does that say anything to you as an expert with your education, qualifications, and training, and experience, whether or not PCE is contributing to that statistically significant finding?

MS. MIRSKY: Objection.

A Absolutely.

So, like when we were looking at TCE alone, odds ratio of 6.1, and then we add in and/or PCE, we see that this magnitude of effect increases, and that would only do so if there was a relationship between PCE and Parkinson's.

BY MR. MICELI:

Q Okay. So again to put it a little bit differently: Based upon your education, training, and experience, and what is reflected in this chart, table three of the Goldman 2012 study, is PCE a contributor to the -- to the statistically significant finding of TCE or PERC at 8.9 and statistically -- odds ratio 8.9 and statistically significant findings?

MS. MIRSKY: Objection.

A Yes.

BY MR. MICELI:
Q Okay. Can you say that to a reasonable degree of scientific certainty?
A Yes.
Q Okay. And with regard to the question that you were asked a number of times about it being less, the -- the -- the odds ratio going down, based upon your education, training, and experience, seeing what you see in table three, what do you believe the likelihood is that the odds ratio would go down if the sample size were increased?
MS. MIRSKY: Objection.
A So regardless, we would still see a strong magnitude of effect and statistical significance.
The extent of the magnitude of effect would depend on the -- how the sample sizes increased but we would still see a very strong magnitude of effect and likely see statistical significance.
BY MR. MICELI:
Q Okay. Can you say that to a reasonable degree of scientific certainty?
A Yes.

Q Okay. Based upon your education, training, and experience, what do you believe -- or do you have an opinion as to what the likelihood is that the -- that if you increased the sample size for PCE that the odds ratio would diminish to the null?

MS. MIRSKY: Objection.

A Very unlikely.

BY MR. MICELI:

Q Okay. Could you say that to a reasonable degree of medical -- or excuse me -- scientific certainty?

A Yes.

Q Okay. And why is that?

A Because of how strong this magnitude of effect is.

In order for this to go the null, this would -- you know, it -- there would have to be a lot of other factors that contribute to that. Increasing the sample size, we are likely to still see a strong magnitude of effect and a statistically significant relationship.

Q And is that evidenced by the fact of what we see with the TCE and the PERC line?

A Yes.
MS. MIRSKY: Objection.
THE WITNESS: Sorry.
BY MR. MICELI:
Q Okay. Have you ever, in your professional career, been asked to measure the loss of dopaminergic neurons in -- in a human or in an animal?
A No.
MS. MIRSKY: Objection.
BY MR. MICELI:
Q You got to let her --
A I'm so sorry.
MS. MIRSKY: It's okay.
BY MR. MICELI:
Q I know it's late and we all want to go home or go to some establishment with a tap.
That's on the record I'm sorry.
But let her make her objection.
A I'm sorry.
Q Hold on a second.
Have you ever been asked as an epidemiologist to measure the loss of dopaminergic neurons in a human or an animal?

MS. MIRSKY: Objection.

A No.

BY MR. MICELI:

Q Okay. Do epidemiologists run experimental experiments on animals to measure things like the loss of dopaminergic neurons from various chemicals?

MS. MIRSKY: Objection.

A In extreme circumstances where people have significant training in both epi and basic science they might, but the general epidemiologist does not.

BY MR. MICELI:

Q Okay. Without -- without training in toxicology or neurotoxicology -- let me just back up. I'll strike that.

Have you ever done it?

A Have I ever done what?

Q Have you ever measured dopaminergic neuron loss?

MS. MIRSKY: Objection.

A No.

BY MR. MICELI:

Q Okay. Do you rely upon or do you defer

to the opinions of specialists like neurotoxicologists for opinions on loss of dopaminergic neurons related to TCE and PCE?

MS. MIRSKY: Objection.

A Yes.

BY MR. MICELI:

Q Okay. So if we look at page six of your report, this is -- this is the part of the report that's trichlorethylene, tetrachloroethylene, and PD. Or excuse me, I'm looking at the wrong part. I apologize.

That's all part of the same line of questions. I'm going to withdraw that question. I'll just stop asking it.

With regard to the -- before I jump too far ahead, I'm going to get myself back oriented. Okay. Okay.

In the section on -- or on page six of your report, you have the -- if you look at the third line down, there's a sentence that begins, The route of exposure for TCE includes inhalation, dermal exposure, and ingestion.

Do you see that?

A Yes.

Q And you have reference to -- reference number 30; do you see that?
A Yes.
Q And I think you were -- you were asked this earlier, but number 30 in your report is the Zaheer study; do you recall that?
A Correct.
Q And do you recall Ms. Mirsky asking you questions about the substance of the Zaheer report or the Zaheer publication?
A Yes.
Q What was the purpose of citing Zaheer to this particular sentence?
A So again, I am not a neurotoxicology expert, but giving background into the different ways in which someone could be exposed to TCE is important in understanding how to measure it for epidemiologic studies.
Q Okay. So when you -- when you cite it, the first sentence, it says the route of exposure to TCE includes inhalation, dermal exposure, and ingestion, you are not getting into the substance of the actual toxicology of that study, are you?
MS. MIRSKY: Objection.

A Correct.
BY MR. MICELI:
Q Okay. So when -- when you -- if you don't site that again in this report, is the sole purpose for citing Zaheer is that it recounts the various routes of ingestion, dermal exposure, and inhalation?
A Yes.
MS. MIRSKY: Objection.
MR. MICELI: Okay.
THE WITNESS: Sorry.
MS. MIRSKY: Objection.
MR. MICELI: All right.
MS. MIRSKY: Thank you.
BY MR. MICELI:
Q So in a case like that -- and you've gone through this a number of times where you've said, you know, I'm not a toxicologist, I'm not a neurotoxicologist.
You've repeated that a couple of times --
A Yes.
Q -- or -- or dozens today?
A I'm not a toxicologist.
Q Okay. Because you are not a

toxicologist, when you look at the findings or when you look at the information, like citing it for inhalation, dermal, ingestion, are you constrained by the literature and the findings of the authors of those studies?

MS. MIRSKY: Objection.

A Yes.

BY MR. MICELI:

Q Okay. You were asked questions about Exhibit 16 in your deposition. This is the second of the Pezzoli articles that you reviewed with Ms. Mirsky, exposure to pesticides.

MS. MIRSKY: Objection.

A Yes.

BY MR. MICELI:

Q Do you recall -- okay.

Do you recall being asked a number of times whether or not this included all of the literature on pesticides and Parkinson's disease?

MS. MIRSKY: Objection.

A Yes.

BY MR. MICELI:

Q Okay. I believe you reviewed with Ms. Mirsky the inclusion and exclusion criteria for

that study -- for that meta-analysis; correct?
A Yes.
MS. MIRSKY: Objection.
A Yes.
BY MR. MICELI:
Q All right. Whether or not -- who would be the best person to ask whether or not Dr. Pezzoli reviewed all of the relevant literature in the world at the time he wrote that article? Would that be you or Dr. Pezzoli?
MS. MIRSKY: Objection.
A It would be Dr. Pezzoli and Dr. Cereda.
BY MR. MICELI:
Q Okay. Let me see.
Okay. Let me go into that.
Hold on a second.
You've talked about -- early in this deposition you talked about the methodology of -- of how you went about investigating and offering -- investigating the issues and offering your opinions in this case; correct?
A Yes.
Q Okay.
MS. MIRSKY: Objection.

THE WITNESS: Sorry.

BY MR. MICELI:

Q Based upon your education, training, and experience, did you satisfy yourself as an epidemiologist that you adequately reviewed the relevant literature in order to draw reliable conclusions on whether or not TCE and PCE are at least as likely as not to cause Parkinson's disease?

MS. MIRSKY: Objection.

A Yes.

MR. MICELI: Okay. I may be done. I want to take a short break. I want to call my colleagues real quick.

MS. MIRSKY: That's fine.

MR. MICELI: Okay. Let's go off the record.

THE VIDEOGRAPHER: The time right now is 7:02 p.m. and we are off the record.

(Whereupon, there was a recess taken from 7:02 p.m. to 7:07 p.m.)

THE VIDEOGRAPHER: The time right now is 7:07 p.m. and we are back on the record.

MR. MICELI: I have nothing further. Thank you, Dr. Boehme.

MS. MIRSKY: And before we go off the record, we'd like to order a rough draft, please. But Monday is fine.

Thank you Dr. Boehme.

THE VIDEOGRAPHER: The time right now --

MS. LaMACCHIA: Dave -- Dave? We don't need a rough, do you?

MR. MICELI: I don't think so.

MS. LaMACCHIA: I don't -- I don't either. No rough for the plaintiffs, please.

COURT REPORTER: Who's speaking, please?

MR. MICELI: That's Leslie LaMacchia. She sounds very different than Pat Telan.

MS. LaMACCHIA: Only on Fridays.

MR. MICELI: That's right. So thank you.

THE VIDEOGRAPHER: Off the record now, counsel?

MR. MICELI: Yes.

MS. MIRSKY: Yes.

THE VIDEOGRAPHER: The time right now is 7:08 p.m. and we are off the record.

(Thereupon, the deposition was concluded at 7:08 p.m.)

C E R T I F I C A T E

I hereby certify that I am a Notary Public, in and for the State of New York, duly commissioned and qualified to administer oaths.

I further certify that the deponent named in the foregoing deposition was by me duly sworn, and thereupon testified as appears in the foregoing deposition; that said deposition was taken by me stenographically in the presence of counsel and reduced to typewriting under my direction, and the foregoing is a true and accurate transcript of the testimony.

I further certify that I am neither of counsel nor attorney to any of the parties to said suit, nor am I an employee of any party to said suit, nor of any counsel in said suit, nor am I interested in the outcome of said cause.

Witness my hand and seal as Notary Public this 14th day of May, 2025.

_____

Clifford Edwards

New York Notary ID Number: 01ED6430906

Notary commission expires: 3/28/2026

J U R A T

I have read the foregoing 414 pages and hereby acknowledge the same to be a true and correct record of the testimony.

_____

AMELIA K. BOEHME, PHD, MSPH

Subscribed and sworn to
_____.
Before me this _____ day of _____,
2025.

_____
Notary Public
My Commission Expires:

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