

Exhibit 170

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

IN RE:

CAMP LEJEUNE WATER LITIGATION)

)

) Case No. 7:23-cv-000897

)

This document relates to:

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ALL CASES.

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The videotape deposition of
BRIANA DE MIRANDA, Ph.D., taken at
Environmental Litigation Group,
2160 Highland Avenue South, Birmingham,
Alabama, on the 14th day of January,
2025, commencing at approximately
9:36 a.m. CST.

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1 VIDEOGRAPHER:

2 We are now on the record. My name is
3 Michael Matheny. I'm the videographer for Golkow,
4 a Veritext division.

5 Today's date, April 14th, 2025, and the
6 time is 9:36.

7 This video deposition is being held in
8 Birmingham, Alabama, in the matter of Camp Lejeune
9 Water Litigation, For the United States District
10 Court for the Eastern District of North Carolina.

11 The deponent is Dr. Briana De Miranda.
12 Counsel will be noted on the stenographic
13 record.

14 The court reporter is Lois Robinson and
15 will now swear in the witness.

16 BRIANA DE MIRANDA, Ph.D.,
17 the witness, after having first been duly
18 sworn to tell the truth, the whole truth, and
19 nothing but the truth, was examined and testified
20 as follows:

21 MR. TELAN:

22 Good morning. My name is Pat Telan, and
23 I represent the plaintiffs.

24 MR. MICELI:

25 David Miceli, and I represent the

1 plaintiffs as well.

2 MS. PLATT:

3 Elizabeth Platt on behalf of Department
4 of Justice.

5 MS. ELLISON:

6 Anna Ellison, also on behalf of the
7 Department of Justice.

8 EXAMINATION

9 BY MS. PLATT:

10 Q Good morning, Dr. De Miranda. Thank
11 you so much for being here today.

12 MS. LaMACCHIA:

13 Leslie LaMacchia via Zoom for the
14 plaintiffs.

15 MS. PLATT:

16 Q Would you please state your full name
17 for the record?

18 A Briana De Miranda.

19 Q And your current address?

20 A My home address?

21 Q Yes.

22 A 2920 Kirkcaldy Lane, Birmingham,
23 Alabama, 35242.

24 Q Thank you.

25 As I said, my name is Elizabeth Platt.

1 I represent the United States in this matter.

2 This is a court proceeding. Even
3 though we are not in a courtroom today, you are
4 under oath.

5 Do you understand you are obligated to
6 tell the truth?

7 A I understand.

8 Q Our court reporter is taking down
9 everything we say, so it's important that you
10 answer questions verbally. For example, say
11 "yes" or "no" rather than nodding your head.

12 Do you understand?

13 A Yes.

14 Q Please talk at a reasonable pace so
15 that the court reporter can get everything down.

16 And we should try not to interrupt each
17 other. Please let me finish my question so that
18 it is fully on the record before you begin to
19 answer, and I will try to do the same for you.

20 A Great.

21 Q If you do not understand a question,
22 please let me know, and I will try to clarify.

23 From time to time, you may hear your
24 attorney say "objection." Unless they instruct
25 you not to answer the question, you should please

1 answer the question.

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

5 A No.

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

8 A No.

9 Q Are you currently taking any medication
10 that might impair you?

11 A No.

12 Q You may ask for breaks. I just ask
13 that you answer my question before we take a
14 break.

15 A Okay.

16 Q I'm gonna go ahead and mark Deposition
17 Exhibit Number 1.

18 (DEPOSITION EXHIBIT NUMBER 1
19 WAS MARKED FOR IDENTIFICATION.)

20 MR. MICELI:

21 We have a wider table than the other
22 day. Thank you.

23 MS. PLATT:

24 Q Dr. De Miranda, do you recognize this
25 document?

1 A Yes.

2 Q And this is the subpoena for your
3 deposition today; correct?

4 A Yes.

5 Q If you turn to Attachment A --
6 It's the last page.

7 Have you reviewed this Request For
8 Production of Documents?

9 A Yes.

10 Q And you and your counsel have produced
11 some documents today; correct?

12 A Yes.

13 Q And that is an updated version of your
14 CV?

15 A Yes.

16 Q And your billing information; correct?

17 A Correct.

18 Q Do you have any other documents to
19 produce today?

20 A I don't believe so.

21 Q Are you withholding any documents in
22 response to this subpoena?

23 A I don't believe so.

24 Q I just want to walk through this
25 subpoena really quickly.

1 The first request is for all emails,
2 letters, correspondence, text messages,
3 conversations, chats, voicemails, data, technical
4 files, and other communications pertaining to
5 Camp Lejeune sent or received prior to your
6 retention as an expert in this matter, and that
7 is with including but not limited to Morris
8 Maslia and David Savitz.

9 Did you have anything to produce in
10 relation to that request?

11 A No. I don't have any communication
12 with those -- those people.

13 MR. TELAN:

14 And, Elizabeth, I -- I just wanted to
15 note our objection I think that we sent over and
16 filed in relation to that particular request that
17 you made. And I didn't mean to interrupt, but I
18 didn't want my silence on that to be construed as
19 a waiver of the objection previously.

20 MS. PLATT:

21 Understood. I have received your
22 objections.

23 Q The second request is for the same
24 communications pertaining to Camp Lejeune,
25 including but not limited to the following

1 people.

2 Do you have any communications with
3 Robert Faye?

4 A No.

5 Q Jason Sautner?

6 A No.

7 Q Rene Suarez-Soto?

8 A No.

9 Q Susan Martel?

10 A No.

11 Q Scott Williams?

12 A No.

13 Q Frank Bove?

14 A No.

15 Q Mike Partain?

16 A No.

17 Q Jerry Ensminger?

18 A No.

19 Q Laurie Freshwater?

20 A No.

21 Q Paul Rosenfeld?

22 A No.

23 Q Do you have any communications with
24 Dr. Samuel Goldman?

25 A Yes.

1 Q And they're related to Camp Lejeune?

2 A They would be related to TCE and
3 Parkinson's disease, including Camp Lejeune.

4 MS. PLATT:

5 Okay. We request those communications
6 be produced following this deposition.

7 MR. TELAN:

8 And we'll -- we can take that up later.
9 But I think we'd have the same objections we made
10 to the request in Dr. Miller's deposition. So
11 that's something we can discuss --

12 MS. PLATT:

13 Yes. We can follow up after the
14 deposition.

15 Q You have published with Dr. Goldman;
16 correct?

17 A Yes.

18 Q Number 3 of the request, all emails,
19 letters, correspondence, text messages,
20 conversations, chats, voicemails, or other
21 communications to, from, or with any individual
22 that has filed a claim with the Department of
23 Navy or the Eastern District of North Carolina
24 pursuant to the Camp Lejeune Justice Act of 2022.

25 Do you have anything to produce in

1 response to that?

2 A Not that I'm aware of. I don't know
3 the names of anybody who's filed a claim
4 specifically. But I have in the past had email
5 communication with people who were stationed at
6 Camp Lejeune and now have Parkinson's disease.

7 Q And do you know if any of those
8 individuals have filed a claim?

9 A I don't know that information.

10 Q Okay. We would request that those
11 communications be produced.

12 MR. TELAN:

13 That may be a bit more difficult. We
14 can discuss that. But we would raise an
15 objection to that, I think, perhaps HIPAA,
16 particularly for those who may not have filed
17 claims. But, again, we can discuss that later.

18 MS. PLATT:

19 We can discuss it after the deposition.

20 MR. TELAN:

21 Yeah.

22 MS. PLATT:

23 Q And, then, final request, all bills,
24 invoices, or other documents reflecting
25 compensation paid to Briana De Miranda related to

1 the Camp -- CLJA litigation.

2 Do you have anything other than what
3 you produced today?

4 A No. Only this sheet.

5 Q Thank you. You can set that aside.
6 And I want to go ahead and mark Exhibit
7 Number 2.

8 (DEPOSITION EXHIBIT NUMBER 2
9 WAS MARKED FOR IDENTIFICATION.)

10 MS. PLATT:

11 Q Dr. De Miranda, do you recognize this
12 document?

13 A Yes.

14 Q This is the report that you have
15 prepared in this litigation; correct?

16 A Correct.

17 Q And I want to turn to your CV, which is
18 about halfway back in your report. It's
19 following page 17, and then the numbers restart
20 as page number 1.

21 Is this your current CV?

22 A No.

23 Q And you produced your current CV this
24 morning; correct?

25 A Yes.

1 Q And what have you updated since
2 producing the one in December that is part of
3 your report?

4 A The updated CV that I produced includes
5 an additional grant that's TX240245, as well as
6 dates for a supplement to my R01 that was -- I
7 did not have at the time I produced this in
8 December.

9 Q And I'm sorry. What page is that on?

10 A Four.

11 And the other update is a couple of
12 grant review panels that I've served on since.
13 That would be on page 8.

14 Q And which ones in particular?

15 A Let's see. NINDS, the 2025 /05 ZES1 --
16 Oh, sorry. I'm reading the wrong one.
17 I apologize.

18 It's NINDS 2025-05 NST-2, and then --

19 I think that's the only different one,
20 if I look between the two. The other one is a
21 Parkinson's Foundation, but I've listed those as
22 just a standing member. So that's not a
23 particular line item that I update.

24 Q Any other changes?

25 A I don't believe so.

1 Q This --
2 Let's go ahead and mark this as Exhibit
3 Number 3.

4 (DEPOSITION EXHIBIT NUMBER 3
5 WAS MARKED FOR IDENTIFICATION.)

6 MS. PLATT:

7 Q Does your copy have writing on it?

8 A This one?

9 Q Yes.

10 A No.

11 MS. PLATT:

12 Can we go ahead and mark that one?

13 MR. TELAN:

14 Sure.

15 MS. PLATT:

16 Just so that we have the new version on
17 record.

18 MR. TELAN:

19 Three is the newest version?

20 MS. PLATT:

21 Yes. The one that was produced this
22 morning.

23 Q So Exhibit 3, your newly produced CV,
24 is that a complete representation of your
25 educational and professional background?

1 A Yes.

2 Q Is there any information that you did
3 not include?

4 MR. TELAN:

5 Object to the form of the question.

6 A I don't have, like, previous jobs on
7 here. This is curriculum vitae. So in terms of
8 education, it includes what I've done in academic
9 research.

10 MS. PLATT:

11 Q Okay. No academic research has been
12 left off?

13 A No.

14 Q You have a bachelor's of science in
15 biological science; correct?

16 A Correct.

17 Q And that's from Colorado State
18 University?

19 A Correct.

20 Q And you have a Ph.D. in toxicology;
21 correct?

22 A Yes.

23 Q From Colorado State University?

24 A Yes.

25 Q Do you hold yourself to be a

1 toxicologist?

2 A Yes.

3 Q And what is the basis for your
4 expertise in toxicology?

5 A The basis for my expertise in
6 toxicology comes from my graduate training and
7 the Ph.D. that I hold in toxicology.

8 Q Anything else?

9 A I currently conduct research in a
10 toxicology field.

11 Q Anything else?

12 A I publish papers in the field of
13 toxicology. I serve as an associate editor on
14 the board of Neurotoxicology. I have been a
15 member of the Society of Toxicology since, I
16 believe, 2011 or '10. Let me look at my list
17 here. 2011. And I regularly review papers that
18 fall under the toxicology research field, as well
19 as serve on grant review panels that -- and
20 review grants in the toxicology field.

21 Q Anything else?

22 A I was -- I formerly held a secondary
23 appointment in the Department of Pharmacology and
24 Toxicology at the University of Alabama at
25 Birmingham.

1 Q Anything else?

2 A I think that predominantly sums it up.

3 Q Fine.

4 Do you have any publications on PCE and
5 Parkinson's?

6 A I have publications on -- mostly on
7 the -- the mechanisms of neurodegeneration of --
8 Parkinson's degeneration caused by TCE. Some
9 review papers are structured around identifying
10 the relationship between TCE and Parkinson's.

11 Q And those would be included in your CV;
12 correct?

13 A Yes.

14 Q Do you have any publications on PCE and
15 Parkinson's?

16 A I have one paper that is published that
17 has a -- an experiment that includes PCE. It is
18 related to neurodegeneration in an animal model
19 or a cell model, in this case.

20 Q And is that included in your CV?

21 A Yes.

22 Q And is that your only publication on
23 PCE and Parkinson's?

24 A Yes.

25 Q Have you ever published on vinyl

1 chloride?

2 A I don't believe so.

3 Q Have you ever published on benzene?

4 A I don't believe so.

5 Q Have you ever published on DCE?

6 A I don't believe so.

7 Q Do you hold yourself to be an expert in
8 epidemiology?

9 A No.

10 Q Are you qualified to judge the
11 acceptability of epidemiological findings?

12 A I review them regularly as part of my
13 work in the toxicology field. I know the general
14 parameters of what constitutes a -- a robust
15 epidemiological study. So, from that sense, in
16 terms of peer-reviewed research, I do understand
17 what's -- what's a robust epidemiological study.

18 Q But you do not have a formal degree in
19 epidemiology?

20 A No.

21 Q Have you ever practiced as an
22 epidemiologist?

23 A No.

24 Q Can epidemiological studies demonstrate
25 causation?

1 A In the context of Parkinson's disease
2 or just in general?

3 Q In general.

4 MR. TELAN:

5 Object to the form.

6 A I suppose if an epidemiological study
7 showed a clear link between a -- a disease and
8 a -- a source of that disease, it's possible that
9 you could, you know, take, in -- in the context
10 of an entire body of literature, that there is a
11 causative source.

12 MS. PLATT:

13 Q You say in the context of the body of
14 literature. Are you saying that one study -- one
15 epidemiological study alone cannot demonstrate
16 causation?

17 MR. TELAN:

18 Object to the form of the question.

19 A No. I think that the way that I view
20 it as a nonepidemiologist is that, you know, an
21 epidemiology -- an epidemiological study can show
22 in itself that there's a cause. And, you know,
23 in science, we typically view that in the context
24 of everything that's presented.

25 So, in theory, it could, if -- if

1 the -- if the epidemiological study was robust.

2 MS. PLATT:

3 Q And what makes an epidemiological study
4 robust?

5 MR. TELAN:

6 Object to the form.

7 A A properly powered study, the
8 methodology that's described, the --

9 In -- in peer review, you know, you
10 would look at the number of participants, the way
11 that the study has been conducted in terms of --
12 of how the -- how the exposure, in this case --

13 I think that's the context. I'm trying
14 to speak generally.

15 But in this case, in an exposure, you
16 know, how that would be -- how that background
17 was -- was researched.

18 MS. PLATT:

19 Q Would you consider statistical
20 significance?

21 MR. TELAN:

22 Object to the form.

23 A So in epidemiological studies,
24 statistical significance is done a little
25 differently than how we do them in

1 experimental -- how we would do that in
2 experimental studies. So in epidemiological
3 studies, whatever ratio they're -- they're
4 bringing up, whether it's a risk ratio or hazard
5 ratio, typically that's done with a number,
6 followed by a confidence interval and then a
7 p-value.

8 So statistical significance in that
9 case is generally produced with that -- that
10 p-value that's in relation to the ratio and the
11 confidence interval.

12 It's a little different than
13 statistical significance in the way that
14 experimental research provides that, where a, you
15 know, statistical test between two or more groups
16 shows statistical difference over the null
17 hypothesis.

18 Q So in considering whether an
19 epidemiological study is robust, in your opinion,
20 would you consider the statistical significance
21 that is reported in the study?

22 MR. TELAN:

23 Object to the form.

24 A I guess the way I would answer that is
25 it would depend on the -- the value and the

1 numbers that are presented there.

2 So statistical significance, like I
3 just mentioned, in an epidemiological study is
4 based on the p-value, the -- the risk ratio or
5 odds ratio, and then the confidence interval.
6 And, so, where you get statistical significance
7 from that is -- is based on -- on those numbers.

8 So it's hard -- it's hard for me to --
9 to talk in the abstract about a statistical
10 significance of a number that is, you know, sort
11 of -- that I'm not looking at. Right? Because
12 in experimental research, which is my research
13 area, we talk about statistical significance from
14 a viewpoint of where you set your power a priori
15 before you run the test. And, so, you know,
16 that's -- I don't -- I don't sort of -- I don't
17 sort of view those two terminologies the same.

18 So statistical significance in an
19 experimental study where we look at power and
20 statistical significance is different than in an
21 epidemiological study where you look at an odds
22 ratio and the confidence interval.

23 MS. PLATT:

24 Q In epidemiological findings, what is
25 the difference between a showing of an

1 association and showing of causation?

2 MR. TELAN:

3 Object to the form.

4 A I don't know that I can answer that
5 specifically. The way that I typically read an
6 epidemiological study is I look at the --
7 whatever odds or risk ratio is provided, the
8 confidence interval, and the p-value associated
9 with that.

10 So I don't know that I would be able to
11 give you an answer for the difference between
12 association and causation per se just based on --
13 on a broad question like that.

14 MS. PLATT:

15 Q Okay. Do you have a formal degree in
16 neuroscience?

17 A No.

18 Q Do you hold yourself out to be an
19 expert in neuroscience?

20 A In the subset of neuroscience of
21 Parkinson's neurotoxicity.

22 Q You do hold yourself out to be an
23 expert?

24 A In that, yes, in that subset.

25 Q And what is your basis for being expert

1 in neuroscience in the subset of Parkinson's
2 disease?

3 A Yes. I've trained in neurotoxicology
4 as it relates to Parkinson's disease in my
5 graduate work, in my postdoctoral work, and I
6 conduct that research now in my own lab at UAB.
7 I publish papers in this research field,
8 Parkinson's disease, neurotoxicology, as well as
9 neuroscience journals. I review papers in those
10 same fields, and I serve as a peer reviewer on
11 grant reviews for those -- for both NINDS, which
12 is Neurologic Disease -- National Institute of
13 Neurologic Disease and Stroke, NIEHS, National
14 Institute of Environmental Health Sciences, a
15 number of Parkinson's-related foundations, such
16 as Parkinson's Foundation, Michael J. Fox
17 Foundation, American Parkinson's Disease
18 Association. And the students in my lab that I
19 train are in -- most of them are in formal
20 neuroscience programs.

21 Q Anything else qualify you as an expert
22 in that?

23 A I think that -- I think I've covered
24 most of it. I guess I would say I also have --
25 I've been funded in the field that -- that I

1 specified. So in the broad sense of
2 neuroscience, under the heading of Parkinson's
3 neurotoxicity, that's where most of my grant
4 funding comes from.

5 I think that's probably a fair
6 summation.

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

9 A No.

10 Q And Parkinson's disease in humans is
11 diagnosed based off motor functioning; correct?

12 A The clinical signs of Parkinson's are
13 based off of a motor test.

14 Q And, so, neurologists typically perform
15 a physical evaluation to look for symptoms such
16 as bradykinesia, tremors, and rigidity; correct?

17 A Yes. Those are some of the movement
18 parameters that a clinical neurologist would
19 perform.

20 Q I want to turn to Exhibit Number 2,
21 your report, page 2. The second sentence under
22 executive summary --

23 A Uh-huh.

24 Q -- you state "the key neuropathological
25 hallmarks of PD are, number 1, the loss of

1 dopaminergic neurons within the substantia nigra
2 (SN) and their terminal projections to the
3 caudate" --

4 Putamen?

5 A Putamen.

6 Q -- "putamen, also called the striatum,
7 and, number 2, the accumulation of Lewy bodies,
8 which are protein inclusions within neurons
9 comprised of aggregated alpha-synuclein" and then
10 abbreviated aSyn. "A third hallmark,
11 neuroinflammation, is considered to be both a
12 consequence of neuron death and dysfunction, as
13 well as a contributor to the pathogenesis and
14 progression of PD."

15 Did I read that correctly?

16 A Yes.

17 Q Neurologists do not measure the loss of
18 dopaminergic neurons within the substantia nigra
19 and their terminal projections; correct?

20 A In living patients, that is not a
21 measurement that can be made.

22 Q Neurologists do not measure, in living
23 patients, the accumulation of Lewy bodies;
24 correct?

25 A Lewy bodies are -- cannot also be

1 measured in living patients. That's correct.

2 Q Neurologists do not measure the
3 accumulation of Lewy bodies --

4 I'm sorry.

5 Neurologists do not measure
6 neuroinflammation in living patients; correct?

7 A There have been studies where, like,
8 blood or CSF has been drawn and inflammatory
9 markers have been measured. So that would be
10 considered a type of inflammation.

11 If you're talking specifically about in
12 the brain, we cannot take brain samples from
13 living patients to measure what would be
14 considered neuroinflammation from a clinical
15 neurologist.

16 Q And, then, in the next paragraph on
17 page 2, the second sentence, you state "Thus,
18 most PD is considered idiopathic, a term
19 suggesting that the underlying cause is
20 spontaneous and does not originate from inherited
21 mutations alone."

22 Is that correct?

23 A Yes. So, traditionally, idiopathic has
24 been used to coin any Parkinson's disease case
25 that physicians deemed not to be from a heritable

1 source specifically. In --

2 As I state here, it's not from
3 inherited mutations alone, indicating that there
4 is --

5 So in Parkinson's, there is exogenous
6 and endogenous risk for disease. The mutations
7 alone would represent that endogenous risk, and
8 then there's exogenous risk.

9 So the way that, as a
10 neurotoxicologist, I talk about idiopathic
11 Parkinson's -- I just said this as at a big
12 Parkinson's meeting a few weeks ago -- what we
13 call idiopathic or sporadic Parkinson's disease,
14 to put it simply, is a combination of gene and
15 environment interaction.

16 Q And I did read that correctly; correct?

17 A Yes.

18 Q And this is the report that you
19 prepared in this litigation. It's Exhibit Number
20 2. Correct?

21 A Yes.

22 Q And this is your complete report;
23 correct?

24 A Yes.

25 Q If you turn to page 11 of your report,

1 that is your signature at the bottom of the page;
2 correct?

3 A Yes.

4 Q Does this report contain all the
5 opinions you have formed to date in this case?

6 A This contains all the opinions on the
7 published literature that I have.

8 Q That you have formed to date --

9 A Yes.

10 Q -- in this case?

11 A Yes.

12 Q Are you planning to form any additional
13 opinions in this litigation?

14 A No.

15 Q What was the scope of your assignment?

16 A I was asked to talk about the -- mostly
17 the mechanisms or the mechanistic data of how TCE
18 can cause dopaminergic neurotoxicity as well
19 as -- as neurodegeneration.

20 Q On page 1, near the bottom, you have
21 "Questions Presented." Do you see that?

22 A Uh-huh.

23 Q It states "I was asked to evaluate
24 evidence whether TCE exposure could cause the
25 neuropathology that underlines [sic] Parkinson's

1 disease and Parkinson's risk."

2 Is that -- did I read that correctly?

3 A Yes.

4 Q And does that sum up the scope of your
5 assignment?

6 A Yes.

7 Q And at the top of the page, your report
8 is titled "Biological Mechanisms of Dopaminergic
9 Neurotoxicity from Trichloroethylene Exposure."

10 Did I read that correctly?

11 A Yes.

12 Q You opined on TCE and Parkinson's
13 disease; correct?

14 A Yes.

15 Q And you reviewed the literature to
16 opine on TCE and Parkinson's disease; correct?

17 A Yes.

18 Q Your opinions are limited to TCE and
19 Parkinson's disease; corrects?

20 A Mostly. I reviewed literature that
21 included other solvents as well. And I do
22 include, on page 10, a discussion on structurally
23 similar chlorinated solvents to TCE.

24 Q And, in your report, do you state an
25 opinion related to PCE and Parkinson's disease?

1 A I don't think directly I have an
2 opinion related to that in there.

3 Q And turning back to page number 1, you
4 have a summary of opinions at the bottom of the
5 page.

6 A Uh-huh.

7 Q Do you see that?

8 A Yes.

9 Q And is this the expert opinion you
10 intend to offer in this litigation?

11 A Yes.

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

14 A I don't believe so.

15 Q Your report is a complete statement of
16 all opinions that you will express and the basis
17 and reasons for them; correct?

18 A Yes.

19 Q You did not offer an opinion on benzene
20 and Parkinson's disease; correct?

21 A Correct.

22 Q And you did not offer an opinion on
23 vinyl chloride and Parkinson's disease; correct?

24 A Correct.

25 Q And you did not offer an opinion on DCE

1 and Parkinson's disease; correct?

2 A Correct.

3 Q If you turn to page 12 of your report,
4 page 12 is titled "References." Correct?

5 A Yes.

6 Q Page 12 through 17 lists --

7 Does page 12 through 17 list all facts
8 or data you considered in forming your opinions?

9 MR. TELAN:

10 Object to the form.

11 A So these are the references that I
12 cited explicitly. Because I spend my career
13 reading papers on neurotoxicity and Parkinson's
14 disease, there's probably a body of literature
15 that exists that forms sort of the basis or
16 foundation of my knowledge in this field. I
17 read, you know, thousands of papers a year.

18 So in terms of how I view this field as
19 a whole, there are many different papers that,
20 you know -- or published literature that might
21 make up some of -- of how I formed the foundation
22 for my understanding of these concepts.

23 The papers that are represented in my
24 reference list are what I've been -- I've used as
25 a direct relationship to the data or the

1 information I cited in this report.

2 MS. PLATT:

3 Q I'm gonna go ahead and mark Exhibit
4 Number 4.

5 (DEPOSITION EXHIBIT NUMBER 4
6 WAS MARKED FOR IDENTIFICATION.)

7 MS. PLATT:

8 Q Do you recognize this document?

9 A This specific document?

10 Q Yes.

11 A No.

12 Q You've never seen this before?

13 A I have never -- I have not seen this
14 page, this individual page before.

15 Q Do you recognize the two studies listed
16 on this page?

17 A I do recognize the studies.

18 Q Okay. Your counsel produced this last
19 week as a supplemental materials considered list
20 to go along with your report. Did you approve
21 this going out?

22 A I did receive these materials.

23 Q Did you approve them sending a list
24 saying that you considered these in writing your
25 report?

1 MR. TELAN:

2 Object to the form of the question.

3 A I don't know that I specifically
4 approved anything for -- in terms of materials
5 considered in writing this -- in this report.

6 MS. PLATT:

7 Q Did you consider these two studies in
8 writing your report?

9 A So this is just one study. It's the
10 same -- it's the same study, and then one's a
11 supplementary material, just to be --

12 Q Uh-huh.

13 A Is that -- I just want to make sure I
14 was answering the question correctly.

15 Q Yes. Let me rephrase it.

16 A Okay.

17 Q Did you consider this study and the
18 supplemental material in writing your report?

19 A No.

20 Q When did you receive this study and the
21 supplemental material?

22 A I was sent -- I was -- I was sort of
23 formally sent this, I believe it was, last week.
24 So that would have been somewhere between
25 April -- maybe 6th through the 9th. But I have

1 read this study previously in my own review of
2 the literature.

3 Q And your report in this case was
4 submitted on December 8th, 2024; correct?

5 A Correct.

6 Q And, so, you received these studies
7 from counsel -- what? -- four months after you
8 submitted your report? Over four months after
9 you submitted your report?

10 MR. TELAN:

11 Object to the form.

12 A That's when it was specifically sent to
13 me as an -- as sort of additional material.

14 MS. PLATT:

15 Q Okay. And, so, these -- this study and
16 the supplemental material are not cited in your
17 report; correct?

18 A I don't think so. I don't see it in my
19 reference list here from the report that's
20 printed out.

21 Q Okay. Why did you not consider this
22 study and the supplemental material in writing
23 your report?

24 MR. TELAN:

25 Object to the form.

1 A So the main reason is because my report
2 is based on the mechanisms of neurodegeneration
3 caused by TCE exposure. The study that -- and
4 its supplemental material that's listed here is
5 an occupational exposure to organic solvents
6 epidemiological study. And while I do read and
7 keep up to date with epidemiological literature,
8 the majority of what I produced in this report
9 has to do with mechanism, biological mechanism.
10 And that was not tested in -- in that -- in this
11 study that's represented in that -- in that
12 citation.

13 MS. PLATT:

14 Q Going back to your references on page
15 12 of your report, are there any materials you
16 considered but did not --

17 Sorry. Let me start over.

18 Are there any materials you considered
19 in writing your report that you did not include
20 on this reference sheet?

21 MR. TELAN:

22 Object to the form.

23 A So, again, because I am, you know, a
24 researcher in this field and I review and read,
25 again, probably thousands of papers a year, there

1 are papers on Parkinson's disease, on specific,
2 you know, types of Parkinson's research that
3 build -- build a foundation of my understanding
4 of this disease that may not be explicitly
5 considered in the report in terms of how I view
6 Parkinson's neurodegeneration.

7 But in terms of the specific
8 relationship between the mechanisms that I talk
9 about about TCE and mechanisms of
10 neurodegeneration as they relate to -- to
11 Parkinson's disease, I -- I believe that all of
12 the references here are represented in terms of
13 what I considered.

14 I can't preclude that, you know, I
15 didn't -- have read a study over the many years
16 I've been in this field that does not form, you
17 know, some kind of basis for biologic disease,
18 you know, that informs myself as a Parkinson's
19 researcher.

20 Q Are there any -- are there any articles
21 that you did not include in this list but you
22 considered and were not consistent with your
23 opinion?

24 A I don't -- I don't know that I
25 explicitly left anything out because I considered

1 it and deemed it to not be part of -- you know,
2 part of the -- the mechanism data that I wrote up
3 here. In the case of, you know, certain --

4 Like, for example, the Sallmén study,
5 it, in my opinion, wasn't related closely enough
6 to the type of -- of report I was writing on
7 biological mechanism of disease.

8 So there might be studies out there
9 that were sort of out on the fringe of how they
10 related to this mechanism of Parkin- -- of
11 Parkinson's neurodegeneration. But I can't say
12 that I left something out specifically because
13 I -- I reviewed it and thought that it, you know,
14 was somehow not related to biological mechanisms,
15 if that makes sense.

16 Q Uh-huh.

17 Did you review any other expert reports
18 in this litigation?

19 A No.

20 Q Did you review any of the water
21 modeling reports?

22 A I have seen those, yes.

23 Q Which reports did you review?

24 A The ATSDR 2017 report on water modeling
25 in -- at Camp Lejeune.

1 Q And that's a public report; correct?

2 A Correct.

3 Q That's not a report by one of
4 plaintiffs' experts; correct?

5 A Right.

6 Q Did you review any water modeling
7 reports by the plaintiffs' experts?

8 A No.

9 Q Did you review any of the water
10 modeling reports by the United States' experts?

11 A No.

12 Q Did you review any of the general
13 causation reports by plaintiffs' experts?

14 A No.

15 Q Did you review any of the general
16 causation reports by the United States' experts?

17 A No.

18 Q You didn't review Dr. Miller's report?

19 A I have not seen it.

20 Q Did you review Dr. Cannon's report?

21 A I have not seen it.

22 Q Dr. Byrd's report?

23 A No.

24 Q Dr. "Bo-Me's" (phonetic) report?

25 MR. MICELI:

1 Boehm.

2 MS. PLATT:

3 Q Boehm?

4 Dr. Freeman's report?

5 A No.

6 Q Or Dr. Costa's report?

7 A No.

8 Q Did anyone assist you in writing your
9 report?

10 A No.

11 Q Are you aware that the other experts
12 for the plaintiffs considered vinyl chloride and
13 benzene in their opinions?

14 MR. TELAN:

15 Object to the form of the question.

16 A I was not aware of that.

17 MS. PLATT:

18 Q Were you asked to evaluate vinyl
19 chloride and benzene in writing your report?

20 A No.

21 Q Do you know why you were not asked to
22 evaluate these chemicals?

23 MR. TELAN:

24 Object to the form.

25 A I don't know why.

1 MS. PLATT:

2 Q Can you please describe the methodology
3 you used in forming your opinions?

4 A Yes. So the way that I -- the
5 methodology I used in this is kind of how I
6 would -- how I would write what would be
7 considered, like, a review for the mechanisms
8 of -- of neurodegeneration from -- from TCE
9 exposure.

10 And, so, the first thing that I would
11 do is to review or re-review the literature, the
12 published literature, on mechanisms of
13 neurodegeneration from TCE exposure.

14 And then, from there, I would -- I go
15 sort of system by system and I identify the
16 extant literature on Parkinson's disease
17 pathology in the brain, how TCE exposure can
18 produce or replicate that type of Parkinson's
19 pathology in the brain, and then go into more of
20 the cellular and molecular mechanisms based,
21 again, on the published literature. And, so,
22 most of that comes from doing -- using PubMed as
23 a source to pull peer-reviewed published
24 literature.

25 And, then, in reviewing that

1 literature, I often will look at outcome measures
2 and statistical power effect size, as well as
3 limitations of that data, in order to come up
4 with sort of the final report that -- that I have
5 on paper.

6 Q You mentioned that you use PubMed to
7 find articles; correct?

8 A Yes.

9 Q Did you use any other databases to find
10 articles?

11 A I probably have also used Google
12 Scholar. Most of the literature that I -- that I
13 reviewed in this comes from PubMed, but I can't
14 preclude there are other search engines, like
15 Google Scholar that --

16 Or I think some of those -- those
17 reports, like the ATSDR report, I'm not sure it's
18 indexed on PubMed. So it might have come from,
19 like, another government repository that's not
20 online.

21 Q Can you turn to page 11 of your report?
22 In the last paragraph, second sentence,
23 you state "I conducted this review using an
24 unbiased scientific approach and relied on
25 published literature indexed in PubMed from the

1 NIH National Library of Medicine using keyword
2 search terms such as trichloroethylene,
3 tetrachlorethylene, Parkinson's disease,
4 dopaminergic, alpha-synuclein, neuroinflammation,
5 lysosomal, mitochondrial, complex I, oxidative
6 stress, microglia, inhalation, and cytochrome
7 P450."

8 Did I read that correctly?

9 A Yes.

10 Q Are those -- is that a complete list of
11 the search terms you used in your literature
12 search?

13 A So, as I note, it's a keyword search
14 term such as. So it's -- it's the majority of
15 the types of keywords that -- that I would look
16 for, but it doesn't preclude that there weren't a
17 few search terms that, you know, may have been,
18 again, related to sort of my general basis of
19 understanding of -- of Parkinson's disease in
20 terms of -- of pathology in terms of
21 neurodegeneration.

22 Q What would those other search terms be?

23 A I can't say specifically that there's
24 any specific search term that, you know, is -- is
25 in relation to -- to this report. It's more,

1 again, from the body of literature that -- that I
2 have in terms of Parkinson's disease that I
3 review on sort of a daily basis throughout --
4 throughout my -- throughout my research.

5 Q Did you come up with these search terms
6 yourself?

7 A Yes.

8 Q Is it common practice to describe
9 inclusion and exclusion criteria as part of a
10 literature research? Sorry. A literature
11 search?

12 A Inclusion and exclusion criteria, could
13 you give an example of what that means?

14 Q In a typical literature search, do you,
15 as you see it published, do you typically see the
16 inclusion and exclusion criteria included in the
17 methodology of that article?

18 A For experimental work, inclusion and
19 exclusion criteria are not considered the same
20 way it would be in an epidemiological study. So
21 for the majority of what I do research on and
22 what I read in terms of mechanisms of
23 neurotoxicity, inclusion/exclusion criteria would
24 not be -- would not be part of those studies
25 because these are -- are experimental studies

1 where we don't use terminology like that. We use
2 different terminology in terms of setting up an
3 experimental study.

4 So that's not necessarily something
5 that we -- that I would go through in a published
6 paper for an experimental study and say if they
7 don't list the specific term inclusion or
8 exclusion criteria, I -- that that study would
9 not be valid.

10 Q You did review epidemiological studies
11 in your report; correct?

12 A I did review them.

13 Q And were these search terms used for
14 finding those epidemiological studies?

15 A Yes.

16 Q And, so, typically, as you said, in an
17 epidemiological study, you would see the
18 inclusion and exclusion criteria in the
19 methodology of a literature search; correct?

20 MR. TELAN:

21 Object. Object to form.

22 A I believe so.

23 MS. PLATT:

24 Q And you did not include those terms in
25 your report; correct?

1 A Correct.

2 Q Did you perform a systematic review of
3 all literature that came up from these searches?

4 A I don't think you would -- I don't
5 think you would refer specifically to what this
6 biological mechanism explanation would be
7 considered a -- a systematic review. This is,
8 again, the -- I'm providing information on
9 mechanisms of disease from both the published
10 literature as well as my own -- my own research.
11 So it's not a systematic review in the sense
12 that --

13 There's certain criteria have to be
14 applied to systematic reviews, and typically
15 you'd form, like, a table of all of the different
16 papers and you'd go through each of the citations
17 kind of individually. And, so, that's not --
18 that's not part of -- of the review that I
19 provided here.

20 Q And, so, your report is not a
21 systematic review of the literature; is that
22 correct?

23 A I would not call it systematic.

24 Q Are there studies that you excluded
25 from your review?

1 A I didn't specifically exclude anything.
2 There may have been literature that, when I was
3 going through and writing this, I didn't deem it
4 to be specifically important in the relationship
5 of each mechanism that I was talking about. But
6 I -- I wasn't necessarily going through and
7 specifically excluding something, other than if
8 it -- if --

9 Something would have been not included
10 if it was not directly related to the mechanisms
11 that I was discussing. So -- so I didn't -- I
12 didn't go through and specifically exclude
13 anything when I was doing this.

14 Q Are you aware of Dr. Bove's 2014 paper
15 titled "Evaluation of mortality among Marines and
16 Navy personnel exposed to contaminated drinking
17 water at USMC base Camp Lejeune: A retrospective
18 cohort study"?

19 A Yes.

20 Q Are -- and you -- are you aware of
21 Dr. Bove's 2014 evaluation of the civilian
22 workers at Camp Lejeune?

23 A Yes.

24 Q And you did not include that study in
25 your report; correct?

1 A Correct.

2 Q Is there a reason you excluded it?

3 A So, again, I didn't exclude anything
4 per se. I mostly just -- because I'm focused on
5 mechanism specifically in this report and
6 because, you know, I have mostly involved with
7 experimental studies, exposure to TCE, the
8 relationship of that in terms of neurotoxicity
9 and neurodegeneration, I didn't find that, you
10 know, it was important for me to go through and
11 extensively characterize those epidemiological
12 studies that are -- that are in relationship to
13 TCE and Parkinson's at -- specifically at
14 Camp Lejeune.

15 Q And you did include some
16 epidemiological studies in your report; correct?

17 A Yes.

18 Q Are you aware of Dr. Bove's 2024 study
19 entitled "Evaluation of Mortality Among
20 Marines/Navy Personnel and Civilian Workers
21 Exposed to Contaminated Drinking Water at USMC
22 Base Camp Lejeune: A Cohort Study"?

23 A Yes.

24 Q And you did not include that in your
25 report; correct?

1 A Correct.

2 Q Is there a reason why you excluded it?

3 MR. TELAN:

4 Object to the form.

5 A Yes. Again, I'm not necessarily
6 excluding anything. The -- you know, the --
7 those studies that Bove published on hazard --

8 Or, sorry. Mortality. I think it's
9 hazard ratio that's reported in those studies.

10 MS. PLATT:

11 Q Uh-huh.

12 A Those are not particularly helpful for
13 me in terms of -- of assessing mechanism of
14 Parkinson's disease, again, for the reasons I
15 mentioned previously in terms of mechanism.

16 It's also hard, you know, to measure
17 Parkinson's disease from a mortality study
18 because people don't die of Parkinson's disease.
19 They die with Parkinson's disease.

20 So in terms of -- of characterizing,
21 you know, hazard ratio or mortality, as a person
22 who's not, you know, making significant
23 statements on, you know, the broad sense of
24 epidemiology in this case, it didn't -- it wasn't
25 something that I deemed to be specifically

1 important to the mechanisms that I was -- that I
2 was focusing on in this.

3 Q And are you aware of ATSDR's morbidity
4 study from 2018 titled "Morbidity Study of Former
5 Marines, Employees, and Dependents Potentially
6 Exposed to Contaminated Drinking Water at US
7 Marine Corps Base Camp Lejeune"?

8 A Yes.

9 Q And you did not include that study in
10 your report; correct?

11 A I don't think so.

12 Q Is there a reason why you did not
13 include that report in your study? Sorry. Why
14 you did not include that study in your report?

15 A Again, for a similar reason. I'm
16 focused more specifically here on mechanism.
17 And, so, I'm really drawing my -- you know, the
18 basis of what I'm drawing from is -- has to do
19 with the -- the mechanistic loss of neurons or
20 Parkinsonian pathology in the brain from TCE
21 exposure.

22 And, so, while, you know, the
23 literature, the broad literature, on exposure to
24 TCE as it relates in a population-wide scale is
25 something I'm familiar with, it's not something

1 that necessarily weighs into how I understand,
2 you know, how TCE is actually physically causing
3 neuron loss in the brain. So it's not something
4 that I -- that was important in terms of how I
5 talked about mechanism in this report.

6 Q We talked a little bit about your
7 methodology in this report, but I want to direct
8 your attention to page 2 of your report. At the
9 top of the page, you have a paragraph starting
10 out "methodology." Do you see that?

11 A Yes.

12 Q "The method they use to assess these
13 conclusions applied the same standards as I would
14 use in preclinical research and peer review.
15 These included reviewing robust and rigorous
16 peer-reviewed literature and epidemiological and
17 experimental studies related to TCE exposure and
18 Parkinson's disease risk, assessing the
19 statistical significance of reported outcome
20 measures in relation to the statistical power and
21 effect size, and weighing the limitations of
22 published data."

23 Did I read that correctly?

24 A Yes.

25 Q Is this a good summary of the method in

1 your report?

2 A Yes.

3 Q And your method was to assess the
4 statistical significance of reported outcome
5 measures; correct?

6 A Sorry. Would you mind repeating that?
7 I got distracted.

8 Q Your method was to assess the
9 statistical significance of reported outcome
10 measures; correct?

11 A Yes.

12 MR. TELAN:

13 Object to the form.

14 A Sorry.

15 MS. PLATT:

16 Q And you did so in relation to the
17 statistical power and effect size and weighing
18 the limitations of published data. Correct?

19 A Yes.

20 Q How --

21 Did you weigh these factors against
22 each other?

23 A Not particularly. You typically
24 wouldn't -- you typically don't weigh them
25 against each other. You know, limitations is --

1 You know, so weighing limitations or
2 that terminology is sort of looking at the study
3 as a whole. And, then, statistical power and
4 effect size is something that you would compare
5 within itself, but not necessarily -- you don't
6 necessarily weigh the -- like, those different
7 parameters, or I wouldn't weigh those parameters
8 against each other in that context, if I
9 understand you correctly.

10 Q You do not weigh one parameter greater
11 than other parameters?

12 A Typically not.

13 Q How did you measure statistical power
14 and effect size?

15 A Based on the statistical power and
16 effect size that was reported in the studies.

17 Q And how did you weigh the limitations
18 of the published data?

19 A In many cases, limitations were often
20 discussed in each publication. Or if it was a
21 limitation that --

22 You know, sometimes you can -- you can
23 come up with your own limitations as you're
24 reading an article.

25 Q And those are the limitations that you

1 would take into consideration in writing your
2 report?

3 A Yes.

4 Q You did not apply the Bradford Hill
5 framework; correct?

6 A Correct.

7 Q Are you familiar with the Bradford Hill
8 criteria?

9 A I'm familiar with it.

10 MR. TELAN:

11 Object to form.

12 MS. PLATT:

13 Q The Bradford Hill criteria includes a
14 biological gradient also known as dose-response;
15 correct?

16 A Yes.

17 MR. TELAN:

18 Object to the form.

19 MS. PLATT:

20 Q When do -- when you do a dose-response
21 analysis and the relationship between the dose
22 and the chemical shows an increased response,
23 that supports the finding of general causation;
24 correct?

25 MR. TELAN:

1 Object to the form.

2 A So I'm not sure I can give an explicit
3 answer on that in terms of causation. I would
4 apply most of this based on experimental data.
5 So cause and effect there might be more of, like,
6 a biological response in terms of -- of toxicity
7 versus causation.

8 There's also cases --

9 Or maybe "case" isn't the right word.

10 There are also examples of where a -- a
11 dose-response is not necessarily related
12 specifically to the type of -- you know,
13 specifically to pathology that's -- that's
14 correlated linearly with what would be
15 considered, like, disease.

16 You can have biological responses in
17 different -- in different dose-response curves
18 that don't necessarily correlate just with, you
19 know, the -- just linearly with more exposure
20 equals more disease. There are cases where
21 that -- that can happen.

22 Q Are you familiar with Pearl's ladder?

23 A I've heard of it, but it's been a long
24 time.

25 Q Can you define it?

1 A No.

2 Q Okay. So you did not apply Pearl's
3 ladder to your report; correct?

4 A No.

5 Q I want to go to page 2 of your report.
6 About halfway down, you have a sentence that
7 starts "in relation."

8 A Uh-huh.

9 Q Do you see that?

10 "In relation to this case, both
11 epidemiological and experimental evidence support
12 a link to PD risk from exposure to chlorinated
13 organic solvents such as trichloroethylene
14 (TCE)."

15 Did I read that correctly?

16 A Yes.

17 Q And then you cite to some footnotes
18 there, numbers 1, 3, and 12 through 17. Is that
19 correct?

20 A Those are references, I think. Yes.

21 Q And they correspond to the references
22 in your report.

23 A Yes.

24 Q I want to start with reference number
25 12.

1 A Uh-huh.

2 Q I'm gonna go ahead and mark that as
3 Exhibit Number 5.

4 (DEPOSITION EXHIBIT NUMBER 5
5 WAS MARKED FOR IDENTIFICATION.)

6 A Can I request a bathroom break really
7 quickly before we get into this?

8 MS. PLATT:

9 Q Sure. Let's go ahead and take a
10 bathroom break.

11 VIDEOGRAPHER:

12 We are going off the record. The time
13 is 10:35.

14 (OFF THE RECORD.)

15 VIDEOGRAPHER:

16 Back on the record. The time is 10:41.

17 MS. PLATT:

18 All right. We're back on the record.
19 And just for clarity of the record, I want to
20 note that Zina Bash for the Plaintiffs'
21 Leadership Group has now entered through the Zoom
22 link, and she is participating.

23 Q Dr. De Miranda, before we took a break,
24 I handed you Exhibit Number 5, and this is
25 Dr. Goldman's 2012 study titled "Solvent

1 Exposures and Parkinson's Disease Risk in Twins."

2 Do you see that?

3 A Yes.

4 Q And you relied on this study in your
5 report; right?

6 A Yes.

7 Q And the sample for the study included
8 198 twins; correct?

9 A It was --

10 Let me -- let me double-check. Because
11 I know there's 99 PD discordant twins. 198 PD
12 discordant pairs. Yes.

13 Q So 99 twin pairs; correct?

14 A And -- yeah. Individual twins with PD,
15 119; individual twins without PD and by both
16 twins in 99 pairs.

17 Q And the study subjects were World War
18 II veterans; correct?

19 A Yes.

20 Q And the study relied on self-reporting
21 of these veterans; correct?

22 A I believe so. Let me make sure.

23 Sorry. Let me just make sure I see
24 self-reporting in here.

25 It was a questionnaire, a

1 questionnaire.

2 Q And these questionnaires were completed
3 by either the individual themselves or proxy
4 informants; correct?

5 A Make sure.

6 Q If you look to the top of the
7 right-hand column, the bottom of that first full
8 paragraph.

9 A On the front page?

10 Q Sorry. On page 777.

11 A So it says, again, "questionnaire."

12 So --

13 Sorry. I'm seeing --

14 On the top of the 777, I'm seeing
15 corresponding 2.5 parts per million, a cumulative
16 special index was calculated for each solvent as
17 the product of exposure intensity hours per year,
18 that paragraph there.

19 Q The next paragraph --

20 A Uh-huh.

21 Q -- the last sentence states "suitable
22 proxy informants were identified for deceased and
23 cognitively impaired subjects scoring less than
24 27 on the education-adjusted Modified Telephone
25 Interview for Cognitive Status."

1 A Yes.

2 Q And, so, back to my original question,
3 these questionnaires were completed by either the
4 individual or a proxy informant if the individual
5 was deceased or cognitively impaired; correct?

6 A Yes.

7 Q And solvent exposures were inferred
8 based upon occupational and hobby history;
9 correct?

10 A Yes.

11 Q For deceased and cognitively impaired
12 subjects, a proxy informant provided the
13 occupational history and hobby history. Correct?

14 A Yes.

15 Q The authors did not have exposure
16 samples from the population. Correct?

17 A Yes.

18 Q And inferring exposure history based on
19 job and hobby histories were collected through
20 self- or deceased or cognitively impaired
21 proxy-reported histories --

22 Sorry. Let me start again.

23 "Inferring exposure based on job and
24 hobby history as collected through self- or
25 proxy-reported histories could lead to exposure

1 misclassification." Correct?

2 MR. TELAN:

3 Object to the form of the question.

4 A There's always going to be risk for
5 misclassification when you look at an occ- -- you
6 know, an occupation or you're doing estimations
7 from -- from occupation or hobby exposure. But
8 there are ways to do it that are more -- that are
9 more robust than others.

10 Q And, in this study, did they do it in a
11 robust way?

12 A So it's better than some because they
13 actually did a questionnaire where they talked
14 about occupational exposure -- or what people
15 were doing in that occupation. And that's where
16 that questionnaire comes in.

17 So just listing strictly by occupation
18 is very hard to understand what a person's doing
19 in terms of their exposure risk. So when a
20 questionnaire is given -- and I don't have the
21 questionnaire in front of me -- sometimes they'll
22 ask questions like what were they doing at that
23 job. And that can help inform a more granular
24 data on what type of exposure a person may have
25 sustained. I assume that's true for hobbies as

1 well.

2 And, so, in terms of -- of assessing if
3 it's robust, that -- that would be one of the
4 ways you would determine if a questionnaire is
5 robust. So if it's just listing strictly based,
6 you know, just occupation, it might be less
7 robust than if it actually goes through and asks
8 more details about what a person did in that
9 occupation.

10 Q And self-reporting could lead to recall
11 bias; correct?

12 A There's always a risk of recall bias in
13 any self-reporting report.

14 Q Data on head injury was also provided
15 through these interviews; correct?

16 A Yes.

17 Q And for deceased and cognitively
18 impaired subjects, a proxy informant provided
19 that data on head injuries; correct?

20 A Yes.

21 Q Twins do not have identical exposures,
22 particularly in adulthood. Correct?

23 A Correct.

24 Q And twins may live in different
25 residential locations that may lead to different

1 exposures; correct?

2 A Yes.

3 Q Goldman 2012 did not have access to
4 other exposure information; correct?

5 A I don't think so.

6 Q The authors did not have information on
7 residential history for each twin; correct?

8 A I'd have to look specifically at that.
9 But I didn't see residential history in part of
10 their analysis.

11 Q The authors inferred the exposure
12 information for each twin; correct?

13 A Yes. It says exposure intensity was
14 coded for each job or hobby as low, medium, or
15 high.

16 Q And that was inferred off of the
17 questionnaire information provided; correct?

18 A Yes.

19 Q TCE and PCE and carbon tetrachloride
20 have been used extensively worldwide; correct?

21 A Yes.

22 Q Confidence intervals provide a measure
23 of precision; correct?

24 MR. TELAN:

25 Object to the form.

1 A "A measure of precision." I don't know
2 that I can give a, like, strong answer for -- for
3 what precision means in that. Again, I'm not an
4 epidemiologist, so bear with me a little bit
5 on -- on some of that.

6 Can you maybe define what precision
7 means in this -- in this case?

8 MS. PLATT:

9 Q Why don't I ask you a different way.
10 Can you define confidence intervals for me?

11 A A confidence interval is the -- it's
12 when you basically go from the lowest --

13 And I'm gonna use the term "risk" here
14 because I'm familiar enough with risk in
15 epidemiology that has to do with exposure.

16 So lowest risk up to the highest risk.
17 And, so, that would be, like, the full spectrum
18 of risk across the population that's been
19 assessed.

20 Q And what does a narrower confidence
21 interval indicate?

22 MR. TELAN:

23 Object to the form of the question.

24 A Um, the way I understand it is that
25 typically a narrow confidence interval indicates

1 that the group that you're assessing is clustered
2 close together. And, so, that would mean that
3 you have people who fall within a value range
4 that's -- that's close to each other.

5 MS. PLATT:

6 Q And what does a wider confidence
7 interval indicate?

8 MR. TELAN:

9 Object to the form of the question.

10 A So, again, my understanding of this is
11 a wider confidence interval indicates that people
12 fall within a wider range of that. So they could
13 fall from low to high risk in that -- in that
14 range.

15 MS. PLATT:

16 Q In interpreting the confidence
17 interval, one should consider if the underlying
18 population is biased; correct?

19 MR. TELAN:

20 Form objection.

21 A "If the underlying population is
22 biased." I don't know if I can answer that
23 specifically.

24 When -- when thinking about a
25 confidence interval --

1 Sorry. Could you repeat that for me
2 just so I can --

3 Q Yeah.

4 A -- make sure I answer that correctly?

5 Q When interpreting a confidence
6 interval, do you consider if the underlying
7 population is biased?

8 MR. TELAN:

9 Object to the form of the question.

10 A Um, I mean, I don't know specifically
11 that, if I'm looking at a confidence interval,
12 I'm looking at population bias. I might look at
13 population bias more in the context of an entire
14 study.

15 Again, I'm not an epidemiologist, so I
16 don't -- I don't traditionally get into things
17 like population bias when I look at a specific
18 factor like a confidence interval unless it's
19 been explicitly stated in that paper or something
20 where they call attention to it, you know, or --
21 or it's been -- it's been discussed in a
22 significant way.

23 So -- so that might partially answer
24 that question and -- and, again, you know, I
25 don't -- I don't generate confidence intervals in

1 my own research, so I can only give you sort of
2 how -- how I -- how I view that.

3 MS. PLATT:

4 Q Uh-huh.

5 Do you find that --

6 Let me start again.

7 A study is less meaningful where the
8 sample of the population is not representative of
9 the underlying population; correct?

10 MR. TELAN:

11 Object to the form of the question.

12 A If a sam- -- if a -- let me repeat
13 that. If a sample of the population is not
14 representative of the general population?

15 MS. PLATT:

16 Q Of the underlying population.

17 A Of the underlying population.

18 I guess it would depend on what
19 "underlying population" meant in that -- in that
20 scenario. So -- so if the underlying
21 population --

22 I guess, if I understand you correctly,
23 the underlying population is the population
24 that's being assessed by the sampling of the
25 epidemiological study.

1 Q Yes.

2 A So if you had --

3 I guess it would determine -- it would
4 depend on what the bias is and what -- what
5 factors make up that. That might be a --
6 something to consider in the -- in the study
7 itself in terms of weighing -- weighing the
8 limitations of that.

9 Q Can you turn to table 3 of Goldman
10 2012?

11 A Uh-huh.

12 Q Which, again, is Exhibit 5.

13 A Uh-huh.

14 Q And if you look at the row for TCE as
15 the solvent --

16 A Uh-huh.

17 Q -- Goldman found a odds ratio for TCE
18 to be 6.1, with a confidence interval of 1.2 to
19 3 -- 33; correct?

20 A Uh-huh.

21 MR. TELAN:

22 Sorry, Elizabeth. Which table are you
23 on?

24 MS. PLATT:

25 Table 3. It's on page 779.

1 MR. TELAN:

2 Thank you.

3 MS. PLATT:

4 Q 1.2 to 33 is a wide confidence
5 interval; correct?

6 MR. TELAN:

7 Object to the form.

8 A It's --

9 I guess "wide" is defined by --

10 I don't know that I could define what a
11 wide confidence interval is per se. It's
12 certainly, you know, it spans from 1 -- 1 to 30;
13 right? So that's --

14 I guess it depends on what the context
15 is. I don't know that I would necessarily say
16 wide --

17 I guess some people might consider
18 wide --

19 I guess it would depend on the context
20 of what you consider a narrow -- a narrow or wide
21 confidence interval.

22 MS. PLATT:

23 Q What do you consider a narrow or wide
24 confidence interval?

25 A Um, I don't really have a numerical

1 value for that. I guess I've never really
2 thought about that before in terms of what's
3 considered narrow or wide.

4 I -- I do know that, you know, you can
5 have --

6 Like I was talking about before, if
7 you're clustered close together, that would
8 probably be considered more narrow, and if
9 they're clustered further apart, that might be
10 considered wide.

11 So in the context of what you're
12 asking, is 1.2 to 33 wide, if you look at the
13 other numbers in this, it's sort of in the middle
14 of the other confidence intervals that are stated
15 in that table 3. But there are confidence
16 intervals that are more narrow than that, and
17 there are some that are wider than that.

18 Q You've relied on this study in writing
19 your report; correct?

20 A Yes.

21 Q Did you take into account this distance
22 of the confidence interval from 1.2 to 33?

23 A I didn't specifically speak to that
24 confidence interval when I -- when I wrote this
25 report.

1 Q Are you familiar with confidence
2 interval ratios?

3 A Not explicitly.

4 Q Could you define them?

5 A No.

6 Q So is it safe to say you have never
7 used a confidence interval ratio to measure the
8 precision of results?

9 A No. I don't -- I don't do that kind of
10 research.

11 Q Do you remember reading any studies
12 that included a confidence interval ratio?

13 A I'm sure I have, but I -- I can't come
14 up with anything off the top of my head.

15 Q Okay. A confidence interval ratio is
16 calculated by taking the higher end of the
17 confidence interval and dividing it by the lower
18 end of the confidence interval.

19 A Okay.

20 MR. TELAN:

21 Object to the form. I don't know that
22 that was a question.

23 MS. PLATT:

24 I didn't ask a question.

25 MS. PLATT:

1 Q So if we take TCE here with a
2 confidence interval and we divide 33 divided by
3 1.2, do you know what that equals?

4 A No.

5 Q I can tell you. 33 divided by 1.2
6 equals 27.5.

7 A Okay.

8 Q Does that sound accurate to you?

9 A I trust you.

10 Q And that would be a large confidence
11 interval ratio; correct?

12 MR. TELAN:

13 Object to the form.

14 A I guess it depends on the context,
15 again. So if you're talking about the context of
16 this table, it looks like it would be somewhere
17 kind of in the middle. But, again, because I
18 don't -- I don't sort of deal in confidence
19 interval ratios, I couldn't make, like, a
20 sweeping statement on that per se.

21 Q And this row we're looking at is only
22 about TCE; correct?

23 A We've just been talking about TCE so
24 far.

25 Q And, so, Goldman 2012 attempted to

1 consider only TCE exposure in this particular
2 row; correct?

3 A Looks like -- like in that row it's --
4 it's just TCE exposure.

5 Q And the sample showed only nine
6 individuals exposed to TCE; correct?

7 A I think it's nine individuals who have
8 Parkinson's exposed to TCE. Is that --

9 I believe that's from the -- from the
10 table.

11 Q Yes. That's a better way to state it.

12 A Okay.

13 Q Nine individuals with Parkinson's
14 disease that were exposed to TCE; correct?

15 A Yes.

16 Q And, then, moving one line up, to
17 PERC --

18 PERC is another name for PCE; correct?

19 A Yes.

20 Q Goldman found an odds ratio for PCE to
21 be 10.5, with a confidence interval of .97 to
22 113. Correct?

23 A Yes.

24 Q When the lower end of the confidence
25 interval is less than one, the increased risk is

1 not statistically significant; correct?

2 MR. TELAN:

3 Object to the form of the question.

4 A I don't -- I don't know how statistical
5 significance is specifically defined by the
6 lower -- the lower end being less than one. The
7 way that I interpret confidence intervals --
8 confidence intervals -- excuse me -- is --

9 I mentioned this earlier. It's to --
10 it's the range at which risk falls along -- in
11 those individuals who are represented. So it
12 goes, in this case, .97 to 113.

13 I don't -- I don't --

14 Again, because I don't do this research
15 specifically, I can't make a statement about how
16 a confidence interval relates specifically to
17 statistical significance of, you know, a -- a --
18 a parameter that's being defined by the study. I
19 sort of take it at face value of what the
20 confidence interval is displayed.

21 Q Part of your methods was to consider
22 statistical significance; correct?

23 A Yes.

24 Q And you relied on this study in your
25 report; correct?

1 A Yes.

2 Q So did you not evaluate statistical
3 significance related to the odds ratio for PERC?

4 MR. TELAN:

5 Object to the form.

6 A Uh --

7 Oops. Excuse me.

8 I used the p-value for statistical
9 significance that's listed in the last column of
10 the table.

11 MS. PLATT:

12 Q And you did not consider the confidence
13 interval, the 95 percent confidence interval?

14 A So in relation to --

15 So -- so in relation to statistical
16 significance, I used the p-value. The confidence
17 interval, as I mentioned, you know, I do assess
18 in terms of -- of -- as it's written, you know,
19 as it's written from the 95 percent confidence
20 interval.

21 Q And we discussed the confidence
22 interval ratio a few moments ago; correct?

23 A Yes.

24 Q If we calculated a confidence interval
25 ratio for PERC, it would be greater than 100;

1 correct?

2 MR. TELAN:

3 Object to the form.

4 A Yes.

5 MS. PLATT:

6 Q And the chart shows that five
7 individuals with Parkinson's disease were exposed
8 to PERC; correct?

9 A Yes.

10 Q A small sample size is a limitation of
11 the study; correct?

12 A Small sample sizes in general can be
13 limitations. Because the study is -- is PD
14 discordant twins, it's often -- it's often sort
15 of defined as being -- as having a little bit
16 more power because they're twins instead of just
17 looking strictly at sample size.

18 Q If you turn to page 781.

19 A Uh-huh.

20 Q At the very bottom of the right-hand
21 column, the study states "the major limitations
22 of the study are in its small sample size, which
23 yielded imprecise exposure estimates, and
24 exposure inferences based on retrospective
25 recall."

1 Did I read that correctly?

2 A Yes.

3 Q The proxy respondent for majority of
4 the sample is a limitation of the study; correct?

5 A It could be.

6 Q Can you turn to page 782? The eighth
7 line down in the left-hand column, "another
8 weakness is reliance on proxy informants for a
9 large proportion of subjects, with a much greater
10 proportion among case subjects."

11 Did I read that correctly?

12 A Yes.

13 Q It is difficult to isolate specific
14 effects of single agents because many work
15 settings involve exposure to multiple agents;
16 correct?

17 MR. TELAN:

18 Object to the form of the question.

19 A Sorry. Did you read that from the
20 paper?

21 MS. PLATT:

22 Q No.

23 A Oh. Would you mind repeating that? I
24 was looking for it in the text.

25 Q Of course.

1 It is difficult to isolate specific
2 effects of single agents because many workplace
3 settings involve exposure to multiple agents;
4 correct?

5 MR. TELAN:

6 Same objection.

7 A I think it could be a challenge. I
8 guess it depends on how -- a little bit on how
9 the surveys are conducted, because sometimes
10 those surveys can do a -- can be better at
11 isolating, like, what a person is working with,
12 for example.

13 I don't know in the context of -- of
14 this study of how those surveys were
15 specifically -- like, how those -- how those --
16 they were structured. I just know kind of in a
17 more general sense that there can be -- it can be
18 difficult with -- with workplace studies to
19 assess single exposures. But if it's done
20 correctly, there are better ways. There are sort
21 of better and worse ways to do it.

22 MS. PLATT:

23 Q If you look at page 782, the
24 second-to-last sentence of the first paragraph on
25 the left-hand column, "another limitation is the

1 difficulty isolating specific effects of single
2 agents because many workplace settings involve
3 exposure to multiple agents."

4 Did I read that correctly?

5 A Yes.

6 Q Did you review the ATSDR's "Assessment
7 of the Evidence for Drinking Water Contaminants
8 at Camp Lejeune and Specific Cancers" in writing
9 your report?

10 A I have read it, yes.

11 Q Did you consider it in writing your
12 report?

13 A I did not --

14 Well, I -- I do -- from the basis of
15 what was at Camp Lejeune, I did consider that.
16 But I don't think I cite it -- I don't know if I
17 cite it specifically.

18 Q I want to go ahead and enter Exhibit
19 Number 6.

20 (DEPOSITION EXHIBIT NUMBER 6
21 WAS MARKED FOR IDENTIFICATION.)

22 MS. PLATT:

23 Q Exhibit number 6 is ATSDR's Assessment
24 of the Evidence; correct?

25 A Yes.

1 Q And if you turn to page 99 --

2 Let's go back to page 96. Do you see
3 that's the start of the Parkinson's disease
4 review section?

5 A Yes.

6 Q And do you see in the fifth row it
7 states "Goldman 2012 Prevalent (Alive)"? That
8 is --

9 Sorry. Do you see that?

10 A Yes.

11 Q That is the study we are discussing;
12 correct?

13 A Yes.

14 Q And, then, turn to page 99. There's a
15 paragraph that starts out "conclusion."

16 A Uh-huh.

17 Q The third sentence states "because only
18 two studies have focused on TCE exposure
19 (Goldman, et al., 2012, and Bove, et al., 2014b),
20 the epidemiological evidence for causation for
21 TCE and Parkinson's disease is very limited."

22 Did I read that correctly?

23 A Yes.

24 Q And that cite to Goldman, et al., 2012,
25 is the same study that we are referring to;

1 correct?

2 A Yes.

3 Q And you were aware of this report when
4 you were drafting your own report; correct?

5 A Yes.

6 Q And ATSDR concludes that the evidence
7 is not suf- -- the evidence for TCE and
8 Parkinson's disease did not meet the sufficient
9 standard; correct?

10 MR. TELAN:

11 Object to the form of the question.

12 A That's what it says in this -- in this
13 paragraph, yes.

14 MS. PLATT:

15 Q All right. And if you turn back to
16 page 6, do you see the header "Classification
17 Scheme Categories"?

18 A Yes.

19 Q And this is the classification scheme
20 that ATSDR applied to this report; correct?

21 A I believe so.

22 Q And the first category is sufficient
23 evidence for causation; correct?

24 A Yes.

25 Q And the second one is equipoise and

1 above evidence for causation; correct?

2 A Yes.

3 Q And, turning to page 7, the third
4 category is below equipoise evidence for
5 causation; correct?

6 A Yes.

7 Q And the final one is evidence against a
8 causal relationship; correct?

9 A Yes.

10 Q Have you ever published a scientific
11 article using this standard?

12 A My -- my published research does not
13 include that specific language.

14 Q Have you ever written an expert report
15 using this standard?

16 A Yes.

17 Q And in what case have you done that?

18 A I previously used language like this in
19 a case involving a person with Parkinson's
20 disease from occupational exposure to TCE.

21 Q And what was that matter?

22 A The --

23 Do you want, like, the name of the --

24 Q Yes.

25 A Okay. Sorry.

1 That was -- the -- the plaintiff was
2 named O'Leary, and I believe it was against
3 Boeing and/or Dow Chemical Company.

4 Q And in what year did you write that
5 report?

6 A 2024.

7 Q And what language specifically from
8 this classification scheme did you use?

9 MR. TELAN:

10 Object to the form of the question.

11 A So I -- in terms of -- of this specific
12 classification scheme, the type of language I
13 used in previous reports is more likely than not,
14 which I think is probably slightly different than
15 how it's written here, or equally likely than
16 not, which I think is represented by equipoise
17 and above. And, so, those are the terminologies
18 that I've previously used in reports.

19 MS. PLATT:

20 Q And in the O'Leary matter, did you use
21 more likely than not?

22 A I believe I did.

23 Q Did you use equally as likely as not?

24 A I don't think so. I'd have to -- I'd
25 have to check to be sure. But I don't recall

1 using that in that specific case.

2 Q And where in ATSDR's classification
3 scheme does it use the term "more likely than
4 not," or the phrase "more likely than not"?

5 A I don't specifically see that in the
6 classification scheme.

7 Q And have you written an expert report
8 using the phrase "equally as likely as not"?

9 A I think in this biological report I do
10 use the words "equally likely as not" in terms of
11 what I was instructed to consider. I think that
12 shows up in -- in that report. Yes, it is. It's
13 on page 1.

14 Q And you're referring to your report in
15 this case, in the Camp Lejeune Water Litigation
16 case.

17 A Yes.

18 Q Other than the Camp Lejeune water
19 litigation report, have you used equally as
20 likely as not in a report for litigation?

21 A I don't believe so.

22 Q Have you ever read a scientific article
23 that used this category scheme used by ATSDR?

24 A Not to my knowledge.

25 Q Is this the typical vernacular used in

1 scientific literature?

2 A No.

3 Q Is it the typical vernacular used in
4 toxicology?

5 A Typically not.

6 Q The typical vernacular used in
7 Parkinson's disease literature?

8 A No.

9 Q In going back to Goldman 2012, ATSDR
10 reviewed Goldman 2012 and concluded the evidence
11 for TCE and Parkinson's disease did not meet the
12 sufficient standard; correct?

13 A In this report?

14 Q Yes.

15 A That's correct. Yes.

16 Q What has changed about Goldman 2012
17 that has led you to conclude there is sufficient
18 evidence to conclude that TCE has a causal
19 association with Parkinson's disease?

20 A What's changed in Goldman 2012 or since
21 the ATSDR report?

22 Q What has changed in Goldman 2012 which
23 you rely on in your opinions?

24 MR. TELAN:

25 Object to the form of the question.

1 A So I don't just rely solely on -- on
2 that in my opinions. So I relied on other --
3 other published data that came out after the 2017
4 report. So I can't say that there's something
5 specifically changed in that -- in that report,
6 but it's in the context of other -- other data.

7 MS. PLATT:

8 Q You also relied on Goldman's 2023 study
9 titled "Risk of Parkinson's Disease Among Service
10 Members at Marine Corps Base Camp Lejeune."
11 Correct?

12 A Yes.

13 Q I want to go ahead and enter that as
14 Exhibit 20 -- sorry -- Exhibit 7.

15 (DEPOSITION EXHIBIT NUMBER 7
16 WAS MARKED FOR IDENTIFICATION.)

17 MS. PLATT:

18 Q Exhibit 7 is the Goldman 2023 study;
19 correct?

20 A Yes.

21 Q And this is the study you relied on in
22 your report; correct?

23 A Yes.

24 Q And if you turn back to your report,
25 page 2 --

1 A Uh-huh.

2 Q -- the last sentence under the heading
3 "Epidemiological" states" the Goldman, et al.,
4 2023 and 2024 provides -- studies provide strong
5 evidence that TCE exposure modeled at a median
6 level of 0.366 milligrams per liter causes a
7 significantly increased risk for Parkinson's
8 disease over an average of three months of
9 exposure."

10 Did I read that correctly?

11 A Yes.

12 Q And that is referring to the study that
13 we are looking at as Exhibit 7. Correct?

14 A That and the 2024 Goldman, et al.,
15 study.

16 Q I want to go ahead and look at Goldman
17 2023.

18 The population in Goldman 2023 is
19 limited to April 1975 through December 1985;
20 correct?

21 A I believe so.

22 Q And this is the same population that
23 was used from a previous study by Dr. Bove of
24 ATSDR; correct?

25 A Sorry. Can you repeat that? I was

1 looking at a number in here, and I missed the
2 first part of your question.

3 Q That's okay.

4 The population used in Goldman 2023 was
5 the same population used for a previous study by
6 Dr. Bove of ATSDR; correct?

7 A I think so. These were VA medical
8 records. So I believe that was used in -- in
9 both, both studies. Or VA medical records from
10 those populations were used in both studies.

11 Q If you look at the first sentence under
12 cohort assembly --

13 A Uh-huh.

14 Q -- "study cohorts were previously
15 assembled by the U.S. Agency for Toxic Substances
16 and Disease Registry (ATSDR) as reported by Bove,
17 et al."

18 Did I read that correctly?

19 A Yes.

20 Q And then within this cohort, Goldman
21 '23 -- 2023 only identified the analytical cohort
22 to be those that used Veterans Health
23 Administration or Medicare health services;
24 correct?

25 A Yes.

1 Q And this excludes individuals that
2 never received VHA or Medicare services; correct?

3 A Yes.

4 Q And this is a limitation of the study
5 population; correct?

6 A Is it a limitation if it -- if it only
7 includes people who have used the VA? Is that
8 what you're asking?

9 Q Excluding those that have never
10 received VHA or Medicare services limits the
11 population studied; correct?

12 MR. TELAN:

13 Object to the form of the question.

14 A It limits the number of people that
15 would have been stationed there in total, I
16 believe. I don't know that -- I don't know the
17 information on, like, the percentage of people
18 who were there and didn't -- and have not
19 received medical care versus the population that
20 did. And if that information is in this paper, I
21 didn't see it.

22 MS. PLATT:

23 Q Would you agree this is a limitation of
24 the study?

25 A A limitation? It could be considered a

1 limitation.

2 Q And this is the same Goldman that
3 published Goldman 2012; correct?

4 A Yes.

5 Q The exposures in Goldman 2023 were
6 based solely on being stationed at Camp Lejeune
7 or Camp Pendleton; correct?

8 A I believe so, yes.

9 Q And there was no data on direct
10 exposures; correct?

11 A No. That data's not available.

12 Q Would you agree the lack of exposure
13 data is a limitation of Goldman 2023?

14 A "Lack of exposure data." It could be
15 considered a limitation. It could be considered
16 a limitation in probably both directions. Having
17 a -- a clearly exposed population might -- might
18 have benefited the -- the study in terms of its
19 power and the ability to come up with Parkinson's
20 risk.

21 So a limitation in this case could have
22 been sort of a limitation in either direction.
23 It could have, you know --

24 And not having that specific exposure
25 data could have -- I guess the word that you

1 might use is biased towards the null as well,
2 because it's considering all people and not just
3 a specific exposure subset.

4 Q But we do not know which way it biases
5 it towards the null; correct?

6 MR. TELAN:

7 Object to the form of the question.

8 A You -- you can't determine that based
9 on this. Just that those -- that sort of both
10 directions exist.

11 MS. PLATT:

12 Q Are you aware whether Goldman did any
13 type of dose-response analysis in this study?

14 A In this study, I don't know if 2023 had
15 a dose-response in it. I don't believe there was
16 a dose-response in the 2023 study.

17 Q Would you agree that doing a
18 dose-response analysis would have shed more light
19 on the causation question?

20 MR. TELAN:

21 Object to the form of the question.

22 A It'd be nice to have that information.
23 You know, if it -- if it were available, it could
24 be helpful.

25 MS. PLATT:

1 Q And you previously said that you are
2 aware of ATSDR's water modeling for Camp Lejeune;
3 correct?

4 A Yes.

5 Q Are you aware that Bove's 2020 --
6 sorry -- Bove's 2014 studies used ATSDR's water
7 modeling to consider whether a dose-response
8 relationship was shown?

9 A That -- that Bove used the water report
10 to do dose-response? Is that --

11 Q Yes.

12 A -- the question?

13 Q Were you aware that he did that in
14 2014?

15 A I don't know if I'm specifically aware
16 that that's exactly the methodology that was
17 used. I do know those two studies were related
18 to each other. But I guess I -- I don't
19 specifically look at Bove's report and look at
20 where he pulled the data from the ATSDR report
21 for -- ATSDR report for the water monitoring. So
22 for -- for the effort being, you know, explicitly
23 correct, I don't know that I knew that exactly.

24 Q Okay. Goldman 2023 considered the
25 association between TCE and Parkinson's disease;

1 correct?

2 A Yes.

3 Q And Goldman noted that the water was
4 also contaminated with other VOCs; correct?

5 A Yes.

6 Q But Goldman's conclusions only
7 considered the connection between TCE and
8 Parkinson's disease; correct?

9 MR. TELAN:

10 Object to the form.

11 A I believe that is -- that is true from
12 this paper.

13 MS. PLATT:

14 Q Would you consider ignoring the
15 potential presence of other chemical exposures a
16 limitation of the study?

17 MR. TELAN:

18 Object to the form of the question.

19 A Um, I think it's -- it's part of the
20 limitations of -- of any exposure study that, you
21 know, we are -- most people are not exposed in
22 isolation, and, so, you can never exclude a
23 limitation that -- that there's, you know, other
24 exposures that are -- that are ongoing.

25 MS. PLATT:

1 Q Turning to table 3 of Goldman 2023,
2 it's on page 677. Do you see that?

3 A Yes.

4 Q Looking at the first row is possible or
5 probable PD; correct?

6 A Yes.

7 Q And Goldman concluded an odds ratio of
8 1.7, with a confidence interval of 1.39 to 2.07;
9 correct?

10 A Yes.

11 Q And based off the confidence interval
12 listed there, that is not -- that would be
13 statistically significant; correct?

14 MR. TELAN:

15 Object to the form of the question.

16 A So, again, I'm looking at the p-value
17 there, which is less than .001 in terms -- to
18 ascertain statistical significance in this case.

19 MS. PLATT:

20 Q And would you say that's statistically
21 significant?

22 A Yes.

23 Q And then if you go three lines down,
24 there's a category titled "PD Ascertained Before
25 January 13th, 2017." Correct?

1 A Yes.

2 Q And Goldman limited this cohort to
3 those diagnosed before January 13th, 2017,
4 because that is when the VA designated
5 Parkinson's disease a presumptive
6 service-connected condition for veterans who
7 served at Camp Lejeune; correct?

8 A I believe so, yes.

9 Q And if we look at the odds ratio for
10 that category, it is 1.28, with a confidence
11 interval of 1.00 to 1.64. Correct?

12 A Yes.

13 Q And then the p-value went up to 0.52;
14 correct?

15 A Yes.

16 Q And that is greater than the p-value
17 shown for possible or probable Parkinson's
18 disease; correct?

19 A Yep. Yes. Excuse me.

20 Q So the statistical significance did not
21 carry through when limiting the category to those
22 diagnosed prior to January 13th, 2017. Correct?

23 MR. TELAN:

24 Object to the form.

25 A So, yeah. So in that smaller

1 population, there was a different odds ratio,
2 confidence interval, and statistical -- and
3 p-value for -- for that population.

4 MS. PLATT:

5 Q And this shows a potential selection
6 bias occurred due to available information in the
7 press after the date of January 13th, 2017?

8 MR. TELAN:

9 Object to the form.

10 A I don't know the answer to that. I
11 don't know how the press played into the, like,
12 collection of this -- this information from 2017
13 to when this was published in 2023.

14 MS. PLATT:

15 Q Would you say that this shows a
16 potential selection bias occurred?

17 MR. TELAN:

18 Object to the form of the question.

19 A Again, I don't know. I don't know the
20 answer to that. I don't know that I could make a
21 decision like that based off of just a single
22 table, you know, from information that I don't --
23 I'm not privy to.

24 MS. PLATT:

25 Q I'm going to your report, page 5.

1 A Uh-huh.

2 Q Never mind on that.

3 Another study that you cited in your
4 report was Bove's 2014 "Evaluation and mortality
5 among Marines and Navy personnel exposed to
6 contaminated drinking water at USMC base
7 Camp Lejeune: A retrospective cohort study."
8 Correct?

9 A I believe that is in -- in the report,
10 yes.

11 Q And that is footnote 18; correct?

12 A Yes.

13 Q I'm gonna go ahead and enter that as
14 Exhibit 8.

15 (DEPOSITION EXHIBIT NUMBER 8
16 WAS MARKED FOR IDENTIFICATION.)

17 MS. PLATT:

18 Q And, as we previously assessed, your
19 report is with regards to Parkinson's disease;
20 correct?

21 A Yes.

22 Q Are you aware that Bove 2014 mortality
23 in Marines and Navy personnel could not analyze
24 Parkinson's disease because of the lack of
25 Parkinson's disease data from Camp Lejeune and

1 Camp Pendleton's cohort?

2 MR. TELAN:

3 Object to the form.

4 A I'm aware in its mortality assessment
5 that Parkinson's cases were, as they discussed in
6 here, the number was low, again, because this is
7 mortality, and people don't die of Parkinson's
8 disease.

9 MS. PLATT:

10 Q If you could turn to page 7 of this
11 study.

12 A Uh-huh.

13 Q In the right-hand column, it states
14 "SMRs for male breast cancer and Parkinson's
15 disease were not calculated because there were
16 less than five cases in each cohort." Correct?

17 A Yes.

18 Q And looking to table 4, the first
19 sentence at the very bottom beneath the line, it
20 states "not evaluated due to small numbers were
21 Parkinson's disease and male breast cancer."
22 Correct?

23 A Yes.

24 Q And turning to table 5, the line at the
25 very bottom below the line states "not evaluated

1 due to small numbers were Parkinson's disease,
2 male breast cancer, and aplastic anemia."

3 Correct?

4 A Yes.

5 Q And you relied on this report in your
6 study?

7 Sorry. You relied on this study in
8 your report?

9 A I believe the citation for this is the
10 estimated median water concentration of 366 [sic]
11 micrograms per liter. That's where I see
12 citation 18 in the report.

13 Let me just see if I have other
14 citations.

15 And I cite it as 18, again, for the
16 .366 what I think should say micrograms per
17 liter, not milligrams per liter, on page 2. So
18 I'm citing it for the water concentration in --
19 in the report.

20 Q And that is the sole reason you
21 considered Bove 2014?

22 A Yeah. So I think in this report, the
23 only time you'll see reference number 18 is in
24 relation to that 366 [sic] micrograms per liter
25 number.

1 Q And, as we previously discussed, you
2 were aware of Bove's 2014 study on civilian
3 employees; correct?

4 A Yes.

5 Q Were you aware that the Bove 2014 study
6 on civilian employees did consider Parkinson's
7 disease?

8 A Yes.

9 Q And it's not in your materials
10 considered list; correct?

11 A I -- I don't think so. I'll have to
12 double-check. I don't think so.

13 Harder to look up than if I do control
14 find on my computer.

15 I think the only Bove is the -- I
16 believe the only Bove citation is the 2014
17 evaluation of mortality among Marines citation.

18 Q And, as we previously discussed, you
19 are aware of Bove's 2024 evaluation of Marines,
20 Navy personnel, and civilian workers; correct?

21 A Is that also a mortality study?

22 Q It's a cohort study. Yes.

23 A It's a cohort study?

24 Yes, I am familiar with it. I just had
25 to remind myself which one that was.

1 Q And you did not consider that in
2 writing your report; correct?

3 A Yeah. Again, because my report
4 specifically focused more on mechanism, I -- I
5 didn't -- I didn't explicitly go into detail on a
6 lot of -- a lot of the epidemiology from -- from
7 these cohorts.

8 Q Are you aware Bove's 2024 study did
9 consider Parkinson's disease in its analysis?

10 A I believe so, yes. Yeah.

11 Q And you also considered Goldman's 2024
12 study titled "Parkinson's Disease Progression and
13 Exposure to Contaminated Water at Camp Lejeune."
14 Correct?

15 A Yes.

16 Q I'm gonna go ahead and enter that as
17 Exhibit Number 9.

18 (DEPOSITION EXHIBIT NUMBER 9
19 WAS MARKED FOR IDENTIFICATION.)

20 MS. PLATT:

21 Q And, as previously discussed, you
22 stated on page 2 that this study provided strong
23 evidence; correct?

24 A Yes.

25 Q And this is the same Dr. Goldman that

1 published Goldman 2023; correct?

2 A Yes.

3 Q And the same one that published Goldman
4 2012; correct?

5 A Yes.

6 Q And this is the same population
7 analyzed by Goldman 2023; correct?

8 A I believe so, yes.

9 Q Which was the same population analyzed
10 by Bove 2024; correct?

11 A I believe so.

12 MR. TELAN:

13 Object to form.

14 MS. PLATT:

15 Q Let me restate.

16 The same population analyzed in Goldman
17 2023 is the same population analyzed in Bove
18 2024; correct?

19 MR. TELAN:

20 Object to the form.

21 A I'm thinking back to when we talked
22 earlier about the specific study population. Let
23 me just make sure I understand correctly.

24 So study cohorts were previously
25 assembled by the U.S. ATSDR's disease registry as

1 reported in Bove, et al. So that -- if -- if
2 that's the same population you're referring to,
3 then yes.

4 MS. PLATT:

5 Q Yes.

6 Goldman 2024 did not limit the
7 population to diagnosis before January 17th,
8 2017, as he did in Goldman 2023; correct?

9 MR. TELAN:

10 Object to the form.

11 A I am so sorry. Could you repeat that?

12 MS. PLATT:

13 Q Goldman 2024 did not limit the
14 population to those diagnosed before January
15 17th, 2017, as he did in Goldman 2023. Correct?

16 MR. TELAN:

17 Same objection.

18 A I don't believe so.

19 MS. PLATT:

20 Q And this study did not consider
21 individual exposure to contaminants; correct?

22 A Um, it says there were monthly
23 estimates, some for each veteran, to obtain
24 cumulative exposure estimates to TCE, PCE, vinyl
25 chloride, benzene, and total VOCs.

1 And then it says they classified
2 exposure status for each contaminant as binary
3 (exposed or unexposed) or as tertiles, so none,
4 median or median -- none, less than median, or
5 greater than median.

6 So I don't know if that means that
7 they -- that they didn't consider, like,
8 individual exposures.

9 Q They did not have samples of individual
10 exposure to contaminants; correct?

11 A That's right. Yes.

12 Q And Goldman inferred exposure based off
13 of residential location and time; correct?

14 A I believe so.

15 Q Goldman did not have any other data on
16 lifetime exposures; correct?

17 A I believe that's correct. Yes.

18 Q Goldman 2024 did not observe an earlier
19 age of Parkinson's diagnosis in exposed
20 individuals; correct?

21 MR. TELAN:

22 Object to the form.

23 A Age. In relationship to unexposed
24 individuals with PD? Is that the context?

25 MS. PLATT:

1 Q Yes.

2 A So looks like they --

3 That's correct. It looks like there
4 is -- that we don't see a difference in age.

5 Q Wouldn't you agree if a chemical
6 increased the rate of progression of PD, the age
7 of diagnosis would be lower?

8 MR. TELAN:

9 Object to the form.

10 A Not necessarily. Risk comes from many
11 different things, and exposure, in some contexts,
12 could potentially accelerate progression or
13 accelerate when a person might get Parkinson's
14 disease. It doesn't necessarily mean that it
15 will have a strong impact on the age at which
16 it's diagnosed in -- in isolation. It doesn't
17 necessarily mean that.

18 MS. PLATT:

19 Q Would you like to take a break?

20 A I'm okay.

21 Q We'll keep going, then.

22 MS. PLATT:

23 Are you okay?

24 THE COURT REPORTER:

25 Yeah. I'm fine.

1 MS. PLATT:

2 Q Turning to page 2 of your report --

3 A Uh-huh.

4 Q -- you summarize the experimental
5 evidence; correct?

6 A Uh-huh.

7 Q And you state, after the word
8 "experimental," "published data from laboratory
9 studies in rats and mice show that exposure to
10 TCE causes the selective degeneration of
11 dopaminergic neurons from the SN and their
12 terminal projections to the striatum in brain
13 tissue, the key pathological hallmark of PD in
14 humans."

15 Correct?

16 A Yes.

17 Q And, in that sentence, you cite to
18 footnotes 1, 3, and 15 through 17; correct?

19 A Yes.

20 Q And footnote number 1 is Ilieva's 2024
21 article; correct?

22 A Ilieva. But yes.

23 Q Ilieva.

24 I want to go ahead and mark that as
25 Exhibit Number 10.

1 (DEPOSITION EXHIBIT NUMBER 10
2 WAS MARKED FOR IDENTIFICATION.)

3 MS. PLATT:

4 Q I just handed you Exhibit Number 10,
5 and that is Ilieva's article titled "LRRK2 kinase
6 inhibition protects against Parkinson's
7 disease-associated environmental toxicants."
8 Correct?

9 A Yes. And it's pronounced "Lark 2
10 kinase," if that's helpful to you.

11 Q Thank you.
12 And you are a coauthor on this paper;
13 correct?

14 A Yes.

15 Q And, in this article, you and your
16 coauthors study the protein leucine rich repeat
17 kinase 2, also known as LRRK2; correct?

18 A We --

19 That is part of the -- part of the
20 study is -- is assessing LRRK2 in this, yes.

21 Q And LRRK2 is an autosomal dominant
22 inherited risk factor for Parkinson's disease;
23 correct?

24 A No. So mutations in LRRK2 are an
25 autosomal dominant form of Parkinson's disease.

1 LRRK2 itself is expressed in everybody. So you
2 have LRRK2. I have LRRK2. It's the mutations
3 that are inherited as an autosomal dominant form
4 of PD.

5 Q And you investigated whether TCE
6 exposure increased activity in the mutations in
7 the LRRK2 gene? Is that correct?

8 A No. So LRRK2 biology is complex. So I
9 apologize.

10 LRRK2 is --

11 Let me just give a quick -- a brief
12 basis so that I can explain.

13 So LRRK2 is a kinase protein, and it
14 is --

15 The pathological mutations in LRRK2
16 increase its kinase activity. And it's the
17 kinase activity of the protein, not the gene. So
18 the gene itself would probably stay relatively
19 consistent, but the protein increases its kinase
20 activity. And so that's -- when we talk about
21 LRRK2, like, pathobiology, it's increased kinase
22 activity.

23 And, so, what we studied in this was
24 related to wild-type LRRK2 -- increases in
25 wild-type LRRK2 kinase activity. We did study

1 that in context of one of the LRRK2 kinase
2 mutations, and then we also studied it in the
3 context of inhibition of LRRK2 kinase activity,
4 all at the protein level.

5 Q Okay. And on page 9, the last
6 paragraph, you state "both genes and
7 environmental factors contribute to PD risk and
8 in most cases likely stem from the interaction
9 between them." Correct?

10 A Yes.

11 Q And in the study at three weeks of
12 exposure, dopaminergic neuron number was only --
13 Sorry. Let me start again.

14 A Okay.

15 MR. TELAN:

16 Are you still on that same paragraph,
17 Elizabeth, or are you moving on?

18 MS. PLATT:

19 No. This is a question.

20 MR. TELAN:

21 Oh, okay.

22 MS. PLATT:

23 Q At three weeks of exposure,
24 dopaminergic neuron number was the only
25 pathological endpoint evaluated; correct?

1 A In this study, at three weeks of
2 exposure, yes, we reported on -- we reported on
3 dopaminergic neuron number. That's correct.

4 Q And no changes were reported. Correct?

5 A Yes.

6 Q At six weeks of exposure, there was a
7 45 percent loss of dopaminergic neurons in the
8 SNPC and elevated markers of oxidative stress,
9 mitochondrial dysfunction, and phagocytic
10 activity by the microglia."

11 A Correct. Phagocytic activity by
12 microglia.

13 That sounds like the conclusions that
14 we -- that we wrote in this paper, yes.

15 Q What is oxidative stress?

16 A Oxidative stress is the result of an
17 imbalance between too many reactive oxygen
18 species in a cell and the detoxification
19 mechanisms to detoxify them. So when you have an
20 overwhelming amount of reactive oxygen species
21 and you can't properly detoxify them, we call
22 that oxidative stress. So more reactive oxygen
23 species than a cell can handle.

24 Q And oxidative stress is a normal
25 function of the body; correct?

1 A So oxi- -- reactive oxygen species can
2 be -- are included as a sort of normal function
3 of cells. You use them --

4 Or you.

5 A cell uses them to signal. So
6 having -- having oxidat- -- reactive oxygen
7 species in the cell can actually be used for
8 signaling mechanisms. It can be used for various
9 other -- well, actually many other things in a
10 cell, including things like protection against
11 pathogens or -- or protection against, you know,
12 other -- other pathological but -- but normal
13 features in a cell.

14 Oxidative stress in itself sort of
15 refers to a pathological feature where you are
16 overwhelming the normal or homeostatic mechanisms
17 that would typically deal with those reactive
18 oxygen species.

19 So oxidative stress itself is not
20 necessarily considered a normal process, but,
21 yes, reactive oxygen species are.

22 Q In the body, cells get old and die or
23 are actively killed; correct?

24 A Some do.

25 Q And what role does the microglia in the

1 brain play in this oxidative stress process?

2 A So microglia can become activated in
3 response to oxidative stress. They -- microglia
4 have sort of end feet that kind of stick out into
5 their environment. They monitor for any change,
6 really, but mostly in the context of what we're
7 talking about in, like, a pathological change.
8 And they can be --

9 If they pick up on a strong amount of
10 oxidative stress, in conjunction with other
11 signaling molecules, that could indicate to a
12 microglia, because there are immune cells in the
13 brain that they need to activate in order to deal
14 with some pathological event that's happening in
15 the brain.

16 So, in that context, they could respond
17 to oxidative stress as part of their surveillance
18 and -- and properly functioning job as an immune
19 cell.

20 Q And oxidative stress is not at a
21 constant in the body; correct?

22 A At a given moment, you would not --

23 If you were healthy, you would not --
24 your cells would not be undergoing an undue
25 amount of oxidative stress in their -- in the --

1 in -- in a cell, yes.

2 Q But there would be some oxidative
3 stress in a healthy human; correct?

4 MR. TELAN:

5 Object to the form of the question.

6 A I don't -- I mean, I guess at any given
7 cell, the ratio of the antioxidant capacity of
8 the reactive oxygen species could be, you know,
9 slightly out of balance. But in terms of what's
10 considered healthy, you want a balance between
11 the detoxification systems and the -- and
12 whatever source, if it's -- if it's endogenous
13 source of oxidative stress or exogenous source
14 of -- excuse me -- reactive oxygen species.

15 So you could -- you know, at various
16 points, there could be oxidative stress going on,
17 and presumably the detoxification systems would
18 handle that, and it would drop down below what we
19 consider stress or oxidative stress.

20 MS. PLATT:

21 Q And humans can reduce their oxidative
22 stress through the foods they eat; correct?

23 A There is literature to suggest that
24 certain foods and certain diets can be considered
25 anti- -- anti-inflammatory or -- or high in

1 antioxidants.

2 Q Uh-huh.

3 And this study demonstrated that at six
4 weeks of exposure, there was a 45 percent loss of
5 dopaminergic neurons; correct?

6 A I just want to make sure I see that.

7 So at six weeks, we measured, so --

8 Yeah. I think, on average, it'd
9 probably be -- it looks like it's between 40 and
10 50. I --

11 If we -- if we explicitly state that
12 number, then it's probably from the actual value.
13 But I don't know if that's coming off of just the
14 graph or if that's a specific value that we
15 published. Let me just double-check.

16 So it doesn't look like we actually
17 state 45 percent in there.

18 Yes, we do. 45 percent loss. It's
19 right there on page 8. 45 percent loss of
20 dopaminergic neurons.

21 Q Are you aware that Dr. Miller and other
22 plaintiffs' experts stated in his report that the
23 motor symptoms of Parkinson's do not appear until
24 60 to 80 percent of the brain dopamine neurons
25 are lost?

1 MR. TELAN:

2 Object to the form of the question.

3 A I'm not aware that that was stated in
4 his report. I'm aware of the literature that
5 states that 60 to 80 percent of dopamine is lost
6 for motor symptoms.

7 MS. PLATT:

8 Q And you have published with Dr. Miller;
9 correct?

10 A We are on one review paper together, I
11 think.

12 Q And this study, I livea 2024, did not
13 assess the effects of TCE on motor function;
14 correct?

15 A That's right. We used this same model
16 in our 2021 paper, and that did assess the motor
17 effect using the same dose -- dose schedule and
18 number -- and age of animal. So we did not
19 separately measure motor function in this paper.

20 Q And this study did not assess the
21 effect on dopamine content in the SNPC; correct?

22 A No. When you lose neurons, that
23 usually supersedes assessing dopamine content.

24 Q You also relied on Adamson's 2023 study
25 titled "Low-dose inhalation exposure to

1 trichloroethylene induces dopaminergic
2 neurodegradation [sic] in rodents." Correct?

3 A Degeneration.

4 Q Degeneration. Sorry about that.

5 A Yes.

6 Q And that is footnote 3 in your report;
7 correct?

8 A Let me just double-check. Yes.

9 Q I'll go ahead and enter that as Exhibit
10 Number 11.

11 (DEPOSITION EXHIBIT NUMBER 11
12 WAS MARKED FOR IDENTIFICATION.)

13 MR. TELAN:

14 Just for planning purposes, Elizabeth,
15 we're coming up on noon. I didn't know what your
16 thoughts were about taking a break for lunch or
17 not.

18 MS. PLATT:

19 We can take a break now.

20 MR. TELAN:

21 How are you doing?

22 THE WITNESS:

23 Yeah. Whatever you guys want to do is
24 fine. Yeah.

25 MS. PLATT:

1 Okay. Let's go ahead and take a break.

2 VIDEOGRAPHER:

3 Going off the record. Time is 11:52.

4 (LUNCH RECESS.)

5 VIDEOGRAPHER:

6 Back on the record. The time is 12:51.

7 MS. PLATT:

8 Q Dr. De Miranda, I'm not sure if I
9 formally entered Exhibit Number 11, so let me go
10 ahead and do that.

11 I handed you what I marked Exhibit
12 Number 11, and that is Adamson's 2023 study
13 titled "Low-Dose inhalation exposure to
14 trichloroethylene induces dopaminergic
15 neurogeneration [sic] in rodents."

16 I think I said that wrong.
17 Neurodegeneration in rodents.

18 A Yes.

19 Q And you have that in front of you;
20 correct?

21 A Yes.

22 Q And you relied on this study in your
23 report; correct?

24 A Yes.

25 Q And you're one of the coauthors on this

1 report; correct?

2 A Yes.

3 Q And I believe you are the senior
4 author?

5 A Corresponding author, yes.

6 Q The corresponding author.

7 What is a hypothesis?

8 A The hypothesis in this study --
9 Sorry.

10 Q What is a hypothesis?

11 A What is a hypothesis. So, broadly
12 speaking, a hypothesis is a -- so evidence-based
13 idea that -- that --

14 Sorry. Distracted.

15 An evidence-based idea where you -- you
16 form -- you know, you postulate that in some -- I
17 guess in some cases a mechanism is causing or
18 related to an effect that you see in a system.
19 So it's very difficult -- right? -- to define
20 broadly.

21 Your hypothesis of a study is the
22 question that you're asking, and then it's the
23 basis for how you form your experiment and then
24 how you would go about proving that hypothesis
25 with the -- with the experimental data that you

1 provide in the paper.

2 Q And what was your hypothesis in Adamson
3 2023?

4 A So our hypothesis was that inhalation
5 of -- of TCE would cause Parkinsonian-like
6 neurodegeneration in the brain of rodents, and
7 we --

8 Let's see if I have a specific
9 hypothesis statement. Sometimes we don't write
10 specifically "we hypothesize" in the study. But
11 our -- we say "we predicted that inhalation
12 exposure would be especially toxic to
13 dopaminergic neurons and produce Parkinsonian
14 pathology."

15 And, so, that's very similar to what I
16 just said, more broadly, about what the -- what
17 we hypothesized in this paper.

18 Q And where are you reading from?

19 A That would be on page 219, the second
20 paragraph underneath the table, underneath table
21 1.

22 Q Were you specifically looking at the
23 alteration that is associated with or produces
24 results in motor dysfunction?

25 A Motor dysfunction was one of the

1 parameters that we -- that we measured, but our
2 hypothesis, as it's stated there, or when we say
3 "we predicted" is -- is looking at dopaminergic
4 toxicity. So specifically the neurons was our
5 primary outcome measure. We did measure motor
6 impairments in this -- in this study.

7 Q Okay. Adamson 2023 tested mice and
8 rats exposed to doses of TCE via inhalation;
9 correct?

10 A Yes.

11 Q This study did not test for other
12 routes of exposure; correct?

13 A Not in this study.

14 Q And your reasoning for testing
15 inhalation was, in part, due to the lack of
16 studies at the time that tested inhalation; is
17 that correct?

18 A At the time, there were no studies that
19 tested Parkinsonian pathology from inhalation.
20 There had been previous studies that looked at
21 TCE inhalation with -- with different outcome
22 measures.

23 Q So on page 218, which is the first page
24 of the document, at the very bottom, the last
25 sentence of that page states "however, previous

1 studies investigating neurodegeneration caused by
2 TCE have used oral gavage or intraperitoneal
3 injection of high doses of TCE in rodents, which
4 limits the translational relevance to most
5 environmental exposure conditions."

6 Did I read that correctly?

7 A Yes. I'll just quickly say it's oral
8 gavage and intraperitoneal injection, in case
9 that's helpful.

10 Q Thank you.

11 And you cite to three studies there,
12 Guehl, et al., 1999?

13 A Yes.

14 Q Liu, et al., 2010?

15 A Yes.

16 Q And Sauerbeck, et al., 2012; correct?

17 A Yes.

18 Q And did you consider those three
19 studies in writing your report?

20 MR. TELAN:

21 Object to the form.

22 A Um, I think -- I think at least Liu,
23 et al., and probably Sauerbeck. I'll have to
24 check -- I'll have to look.

25 Some of these are some of the older TCE

1 studies.

2 I don't know if I specifically cite
3 Sauerbeck. Yes, I -- yes, the Sauerbeck.

4 And then Liu 2010 I probably cited,
5 because that's another early paper. That's 2018.
6 2010.

7 And then I also don't know how you
8 pronounce that. Guehl, "Gu-el"? Let me see.

9 Q I believe it's footnote 55.

10 A Yes. Footnote 55, Guehl, 1999.

11 Q So just so we're clear on the record,
12 you considered these three studies, Guehl,
13 et al., in 1999, Liu, et al., 2010, and
14 Sauerbeck, et al., 2012, in writing your report;
15 correct?

16 A They were part of my citation list,
17 yes.

18 Q Rodents have higher metabolic rates and
19 shorter lifespans than humans; correct?

20 A Yes.

21 Q And this leads to different
22 toxicological responses between rodents and
23 humans; correct?

24 A Yes.

25 Q And the effects of TCE in rodents are

1 not precisely replicated in humans; correct?

2 A There is no -- no animal study that can
3 replicate anything in a human. That's true
4 across any disease, any health condition ever,
5 period, full stop.

6 Q And on page 224, at the bottom of the
7 left-hand column, you and your coauthors state
8 "thus, dose conversions of TCE exposure from
9 animal to human and vice versa are complex, rely
10 on imperfect conversion assumptions, and should
11 be considered in the temporal context of a human
12 lifespan."

13 Did I read that correctly?

14 A Yes.

15 MR. MICELI:

16 Leslie dropped off, but she'll be
17 getting back on.

18 MS. PLATT:

19 Q On page 220 of Adamson 2023, under the
20 heading "Results," it states "to model a low
21 human equivalent dose (HED) of TCE in rodents, we
22 used allometric scaling to normalize TCE dose to
23 body surface area between humans and rats or
24 mice."

25 Did I read that correctly?

1 A Yes.

2 Q And that is the process used in this
3 study; correct?

4 A That's simply a calculation modeling
5 human equivalent dose between the dose that was
6 used in rodents and what would be an extrapolated
7 dose in humans.

8 So -- so the basis for the study wasn't
9 necessarily based off of this. It was just one
10 of the ways that we calculated human equivalent
11 dose.

12 Q And, so, this calculation was done or
13 used in your study; correct?

14 A Yes. I mean, it's -- it's reported in
15 the -- in the paper. So it was -- we -- we
16 exposed these animals. We also calculated a
17 human equivalent dose.

18 Q And you exposed rats to a scaled dose;
19 is that correct?

20 A Scaled dose?

21 Q Yes.

22 A Can you define what you mean by "scaled
23 dose" there? Sorry. I'm a little unclear if
24 that's what that means.

25 Q Sure.

1 On page 219 --

2 A Uh-huh.

3 Q -- about halfway down the middle
4 paragraph on the left-hand column --

5 A Uh-huh.

6 Q -- it states: We found that exposure
7 to time weighted average (TWA) of 50 ppm TCE for
8 rats and 150 ppm TCE for mice over 8 and 12
9 weeks, respectively, resulted in a significant
10 loss of the nigrostriatal dopaminergic neurons
11 that corresponded with motor behavior defects
12 [sic].

13 Did I read that correctly?

14 A Behavior deficits. But, yes, the
15 same -- the same applies.

16 Q Sorry. Deficits.

17 And, so, you exposed rats to 50 ppm
18 over 8 weeks; correct?

19 A Correct.

20 Q And that was --

21 Was that 7 days per week?

22 A Five.

23 Q Five days per week.

24 So you exposed rats 50 ppm, five days
25 per week, over 8 weeks; correct?

1 A That's correct.

2 Q And was the dose that you gave them 50
3 ppm, or did that -- yeah. Was the dose that you
4 gave them 50 ppm?

5 A Okay. I understand.

6 Yes. The dose we gave them was 50
7 parts per million.

8 Q Did it fluctuate throughout the day?

9 A So -- so, yes. So you -- when you do a
10 VOC in a positive inhalation chamber, you're
11 always going to have, like, a small amount of
12 fluctuation. I believe in this -- you can see in
13 figure 1 we actually show an example of that.

14 And, so, when you report VOC total
15 exposure, you use a time-weighted average. And
16 our time-weighted average was 50 parts per
17 million. So that's how that -- that is stated.

18 So you're always gonna have fluctuation
19 because it's a volatile and it's -- it's in the
20 air.

21 But as you can see from this -- from
22 the graph in figure 1C, we did map where -- how
23 the fluctuations occurred, and then we calculated
24 TWA from that.

25 Q And, so, there were times in the day

1 when a rat was exposed to a higher dosage than 50
2 ppm TCE; correct?

3 A There could be fluctuations in either
4 direction, high and low. For --

5 Usually, it'd usually be a matter of
6 seconds. But the fluctuations occur pretty
7 quickly.

8 Q And you exposed mice to a scaled dose
9 of 100 ppm TCE for 5 hours a day for 12 weeks;
10 correct?

11 MR. TELAN:

12 Object to the form.

13 A So, again, I don't know what "scaled"
14 means in this context. We just exposed them to a
15 hundred parts per million of -- of TCE for 12
16 weeks, yes.

17 MS. PLATT:

18 Q And there were fluctuations in that
19 dosage; correct?

20 A So the time-weighted average is 100
21 parts per million. So over the hours of the day
22 that we exposed them, fluctuations could occur.
23 But the time-weighted average is what we report
24 in the paper.

25 Q And there were times in the day when a

1 mouse was exposed to more than 100 ppm; correct?

2 A And -- and less than 100 parts per
3 million.

4 Q Where in Adamson 2023 do you note that
5 the inhalation concentration for mice and rats
6 was, in fact, higher than what you reported?

7 A So in figure 1C, we show the
8 fluctuations as an example, but we only report
9 time-weighted average in this paper.
10 Fluctuations throughout -- in a -- in an
11 inhalation chamber in terms of volatile standards
12 in this field are reported as time-weighted
13 average.

14 Q And figure 1C is a scatter plot;
15 correct?

16 A It's -- they're points shown over time.
17 I don't know --

18 This is a -- it would be an XY -- I
19 don't know that that's necessarily a scatter
20 plot, no, because it's over time.

21 Q What is the solid green line in figure
22 1C?

23 A That's a time-weighted average.

24 Q And what is the upper dotted line in
25 1C?

1 A Those are the standard deviation --
2 those are standard deviation lines from the
3 time-weighted average. So whenever you have an
4 average, you also have a standard deviation. So
5 that shows the standard deviation.

6 Q Is 1C for both rats and mice?

7 A This is just for rats.

8 Q And, so, figure 1C shows that at at
9 least one point in time, the dose went up to
10 almost 100 ppm; correct?

11 MR. TELAN:

12 Object to the form.

13 A Yes. And also down to probably 10
14 parts per million as well.

15 MS. PLATT:

16 Q Do you note that at all in the text of
17 Adamson 2023?

18 A No. We are focused on time-weighted
19 average, as we reported in the paper.

20 Q Okay. How did you determine the doses
21 to give to the mice and the rats?

22 A 100 parts per million at the time was
23 the OSHA time-weighted average exposure limit for
24 a workday, and 50 parts per million was half of
25 that. And, so, we chose those doses based on the

1 sensitivity of each rodent species in the
2 reported literature to TCE.

3 We know that rats tend to be more
4 sensitive to -- to toxicants, and mice, slightly
5 less sensitive to toxicants. And so the dosing
6 strategy in both rats and mice was based off of
7 what's been previously reported in terms of their
8 response to toxicity. And then the -- the doses
9 themselves were based off of the OSHA workplace
10 standards.

11 Q And OSHA workplace standards are
12 occupational level exposures; correct?

13 A Yep.

14 Q And what do you rely on to come to the
15 conclusion that a -- that the amount for a mouse
16 should be greater than the amount for a rat?

17 A Mice metabolize TCE faster than rats.
18 And in the Parkinson's research field, it's a
19 pretty well-known standard that mice tend to
20 produce less Parkinson's pathology in the brain
21 than rats. And so you typically have to push the
22 system a little bit further in mice in order to
23 see changes.

24 Rats have more sensitivity. They
25 show -- they more readily show pathology than --

1 than mice do. And, so, it's -- it's pretty
2 typical that you have to use -- if you're
3 comparing toxins, you have to use higher doses in
4 mice.

5 There's also published literature from
6 both rats and mice with TCE exposure, and in
7 previous studies, people had used higher doses in
8 mice than they had in rats. And, so, we also
9 based it off of some of the existing literature
10 as well.

11 Q Do you cite to that in Adamson 2023?

12 A We talk about the differences in
13 metabolism, and I know we cite in a couple --
14 like, we site Liu, et al., in 2018, which uses
15 the higher doses in mice of TCE.

16 Q Are you an expert in pharmacokinetics
17 or toxicokinetics?

18 A I wouldn't say I'm an expert in either
19 of those, toxicology or pharmacology parameters.

20 Q Did you consult with an expert in
21 pharmacokinetics or toxicokinetics in creating
22 this study?

23 A Since this was not a pharmacokinetic or
24 toxicokinetic study, we did not.

25 Q To find the human equivalent dose of

1 TCE in a rodent, you used allometric scaling to
2 normalize TCE dose to the body surface area
3 between humans and rats and mice; correct?

4 A Yes.

5 Q For oral dose, the default
6 extrapolation is surface area or body weight;
7 correct?

8 A Yes.

9 Q And you and your coauthors measured
10 inhalation, not oral dose; correct?

11 A Yes. There is no direct inhalation
12 relationship that you can measure with an
13 equation like this. So you have to extrapolate
14 from the oral dose.

15 Q Are you familiar with the human
16 equivalent concentration?

17 A Yes.

18 Q And why did you not use the human
19 equivalent concentration in this study?

20 A Again, because we don't have the
21 ability to take a standardized, like, correction
22 factor like you do with this human equivalent
23 dose, and, so, we didn't have the ability in this
24 study to take into consideration things like
25 respiration rate and various other inhalation

1 parameters. So we used a tool and we noted,
2 as -- as you have mentioned earlier, we note that
3 it involves imprecise modeling. We -- we -- we
4 establish it as a human equivalent dose with
5 those limitations. We discussed that pretty --
6 pretty readily throughout the manuscript.

7 Q Are you aware that the human equivalent
8 concentration can be determined using the
9 blood-air partition coefficient?

10 A Yes.

11 Q And you did not use the blood-air
12 partition coefficient, did you?

13 A Not in this context, no.

14 Q Are you familiar with the extrapolation
15 for inhalation using physiologically based
16 pharmacokinetic modeling?

17 A Yes.

18 Q And you did not use that modeling in
19 this study; correct?

20 A No. We don't do PBBK modeling in --
21 really in, like, a mechanism neurotoxicity study.
22 That's sort of its own study on its own. It
23 would be a study on its own.

24 Q Are you familiar with what a category 3
25 chemical is?

1 A I've heard it before, but I -- I
2 couldn't tell you exactly the definition.

3 Q Okay. Do you know if TCE is a
4 category 3 chemical?

5 A I would have to know the context of
6 that.

7 Q Under EPA's method of determining what
8 category a chemical falls into.

9 A I --
10 You know, we don't -- we don't use that
11 kind of categori- -- categorization --

12 Is that right?

13 -- in this -- in the type of research
14 we do. So, you know, again, I know that those
15 parameters exist. That's not really something
16 that we take into consideration that often.

17 Q Okay. TCE produce effects -- TCE
18 produces effects in remote and systemic tissues
19 rather in the -- rather than in the portal of
20 entry tissues; correct?

21 MR. TELAN:

22 Object to the form of the question.

23 A Uh, okay. So portal of entry tissues,
24 what does that mean in this context?

25 MS. PLATT:

1 Q That is the definition of a category 3
2 chemical.

3 A Okay. So the published literature
4 suggests that that's probably true for TCE. I'm
5 thinking solely in the context of Parkinson's
6 toxicity. I -- I can't comment on the specific
7 effects of that in cancer or some of the other
8 effects that TCE has been associated with.

9 I guess the published literature would
10 suggest that that's probably true. You know,
11 portal of entry here is a little bit of a gray
12 area because, you know, the olfactory bulb is
13 technically part of your brain, and it's -- I
14 don't know if you consider that a portal of entry
15 or not in this case.

16 But, you know, we are -- if -- if
17 that's true, if, you know, if it's just talking
18 about, like, the nasal cavity, you know, versus
19 brain, then I guess -- I think that would be true
20 under this context.

21 Q TCE is absorbed more highly or more
22 efficiently in rat and mouse blood than in human
23 blood; correct?

24 A I've -- I've heard that, but I don't --
25 I have not specifically studied that myself.

1 I've read that before. I don't know that I fully
2 understand the -- the sort of concept behind
3 that, but I have read that before.

4 Q Because it is absorbed more
5 efficiently, TCE reaches the tissues at a higher
6 rate in rodents than in humans. Correct?

7 A Again, I don't know that that's 100
8 percent true for -- for things like the brain,
9 particularly when we're talking about inhalation.
10 Like, I don't know if that's true for the blood
11 of -- you know, if those studies have encompassed
12 things like inhalation and how that corresponds
13 with brain concentrations.

14 So -- so from the context of what's
15 been published, I'm sure that there are data that
16 show that in an animal it could be --

17 Is it absorbed? Is that the word you
18 used? Partitioned?

19 Q It reaches the tissues at a higher
20 rate.

21 A Reaches the tissues at a higher rate.
22 It's -- it's --

23 If -- if -- under the context of how
24 that was measured, that's very likely true. I
25 just don't know in the context of if it's

1 measured simil- -- like in a similar mechanism to
2 inhalation and then reaching the brain. So in
3 terms of how that was studied, that would -- that
4 would be the determining factor of -- of the
5 limitations of that data.

6 Q You studied rats and mice in Adamson
7 2023; correct?

8 A Yes.

9 Q And your --

10 Individuals at Camp Lejeune were not
11 exposed to 50 ppm TCE for 7 hours a day 5 days a
12 week for 8 weeks; correct?

13 MR. TELAN:

14 Object to the form.

15 A I don't think so, but I don't know.

16 MS. PLATT:

17 Q On page 224 of Adamson 2023, the second
18 line in the left-hand column, "however, exposure
19 to contaminated water at Camp Lejeune occurred
20 for over 30 years, varied by time, season, and
21 location, and included other toxicants, such as
22 tetrachlorethylene and benzene," citing to
23 Maslia, et al., 2016, "all of which are
24 conditions that cannot be fully recapitulated in
25 laboratory animals."

1 Did I read that correctly?

2 A Yes.

3 Q Turning to Figure 2 on page 222 of
4 Adamson --

5 Are you on that page?

6 A Yes.

7 Q And you reproduced figure 2 in your
8 report in this case; correct? It's on page 5.

9 A That's correct.

10 Q Figure 2 shows inhalation exposure to
11 TCE induces nigrostriatal dopaminergic
12 neurodegeneration and motor deficit in adult
13 Lewis rats; correct?

14 A Yes.

15 Q And, as we discussed, the exposure for
16 rats was 50 ppm of TCE for 7 hours a day, 5 hours
17 [sic] per week, for 8 weeks; correct?

18 MR. TELAN:

19 Object to the form.

20 A Yeah. I think -- I think -- I think
21 you meant 7 hours a day, 5 days a week.

22 MS. PLATT:

23 Q Yes. Can I restate that for the
24 record?

25 MR. TELAN:

1 Sure.

2 MS. PLATT:

3 Q Thank you.

4 Exposure for rats was 50 ppm of TCE for
5 7 hours a day -- no -- for 5 hours a day --

6 MR. TELAN:

7 Nope.

8 A You were right the first time.

9 MS. PLATT:

10 Q -- 7 hours a day, 5 days a week, for 8
11 weeks.

12 A That's right.

13 Q Okay.

14 A Correct.

15 Q Thank you.

16 And, as we discussed, there was some
17 fluctuation in that dose during the course of the
18 day; correct?

19 A Yes.

20 Q Looking at Figure 2, panel D --

21 A Uh-huh.

22 Q -- the average for the controls is
23 about 23,000; correct?

24 A That looks about right, based on that
25 graph, yes.

1 Q And that is measuring the tyrosine
2 hydroxylase?

3 A That is measuring TH neurons. So
4 it's -- it's not just measuring TH. It's also --

5 The way that we do stereology, which is
6 what's represented in panel B, is we co-localize
7 TH, which is a marker for dopaminergic neurons,
8 with Nissl, which is a marker for cell bodies.

9 And, so, TH-positive neurons, not just
10 TH-positive signal, is represented in panel D
11 there.

12 Q And for the controls, it was about
13 23,000 measured; correct?

14 A Yes.

15 Q And for the exposed, it was about
16 12,000?

17 A That looks about right.

18 Q So this is about a 50 percent loss;
19 correct?

20 A Yes.

21 Q These violin plots in 2D show there is
22 some overlap between the results of the control
23 and the exposure; correct?

24 A Yes. There's one data point that looks
25 like it's overlapped.

1 Q Exposure to inhaled TCE did not have
2 the same effect on each of the exposed rats;
3 correct?

4 A Yeah.

5 In any animal study, you will have
6 variation in the number of dopaminergic neurons
7 in an -- in an animal substantia nigra.

8 Q So at least one rat in the exposed
9 group had approximately the same number of TH
10 neurons as some in the control group; correct?

11 A Um, based on that graph specifically,
12 there was one animal that looked like it
13 overlapped in terms of total number.

14 Q Looking below to panels E through L,
15 these violin plots show there is overlap between
16 the results of the control and the exposure
17 groups; correct?

18 A So -- yes. So you're --

19 In behavioral parameters and in violin
20 plots, when you are comparing two groups like
21 this with a T test, you oftentimes will have
22 overlap. And then the statistical significance,
23 which is reported with the p-values or the stars
24 above each bar, is showing you the difference in
25 means from those groups.

1 Q And I'm sorry. Did you answer the
2 question? There is showing some overlap between
3 the exposed and the control?

4 A I said in any violin plot, you're going
5 to have in between -- if you're -- you're
6 comparing statistically, you can have -- you may
7 have difference in means while still having
8 overlap between the groups.

9 Q Thank you.

10 A Uh-huh.

11 Q Were the same animals used in panel 2D
12 as in E through L?

13 A Yes.

14 Q It doesn't appear that the number of
15 dots are the same in D as in panels E through L.

16 A Yes. So some of the animals that were
17 in the behavioral study did not --

18 Some of -- some of the animals that we
19 used in the behavioral study were not included
20 in --

21 Excuse me. Let me reverse that.

22 Some of the animals we used for
23 histology were not included in the behavioral
24 study.

25 Q Why were they not included?

1 A So it would have been a subset of
2 animals that we took to do behavior and then some
3 that we took to do histopath on.

4 Q How many rats were included in the
5 study overall?

6 A I think overall --
7 Let's see. I think we had an n of
8 eight overall of rats.

9 Q Do you see that reported in Adamson
10 2023?

11 A An n of 8? Yes.

12 Q Where is that at?

13 A So in each figure legend, we report how
14 many animals were assessed in each experiment.
15 So it would be in the figure legends.

16 Q So for figure 2, where is that
17 reported? Oh, at the very end.

18 A Uh-huh.

19 Q Okay. And it looks like for E through
20 L, there was about sometimes six, sometimes less,
21 in that portion of the study. Correct?

22 A Yes.

23 Q How did you determine what subset was
24 used for E through L?

25 A Yeah. So in this behavioral assay,

1 some of the -- some of the behavioral assays were
2 too -- they would have taken, like, too long to
3 go through, so you use a subset of animals in
4 order to do, like, the longer behavioral test.

5 And, so, if they were --

6 Some of the data would have been
7 reported from, again, a subset of those animals
8 because the behavioral tests were really lengthy,
9 and especially with rats, because it's hard to
10 get them through behavior in comparison to mice.
11 So they're a little more lengthy.

12 Q So how did you choose which specific
13 six we're going to use for the behavioral?

14 A That would have been done randomly.

15 Q Did you replicate the study, then, with
16 the remaining ones?

17 A They would have been dead and taken for
18 histopath.

19 Q According to this figure, you did not
20 do a paired analysis. Is that correct?

21 A This is a standard student's t-test.
22 So in a -- when you compare two groups, it's a --
23 we use a statistical t-test.

24 Q So you did not pair which dots that
25 appear in D correlate with which dots appear in

1 panel E?

2 A No. That is not the way that we
3 traditionally run behavior versus histopathology.

4 Q Did you consider doing such pairing?

5 A Some -- very rarely, some people report
6 that. That's typically more for, like, cognitive
7 behavior.

8 In motor behavior reports in
9 neurodegeneration studies, pairing an animal with
10 its histopathology and motor behavior is not
11 commonly done. We typically don't do that
12 because we run sort of batches of animals through
13 this.

14 And, you know, because we're -- they're
15 experimental animals and because they all come
16 from the same source, we -- I set expectations
17 for them to have certain similarities across
18 different animals. So whereas a human you might
19 want to pair because there are many variations
20 within a human, we don't see a lot of variations
21 between these rats. They're inbred rats. We get
22 them from the same vendor. They're --

23 In the case of mice, they're likely
24 litter mates, that sort of thing. So we
25 typically don't carry that across from

1 histopathology to behavior in an experimental rat
2 study.

3 Q So, as we discussed, there's at least
4 one dot in panel D that shows overlap with the
5 control group; correct?

6 MR. TELAN:

7 Object to the form. Asked and
8 answered.

9 A Yes.

10 MS. PLATT:

11 Q And we cannot determine whether that
12 dot from panel D is part of the study that
13 resulted in panels E through L; correct?

14 A That dot being --

15 I mean, can you determine that that
16 dot --

17 Actually, we probably could if we went
18 back and looked at the data. But from this, no,
19 you can't determine that. Yeah.

20 Q It's not recorded in Adamson.

21 A It's not recorded in this paper, yes.
22 But in theory, we could. We could look at that.

23 Q And so we cannot determine whether the
24 altered dopaminergic function was paired with an
25 altered gait; correct?

1 MR. TELAN:

2 Object to the form.

3 A Um, I disagree with that. I think that
4 because you have a loss of dopaminergic neurons
5 statistically -- and it is highly statistically
6 significant -- and you see a statistical
7 impairment of gait and motor features that are
8 aligned at -- with face validity of -- of gait
9 disturbances and what would be considered
10 Parkinsonian, that, you know, you can actually
11 say that a loss of dopaminergic neurons is
12 related to gait impairments that we measured in
13 panels E through L. Because the averages are
14 what we're detecting. We don't detect a single
15 animal and then make any inference on that. That
16 would be not -- that would not be powered
17 correctly to do that, and that's not the way
18 experimental research is done.

19 Q In panels E through L, not all of the
20 panels are statistically significant; correct?

21 A Every panel there shows statistical
22 significance.

23 Q What is the distinction between one
24 asterisk and two asterisks?

25 A If you look at our statistical

1 results -- or our statistical analysis methods,
2 we should have in there a definition of where
3 that p-value is based off of the asterisk. So
4 one star is a p-value of less than .05, two stars
5 is a p-value of less than .01, three stars, .001,
6 and four stars, .0001.

7 And, then, in each of the -- in the
8 figure legend of figure 2, you can actually see
9 the p-value. So if you start at E, front swing
10 speed, the p-value is .035, hind swing speed,
11 .034, and so on.

12 Q Uh-huh.

13 So for panel E, it has a lower p-value
14 than panel F; correct?

15 A Yes.

16 Q On page 226, in the last paragraph --
17 sorry -- the last sentence above the word
18 "conclusions" --

19 A Uh-huh.

20 Q -- states "future studies to assess TCE
21 exposure over the course of a lifespan, extremely
22 low doses as observed in common environmental
23 conditions, and the interaction with other PD
24 risk factors will provide more information on the
25 mechanisms of TCE-induced neurodegeneration."

1 Did I read that correctly?

2 A Yes.

3 Q Have those studies occurred?

4 A We're doing them now.

5 Q They're not published yet.

6 A No.

7 Q I'm going back to your report, page 10.

8 In the top paragraph, third sentence starts with

9 the word "published."

10 A Uh-huh.

11 Q Do you see that?

12 A Yes.

13 Q "Published data in Adamson et al. 2023

14 on TCE inhalation show that even with much lower

15 doses, inhaled TCE -- of inhaled TCE, rats and

16 mice had more severe dopaminergic

17 neurodegeneration than animals that received TCE

18 via oral gavage."

19 Did I read that correctly?

20 A Yes.

21 Q So the oral gavage studies had a

22 smaller amount of loss of the SNPC neurons than

23 seen in Adamson; correct?

24 A In some of them, yes.

25 Q And the use of oral gavage limits the

1 translational relevance to more environmental --
2 relevance to environmental exposure conditions;
3 correct?

4 MR. TELAN:

5 Object to form.

6 A In some cases, yes. What we showed in
7 the Adamson et al. paper helped us to understand
8 something specifically about that, though, which
9 is that when we modeled -- because we modeled
10 the --

11 In, like, the previous papers using
12 oral gavage, we used the 200 milligrams per
13 kilogram dose.

14 Because we used that higher dose and
15 saw a similar amount of neurodegeneration, that
16 actually helped us to understand that the amount
17 of TCE that's likely reaching the brain in that
18 case in order to model that is -- is more
19 likely -- or is more like the amount of TCE
20 that's reaching the brain via inhalation.

21 And, so, what -- the way that we
22 interpret this -- and we discuss this pretty
23 extensively in Adamson et al. -- is that you can
24 use lower doses of inhaled TCE to induce this
25 same type or more severe dopaminergic

1 neurodegeneration than oral gavage. And, so,
2 those -- that's kind of the relationship between
3 those two -- those two data points.

4 MS. PLATT:

5 Q Turning back to Adamson 2023, Exhibit
6 11, page 219, the top paragraph -- sorry -- the
7 second paragraph, the second sentence, "we
8 predicted that inhalation exposure could be
9 especially toxic to dopaminergic neurons and
10 produce Parkinsonian pathology at doses lower
11 than previously used in other routes of
12 exposure."

13 Correct?

14 A Yes.

15 Q The other routes of exposure that
16 you're referring to include oral route, or
17 ingestion; correct?

18 A Ingestion as well as intraperitoneal
19 injection.

20 Q And that is shooting it into the belly;
21 correct?

22 A Essentially, yes. Intraperitoneal
23 space. So you try to get it in the space where
24 there's no organs and it can be readily absorbed
25 with -- within the body without necessarily

1 hitting -- without going specifically to a target
2 organ intraperitoneal-wise.

3 Q Toxicity would be less for an oral
4 route in part due to first pass effects; correct?

5 A In the brain --

6 MR. TELAN:

7 Object to the form of the question.

8 A In the brain, we think that that's
9 true. It'd be less toxic in the brain.

10 MS. PLATT:

11 Q And can you describe what first pass
12 effects are?

13 A Yes. So when you ingest something,
14 your blood flow from your stomach and -- and
15 the -- I guess, in this case, the chemicals that
16 are absorbed through -- through the oral
17 ingestion route go -- your blood goes first to
18 your liver and then out to the rest of your body.
19 And, so, that's considered first pass, hepatic
20 metabolism.

21 And, so, especially for a chemical that
22 reaches the liver, it's going to be metabolized
23 in the liver before it reaches much of the rest
24 of the body.

25 Q So when it is -- when a chemical is

1 metabolized through the liver first, less of the
2 parent chemical may reach the other parts of the
3 body; correct?

4 A That's typically what is considered
5 true.

6 Q So when TCE is administered through
7 inhalation, the first pass is avoided, and the
8 parent chemical, TCE, not the metabolite, reaches
9 the brain; correct?

10 A We don't know the specific relationship
11 between how much of the parent compound and
12 metabolite reach the brain, but we -- we suspect
13 that, with inhalation, you get more of the parent
14 compound into the brain than you would if it
15 underwent first pass hepatic metabolism. But --
16 so that's sort of what we postulate, is that more
17 of the parent compound reaches the brain. I --
18 we -- we have not explicitly measured that in
19 this -- in this case.

20 Q I think you can set that one aside.

21 In your report, you also cited to your
22 own article from 2021 titled "The industrial
23 solvent trichloroethylene induces LRRK2 kinase
24 activity and dopaminergic neurodegeneration in a
25 rat model of Parkinson's disease." Correct?

1 A Yes.

2 Q I'm gonna go ahead and mark that as
3 Exhibit Number 12.

4 (DEPOSITION EXHIBIT NUMBER 12
5 WAS MARKED FOR IDENTIFICATION.)

6 MS. PLATT:

7 Q And I just handed you Exhibit
8 Number 12. That is your article from 2021;
9 correct?

10 A Yes.

11 Q In this study, you administered TCE via
12 olive oil gavage at doses of 50, 100, 200, 400,
13 and 800 -- 800 milligrams per kilograms daily for
14 up to six weeks; correct?

15 A No. Actually, we only did the dose --
16 the different doses for a three-week period. The
17 rest of the study was a
18 200-milligrams-per-kilogram dose for six weeks.

19 Q So for three weeks, you administered
20 50, 100, 200, 400, and 800; correct?

21 A That's correct.

22 Q And then for six weeks, you
23 administered 200 milligrams per kilogram;
24 correct?

25 A Yes.

1 Q Thank you.

2 A Uh-huh.

3 Q The 200 milligrams per kilogram for six
4 weeks resulted in approximately 30 percent
5 reduction in TH intensity within the striatum;
6 correct?

7 A Goes from about 750 --

8 Yeah. So that's about -- about 30 --
9 35, perhaps, percent.

10 Q I believe on page 5, left-hand
11 column --

12 A Yeah.

13 Oh. I was thinking of dopamine neuron.
14 Sorry. The striatum -- the striatum is, yes, 30
15 percent.

16 Q Okay. Just for clarity in the
17 record --

18 A Yes.

19 Q -- let me ask that one more time.

20 A Yes.

21 Q For 200 milligrams per kilogram for six
22 weeks resulted in approximately 30 percent
23 reduction in TH intensity within the striatum;
24 correct?

25 A Yes.

1 Q And 200 milligrams per kilogram for six
2 weeks caused approximately a 32 percent loss of
3 dopaminergic neurons in the substantia nigra;
4 correct?

5 A Yes.

6 Q And some animals showed a greater
7 effect; correct?

8 A There is a -- a mean and a standard
9 deviation, just like in any animal study, and
10 that's reported in the -- in the graphs in figure
11 1.

12 Q So some animals showed a greater
13 effect, and some animals showed a lesser effect;
14 correct?

15 A The -- we --

16 I mean, so we display interquartile
17 ranges in here, but we don't do statistics to
18 show individually a difference between each
19 animal. Right? So that's what the interquartile
20 ranges are showing. It's just showing the -- the
21 range over which we measured these effects.

22 And, so, you could say that the range,
23 or the interquartile range, you know, is from --

24 I mean, we have degrees of freedom in
25 here. So, you know, the F statistic is reported

1 in the bottom of figure 1. So -- so it's hard to
2 know from -- because we don't have individual
3 points on this, you know, where each animal fell.
4 We're showing interquartile ranges.

5 Q So, to state it differently, the
6 animals did show a range of effects; correct?

7 A That's correct.

8 Q Thank you.

9 In your report, you also cited to Liu
10 2010; correct?

11 A Yes.

12 Q I'm going to mark that as Exhibit
13 Number 13.

14 (DEPOSITION EXHIBIT NUMBER 13
15 WAS MARKED FOR IDENTIFICATION.)

16 MS. PLATT:

17 Q I just handed you Exhibit Number 13,
18 which is Liu's 2010 article "Trichloroethylene
19 induces dopaminergic neurodegeneration in Fisher
20 344 rats." Correct?

21 A Yes.

22 Q This study exposed animals via oral
23 gavage; correct?

24 A Yes.

25 Q If you turn to page 776 --

1 A Uh-huh.

2 Q -- there is the bar graph in figure 1B.
3 Do you see that?

4 A Yes.

5 Q The Y axis unit is measuring thousands
6 of TH blood cells; correct?

7 A Yes.

8 Q And the cell count appears to be about
9 45, 30, 33, and 25 thousands; correct?

10 Would you like me to go through those
11 slower?

12 A Maybe a little bit. Maybe 45. It
13 looks like 35, 30 --

14 You know, it's a little bit higher than
15 30, I think. But other than that, yes, I think
16 that's -- that was a fairly good assessment.

17 Q For the last one, would you agree it's
18 about 25,000?

19 A It looks like it's about 25 on the
20 graph.

21 Q Okay. And those correspond to zero,
22 200, 500, and 1,000 milligrams per kilogram five
23 days a week for six weeks; correct?

24 A I need to see if it's five days a week.
25 Yes, it was. Five days a week for six

1 weeks. That's correct.

2 Q Okay. And in the highest dose, there
3 is still more than 50 percent of the neurons
4 left; correct?

5 A It looks like that, yes.

6 Q You also cited to Liu's follow-up
7 assessment from 2018 titled "Trichloroethylene
8 and Parkinson's disease risk assessment."
9 Correct?

10 A Yes.

11 Q I'm gonna go ahead and mark that as
12 Exhibit 14.

13 (DEPOSITION EXHIBIT NUMBER 14
14 WAS MARKED FOR IDENTIFICATION.)

15 MS. PLATT:

16 I apologize. These are not stapled.

17 I just handed you Exhibit 14. This is
18 the article you cited to in your report; correct?

19 A Yes.

20 Q Turning to page 6,204 --

21 MR. TELAN:

22 What was that page number, again,
23 Elizabeth?

24 MS. PLATT:

25 6,204.

1 MR. TELAN:

2 Thank you.

3 MS. PLATT:

4 Q Sorry. Give me one moment.

5 In the paragraph under "results," about
6 a third of the way down there's a sentence that
7 starts "statistical analysis." Do you see that?

8 A I see it.

9 Q "Statistical analysis of cell counting
10 indicated a significant loss of TH-positive
11 neurons by 32.7 percent at three months and 52.3
12 percent after an 8-month treatment with TCE
13 compared to vehicle."

14 Correct?

15 A Yes.

16 Q "For mice given 30 milligrams per
17 kilogram five days per week for 8 months, there
18 is a 52.3 percent loss." Correct?

19 A I just want to check the dose on that.

20 So I don't remember this study being 30
21 milligrams per kilogram. Do you have a place
22 where that is on the paper that I can look at?

23 Q I don't have it noted, but I can check.

24 A Okay. I believe this was 400
25 milligrams per kilogram.

1 MR. MICELI:

2 6202, Materials and Methods, about
3 halfway down, two-thirds of the way down that
4 paragraph.

5 MS. PLATT:

6 Q Okay. So let me rephrase my question,
7 then.

8 A Uh-huh. Uh-huh.

9 Q For mice given 400 milligrams per
10 kilogram per day five days a week for 8 months,
11 there was a 52.3 percent loss; correct?

12 A Of -- of TH-positive neurons.

13 Q Of TH-positive neurons; correct?

14 A Uh-huh. Yes.

15 Q Do you know how those doses compare to
16 the exposures at Camp Lejeune?

17 MR. TELAN:

18 Object to the form of the question.

19 A No, I don't know how those compare.

20 MS. PLATT:

21 Q And, then, going to the first page, the
22 end of the abstract, the last sentence starts
23 with "however."

24 Do you see that?

25 A Yes.

1 Q "However, even prolonged administration
2 of TCE was insufficient for producing a greater
3 than 50 percent loss of nigral dopamine neurons,
4 indicating that additional comorbid factors would
5 be needed for mimicking the profound loss of
6 dopamine neurons seen in Parkinson's disease."

7 Did I read that correctly?

8 A You read it correctly.

9 Q With respect to the animal studies that
10 you rely on in your report and that we've
11 discussed here today, do any of these studies
12 report on the permanence of the effect after the
13 exposure is ended?

14 A So the best answer to that is how the
15 dopamine neurons are counted. So, for example,
16 in all of our studies, we co-localize, as I
17 mentioned, TH, which is the marker for dopamine
18 neurons with Nissl. I believe the same thing was
19 done in Liu et al. 2018. They report Nissl in
20 that as well.

21 And, so, the loss of Nissl means that
22 the cell is gone. And, so, why that's important
23 in relation to TH is that just loss of TH in
24 itself could indicate that the cell may still be
25 alive but the TH has been downregulated. But

1 when you include Nissl and the cell body is gone,
2 because dopaminergic neurons are postmitotic
3 cells and once -- you're sort of born with all
4 the neurons you have, so -- dopamine neurons that
5 you have, so once those -- those cells are gone,
6 they can't come back.

7 And, so, from the idea of permanence,
8 there would be no way for those neurons to regrow
9 or regenerate. So once that Nissl cell body
10 corresponding or co-localized with the TH is
11 gone, it can't come back.

12 Q Turning back to your report in this
13 case --

14 Sorry. Are you okay or do you need to
15 take a break?

16 A I'm good for right now.

17 Q Okay.

18 A I'll let you know, if that's okay.

19 Q Turning to page 4 of your report, the
20 last sentence of the fourth paragraph starts with
21 "for the purposes."

22 Do you see that?

23 A Uh-huh.

24 Q "For the purposes of this report and
25 where relevant, I have applied the standards and

1 terminology used under legal review, which I have
2 been instructed in this case to be equally as
3 likely as not regarding risk for PD from
4 sustained exposures to TCE and other solvents."

5 Did I read that correctly?

6 A Yes.

7 Q What does equally as likely as not
8 mean?

9 A In the context in which I have written
10 it here, I define this as meaning that the risk
11 for PD from TCE exposure is -- is sort of like if
12 you imagined, like, a scale -- right? -- where
13 sort of more likely than not would be tipping the
14 scale, you know, if you think out of 100, like 51
15 to 49, equally likely as not being sort of a
16 50/50. So meaning that --

17 And the data could show -- the data
18 show that it is --

19 Sort of tricky not to say "equally
20 likely than not" when you're trying to say that.

21 -- but equally as likely to show an
22 effect as not, which, you know, typically when we
23 report, like, neuron loss in a paper, it's with
24 statistical significance.

25 So when I say in this paper I've

1 applied the standards and the terminology of a
2 legal review, I'm simply explaining how I
3 extrapolate what would be statistical
4 significance to the, in this case, sort of lower
5 standards of equally likely as not.

6 Q And where does the phrase "equally --
7 equally as likely as not" originate from?

8 A I first came upon that terminology in
9 the Camp Lejeune Justice Act document.

10 Q And when you say the Camp Lejeune
11 Justice Act document, are you referring to the
12 statute?

13 A Yes. The equipoise or above.

14 Q Where do you apply the standard in your
15 report?

16 A So in the -- so on page 11 I talk about
17 how -- at least as likely as not versus more
18 likely than not. And, so, that's where, in -- in
19 this -- in this document I talk about how
20 translating sort of the statistical significance
21 from these experimental studies, how that
22 translates to sort of the legal terminology
23 that's been defined under -- in this specific
24 case.

25 Q And where on page 11 are you

1 specifically looking?

2 A The third paragraph, sort of two-thirds
3 of the way down. So together with
4 epidemiological evidence showing, that's -- that
5 sentence.

6 Q Okay. And, so, this sentence states
7 "together with the epidemiological evidence
8 showing increased risk for PD from TCE exposure
9 as well as potentially worsened morbidity, these
10 data support a conclusion that TCE exposure under
11 the conditions described at Camp Lejeune, North
12 Carolina, is more likely than not to cause
13 Park -- PD. Under the Camp Lejeune Justice Act,
14 the standard of causation is defined as at least
15 as likely as not, which is lower than the more
16 likely as not standard, which, in my opinion, is
17 reached."

18 Did I read that correctly?

19 A Yes.

20 Q Have you had any legal training?

21 A I have not gone to law school, no.

22 Q You did not attend law school?

23 A I did not attend law school.

24 Q You have not passed the bar?

25 A I have not passed the bar. Maybe in --

1 maybe in my next career.

2 MS. PLATT:

3 Is someone on here now?

4 MR. TELAN:

5 Leslie LaMacchia has reentered.

6 MS. PLATT:

7 Okay. Can we put that on the record?

8 Thank you.

9 Q Is there anywhere else in your expert
10 report that you apply this standard of equally as
11 likely as not?

12 MR. TELAN:

13 Object to the form.

14 A I think those are the only two places,
15 unless I'm missing something.

16 That's the only two places where I use
17 the terminology "equally as likely as not."

18 MS. PLATT:

19 Q And you're referring to page 1 and page
20 11; correct?

21 A Uh-huh, even though on page 11 I do
22 state it a little bit differently. I say --
23 instead of equally likely, I say at least as
24 likely as not. So there's just a slight
25 difference in the wording there, but my meaning

1 was the same.

2 Q You would define those two phrases the
3 same?

4 A Yeah. I don't --

5 My intention was not to have them
6 actually written out differently. I think it was
7 equally -- equally is -- is as -- is the same as
8 at least in this case.

9 Q Okay. Going back to page 1, you have
10 your summary of opinions listed here. Do you see
11 that?

12 A Yes.

13 Q You state "to a reasonable degree of
14 scientific probability, my opinions are that TCE
15 can cause neurotoxicity that recapitulates the
16 pathological hallmarks of Parkinson's disease,
17 dopaminergic neurodegeneration, alpha-synuclein
18 accumulation, and neuroinflammation."

19 Did I read that correctly?

20 A Yes.

21 Q And is this based on the equally as
22 likely as not standard?

23 MR. TELAN:

24 Object to the form.

25 A So this is based on --

1 Because I'm saying that -- I'm talking
2 specifically about neurotoxicity that
3 recapitulates pathological hallmarks of
4 Parkinson's disease, this is based on -- more on
5 the sort of statistical significance I spoke
6 about earlier in terms of experimental studies,
7 so defined a priori by statistical power.

8 So -- so saying that -- so saying that
9 can cause neurotoxicity in this case, that would
10 be related directly to the data that -- that
11 is -- makes up the -- sort of makes up the, I
12 guess, neurotoxicity neurodegeneration of
13 pathological hallmark of Parkinson's disease, if
14 that makes sense.

15 MS. PLATT:

16 Q And continuing on in that paragraph,
17 "my assessment is that published epidemiological
18 data shows that TCE exposure causes increased
19 risk for Parkinson's disease, and experimental
20 evidence supports a biological basis for the
21 underlying neuropathology that drives Parkinson's
22 neurodegeneration."

23 Did I read that correctly?

24 A Yes.

25 Q What does an increased risk for

1 Parkinson's disease mean?

2 A So in a Parkinson's field, increased
3 risk for -- for PD is -- we refer to that as when
4 we have data that shows increased odds ratios or
5 increased associations. This often encompasses
6 an entire body of published literature.

7 In the context of this, the published
8 epidemiological data showing -- showing what we
9 call risk; right? So that's -- that's different
10 from being causal. Right? So that's just
11 showing an associated risk with Parkinson's
12 disease.

13 And, so, in -- in the PD field,
14 Parkinson's risk is often talked about in terms
15 of these -- these studies that show this
16 association or this, you know, I guess, odds
17 ratio or increased risk for -- for PD.

18 Q And, so, you would characterize that
19 differently than causing Parkinson's disease;
20 correct?

21 MR. TELAN:

22 Object to the form.

23 A So in terms of -- in terms of when you
24 use --

25 When I use the word "increased risk,"

1 that is different than saying cause.

2 MS. PLATT:

3 Q And that statement meets the equally as
4 likely as not standard; correct?

5 MR. TELAN:

6 Object to the form of the question.

7 A So that statement is not specifically
8 relating to equally likely as not. So -- so
9 that's not -- that statement specifically, in the
10 way it's written here, is -- is not relating that
11 specifically equally likely as not.

12 MS. PLATT:

13 Q Are you applying a standard there?

14 A No. I'm simply just stating sort of
15 what it says in -- in that -- in that sentence.
16 I think the standards, what you're asking is
17 applied later on in that paragraph.

18 Q Okay. So let's keep reading that
19 paragraph.

20 A Sure.

21 Q "Thus, based upon my analysis of the
22 scientific evidence, including the consistency
23 between the three pillars of research science,
24 epidemiological studies, in vivo experimental
25 studies, and mechanistic data, it is my

1 professional opinion that supports a conclusion
2 that TCE exposure is more likely than not known
3 to cause Parkinson's disease."

4 Did I read that correctly?

5 A Yes.

6 Q Let's finish up that paragraph. "My
7 opinions are based upon my education, training,
8 experience, and research in the field of
9 neurotoxicology and Parkinson's disease."

10 Did I read that correctly?

11 A Yes.

12 Q And that is -- that is applying the
13 equally as likely as not standard; correct?

14 MR. TELAN:

15 Object to the form.

16 Mischaracterization.

17 A So that's just simply applying the --
18 as I say in the -- in the paragraph, the last
19 paragraph on page 1, that I'm applying the
20 standards under sort of legal review and, in this
21 case, using the totality of the data, applying
22 the standards of more likely than not known to
23 cause Parkinson's disease.

24 MS. PLATT:

25 Q And you state in that sentence "known

1 to cause Parkinson's disease." Correct?

2 A Yes.

3 Q Known by who?

4 A The scientific community.

5 Q And who would you describe the
6 scientific community to be?

7 A The individuals who publish or who are
8 authors on the published papers that make up or
9 comprise the data that I analyze in this report.

10 MR. TELAN:

11 When you get to a convenient break,
12 just for a grace break. Doesn't have to be now.
13 Just the next few minutes.

14 MS. PLATT:

15 Okay. Give me a few more minutes --

16 MR. TELAN:

17 Sure.

18 MS. PLATT:

19 -- and then we can take a break.

20 Q Turning to page 11 of your report,
21 there's a heading "Weight of Experimental
22 Evidence and Relationship to Epidemiological
23 Data."

24 You applied a weight of the evidence
25 approach; correct?

1 A Yes.

2 Q And what is a weight of the evidence
3 approach?

4 A So the weight of the evidence approach
5 that I applied in this case is the one that I
6 summarized on the first or second page where I'm
7 talking about totality of data across both
8 experimental and epidemiological data, so taking
9 into consideration the data across the different
10 scientific disciplines, that study, the
11 relationship between TCE and Parkinson's disease.

12 Q And you state -- about halfway down,
13 there's a sentence that starts "there are." Do
14 you see that?

15 A Yes.

16 Q "There are limitations to experimental
17 and animal models of disease."

18 Did I read that correctly?

19 A Yes.

20 Q What are the limitations?

21 MR. TELAN:

22 Object to the form.

23 A So we've already talked a little bit
24 about this, but the -- the major limitation that
25 we talked earlier about is that when you model

1 something especially as complex as a disease,
2 you're never going to completely recapitulate
3 every feature of every disease in a model system.
4 And that's true across every single scientific
5 discipline. It doesn't matter if it's disease
6 or, you know, it's Parkinson's or cancer or heart
7 disease.

8 And, so, when you -- when you're doing
9 that work, you work within those limitations.
10 And by assessing the work within those
11 limitations, you determine whether or not the
12 data that's coming out of those papers -- you
13 know, how to weigh that data.

14 And, so, the, you know, second part of
15 that is talking about how even with the
16 limitations, there are strong data to sort of
17 reach the conclusions that -- to reach the
18 conclusions that I have in this. And, so, you
19 know, talking about weighing -- weighing that.
20 So you're weighing the strengths against the
21 limitations.

22 Q And your opinions that you stated in
23 this report are based off of your analysis of
24 both the mechanistic data as well as the
25 experimental and epidemiological studies;

1 correct?

2 A Yes. So the mechanistic data comes
3 from experimental data as sort of a subset of
4 that. But, yes, that is -- that is true.

5 Q Okay. And we cannot diagnose a rat or
6 a mouse with Parkinson's disease; correct?

7 A That's true.

8 Q And, then, reading on in that
9 paragraph, the next sentence, "furthermore," do
10 you see that?

11 A Yes.

12 Q "Furthermore, TCE can induce the
13 molecular pathology that underlies hallmark PD
14 pathology, such as oxidative damage and
15 endolysosomal dysfunction."

16 A Lysosomal.

17 Q "Lysosomal dysfunction."
18 Did I read that correctly?

19 A Yes.

20 Q Oxidative damage can lead to other
21 health outcomes than PD; correct?

22 A Yeah, in the -- in -- I mean, that's
23 really broad. So, yes, so you have oxidative
24 damage in your liver, in your heart. And, so,
25 that could be true of a lot of different disease

1 conditions, yeah.

2 Q Okay. So oxidative damage could lead
3 to Alzheimer's disease?

4 MR. TELAN:

5 Object to the form.

6 A Well, dopamine neurons don't --
7 don't -- don't selectively degenerate in
8 Alzheimer's disease. So while there is oxidative
9 stress in cells, you know, in relation to
10 Alzheimer's disease, this is talking about
11 hallmark PD pathology. And we don't see
12 dopaminergic neurodegeneration, the selective
13 loss of dopamine neurons, in Alzheimer's disease.

14 Q I'm asking you just generally, can
15 oxidative damage lead to Alzheimer's disease?

16 MR. TELAN:

17 Object to the form.

18 A There are some data that suggest that
19 oxidative damage is related to Alzheimer's
20 disease. I don't know specifically that there's,
21 like, a -- there's, like, as close of a link with
22 AD in terms of just specifically oxidative stress
23 or oxidative damage, oxidative stress.

24 Q Endolysosomal dysfunction can lead to
25 other outcomes other than Parkinson's disease;

1 correct?

2 A Yes.

3 Q And you state in the next sentence on
4 paragraph 11 in that middle paragraph -- sorry --
5 on page 11 in that middle paragraph, starting
6 with "together."

7 Do you see that?

8 A Yes.

9 Q "Together with the epidemiological
10 evidence showing increased risk for PD from TCE
11 exposure, as well as a potentially worsened
12 morbidity, these data support a conclusion that
13 TCE exposure under the conditions described at
14 Camp Lejeune, North Carolina, is more likely than
15 not to cause Parkinson's disease, or PD."

16 Did I read that correctly?

17 A Yes.

18 Q In that sentence, you cite to the
19 footnote, which is Goldman 2023; correct?

20 A I believe that's number 14? Yes.

21 Q And, as we discussed earlier today,
22 Goldman 2023 did not show a statistically
23 significant OR when limiting the population to
24 those diagnosed before the VA began providing
25 benefits to individuals with Parkinson's disease

1 that were at Camp Lejeune.

2 MR. TELAN:

3 Object to the form and asked and
4 answered.

5 A Um, I did not consider the delineation
6 between that in writing that sentence.

7 MS. PLATT:

8 Q What do you mean, you did not consider
9 the delineation?

10 A I -- I didn't delineate between the
11 2017 and 2023 data in that -- when I wrote that
12 sentence.

13 Q But that is a true statement?

14 MR. TELAN:

15 Form objection.

16 A Um --

17 Sorry. Go ahead.

18 MS. PLATT:

19 Q That is a true statement that Goldman
20 2023 did not show a statistically significant OR
21 when limiting the population to those diagnosed
22 before the VA began providing benefits to the
23 individuals with Parkinson's disease that were at
24 Camp Lejeune; correct?

25 MR. TELAN:

1 Same -- same objection, and asked and
2 answered.

3 A As we talked about earlier, that is
4 what we -- that's what we discussed, yes.

5 MS. PLATT:

6 Q Thank you.

7 And in that sentence you state
8 "potentially worsened morbidity here."

9 What do you mean by "potentially"?

10 A So that refers to the 2024 study of
11 Parkinson's disease progression and exposure from
12 Goldman et al. in 2024 showing that there was a
13 relationship between TCE exposure, and there were
14 four parameters. I could look at them. But it's
15 time -- I think it was -- I know one's time to
16 psychosis. One's time to fall -- latency to
17 fall.

18 And, so, those showed a faster time or
19 a shorter time to reach those parameters that are
20 typically considered with Parkinson's morbidity.

21 Q And, as we discussed, Goldman 2023 did
22 not find a significantly-statistically
23 significant lower age of diagnosis in the
24 exposure group. Correct?

25 MR. TELAN:

1 Object.

2 A Yes. We talked about that earlier.

3 MS. PLATT:

4 Q And you characterize this as
5 potentially. Is it your position that there is
6 not sufficient evidence?

7 A So I'm making a general statement there
8 with morbidity instead of listing out those
9 specific parameters, like latency to fall,
10 psychosis. So that's where that terminology
11 comes from. It's just talking about using --

12 Like, I'm extrapolating my own opinions
13 on how that's described as morbidity from those
14 four parameters that were measured in that study.

15 Q And that Goldman 20 -- sorry -- Goldman
16 2024 is the only study that you cite to support
17 that; correct?

18 A It's the only study I know of that --
19 that has measured those parameters.

20 Q Is it typical in the scientific world
21 to reach a conclusion based off of solely one
22 study?

23 MR. TELAN:

24 Object to the form.

25 A Um, I think, you know, seems like we

1 talked about this a little bit already. You can
2 infer -- you can infer a lot from a -- from a
3 study, as I -- as I do here in this report and as
4 I do every day in the research. Everything I do
5 is based on the underlying body of research that,
6 you know, I'm familiar with. I think I mentioned
7 before, you know, I've probably read thousands of
8 papers a year on Parkinson's disease.

9 And so, you know, when I -- when you're
10 looking in totality at, you know, a field with
11 lots of different publications and lots of
12 different outcome measures, you're sort of --
13 you -- it'd be very difficult to sort of forget
14 all of that and then just focus on one study and
15 say, well, now, this -- this, you know, negates
16 everything else and I only believe -- you know, I
17 only believe what's in that study.

18 And so, you know, one study can, in
19 fact, have a lot of information in it that can
20 determine a specific outcome measure, and that
21 could be, you know -- that specific outcome
22 measure could determine -- could weigh very
23 heavily in terms of how a -- you know, how a
24 specific conclusion is reached.

25 But, you know, to say that you just

1 forget all the other -- all the other information
2 that's, you know, published in the field would be
3 very difficult to do. So -- so all of that's --
4 I guess it's a little bit of a semantic problem
5 to say that, you know, you determine something
6 all in one, because we don't really do that in
7 the -- in the research field.

8 Q And you also stated in the sentence
9 "TCE exposure under conditions described at
10 Camp Lejeune, North Carolina."

11 What were the levels of TCE at
12 Camp Lejeune?

13 A So I noted in here the
14 366-micrograms-per-liter amount.

15 I just want to double-check it's not
16 milligrams.

17 366 micrograms per milliliter that was
18 reported in the --

19 I'll just get the citation. That's
20 reported in the Bove et al. 2014 drinking water
21 contamination. So -- so that's based on that and
22 the information that's been reported on, you
23 know, this TCE contamination at this site.

24 So, again, you know, I'm not an
25 epidemiologist. I don't model this stuff. But

1 from my -- so -- so from my position as a
2 toxicologist looking at, you know, a site that's
3 been heavily contaminated with -- with TCE, the
4 increased Parkinson's association from that area
5 and then the work that we do experimentally,
6 that's how, you know, you -- that's how I state
7 that they support a conclusion that the TCE
8 exposure under those conditions is reached.

9 Q I just want to make sure that we have
10 the right unit of measure here. You state in
11 your report on page 2 0.366 milligrams per liter.

12 A Yes. I think that might be a typo.
13 And that's why I was going back to check, because
14 I also have micrograms per liter. I think it's
15 micrograms per liter. I'll double-check. Could
16 be milligrams per liter. It's published in the
17 Bove study, so we should be able to find the
18 actual amount.

19 And nobody knows off the top of their
20 head what it is?

21 MR. TELAN:

22 I'll try to find it.

23 A I think it's micrograms. It's --

24 The reason why that's confusing is
25 because, on a Mac, when you go to type the

1 shortcut for macro, it's command M. And, so, if
2 you don't hit it just right, it will just put an
3 M in the space.

4 But this 2014 study is all in
5 micrograms per liter. So the citation on page 5
6 is the correct one, instead of milligrams.

7 MS. PLATT:

8 Q On page 5 of your report.

9 A Yeah. So if you look at --

10 That corresponds with what's measured
11 in the Bove study that's cited there.

12 Q Which is 366 micrograms per liter;
13 correct?

14 A Yes.

15 Q So the other instances on page 2 and
16 page 10 of your report would be incorrect?

17 A Yeah. I think those are typos. Let me
18 just make sure.

19 It's micrograms. It's in the
20 Goldman -- in the first paragraph of the Goldman
21 study.

22 Q Of 2023.

23 A Yes.

24 Q So just so we're clear on the record,
25 the reference on page 2 and the reference on page

1 10 to 0.366 milligrams per liter should be 366
2 micrograms per liter; correct?

3 A That is correct. Where the -- where
4 the typo is is actually in the -- in the decimal
5 point. So it shouldn't be --

6 It should be 366 micrograms per liter
7 or .366 milligrams per liter. Does that make
8 sense? So it's just where you put the decimal
9 place. I'm just making sure I'm using micro and
10 milli annotated correctly --

11 Q Okay.

12 A -- in there. They're the same number.
13 It's just where the decimal is. I just want to
14 make sure it's consistent. So apologies for
15 looking through this a thousand times.

16 Q Why don't we go ahead and take a break,
17 and you can look through that and we can, on the
18 record, fix it.

19 A Perfect.

20 Q Thank you.

21 VIDEOGRAPHER:

22 Going off the record. The time is 2:19
23 p.m.

24 (OFF THE RECORD.)

25 VIDEOGRAPHER:

1 We're back on the record. The time is
2 2:29.

3 MS. PLATT:

4 Q Dr. De Miranda, we were talking about
5 this median reported in your reports. Do you
6 have a correction?

7 A I actually don't have a correction.
8 It's the same value. I reported it as 0.366
9 milligrams per liter in two places, and in one
10 place I reported it as 366 micrograms per liter,
11 which is the same value. I was confused over the
12 decimal point and just wanted to make sure I was
13 correct. And sometimes you think you're wrong
14 but you're actually right. And that was the case
15 in this case.

16 Q Thank you, though.

17 A Thank you for letting me check that.

18 Q You are not an expert in water
19 modeling; correct?

20 A That's correct.

21 Q And you relied on Dr. Bove's 2014
22 cohort study of Marines to cite to the level of
23 TCE; correct?

24 A That's correct. I cited the level in
25 that report.

1 Q And Dr. Bove relied on ATSDR's water
2 modeling to find that median; correct?

3 A I believe so.

4 Q Are you aware that ATSDR stated that
5 its water modeling cannot be used to estimate an
6 individual's exposure to contaminants in the
7 water system?

8 MR. TELAN:

9 Objection to form.

10 A Yes, I'm aware.

11 MS. PLATT:

12 Q And you still relied on that
13 information?

14 A Yes. I'm not -- I didn't refer to
15 individual contamination levels anywhere in my
16 report.

17 MS. PLATT:

18 Q Are you aware that the ATSDR has stated
19 that the results do not reflect the actual
20 exposure of any specific individuals to
21 contaminants in the water system?

22 MR. TELAN:

23 Object to the form.

24 A Yes.

25 MS. PLATT:

1 Q Are you aware there were eight water
2 systems at Camp Lejeune?

3 A Yes.

4 Q And are you aware that only two of
5 those eight water systems are alleged to be
6 contaminated?

7 A Yes.

8 Q And are you aware that ATSDR modeled
9 contamination for only two of those eight water
10 systems?

11 A Yes. I'm aware that that's how they
12 did the modeling.

13 Q And ATSDR concluded that -- are you
14 aware that ATSDR concluded that the remaining
15 five water systems were not contaminated?

16 A Yes. I think I've read that in the --
17 in the water report.

18 Q Are you aware that there were very few
19 measurements made of chemicals in the water at
20 Camp Lejeune during the overall time period of
21 interest, which is 1953 to 1987?

22 A Yes. I've been -- I'm aware of -- that
23 a lot of the data that comes from that is from
24 modeling. I don't have -- like, I don't have
25 data on the actual numbers beyond what was in

1 the -- in the Bove report on actual values that
2 were pulled out. So I'm aware of the modeling.

3 Q On page 4 of your expert report, the
4 second paragraph under the heading "Environmental
5 exposure and PD risk," do you see that?

6 A Uh-huh.

7 Q The first sentence, you state "a number
8 of environmental contaminants increased risk for
9 PD, such as pesticides, metals, and industrial
10 solvents, include" -- sorry -- "including TCE."
11 Did I read that correctly?

12 A Yes.

13 Q Would you agree that, in studying TCE
14 exposures, it is important to know if a person is
15 exposed to these other environmental exposures,
16 such as pesticides?

17 MR. TELAN:

18 Object to the form of the question.

19 A So if you're talking about an
20 individual person exposed to TCE, on the
21 individual basis, having an exposure history
22 could be -- could be useful.

23 But the way that -- if I could give a
24 metaphor. The way that I usually think about
25 Parkinson's risk on an individual basis as it

1 relates to environmental exposure, sort of like a
2 glass filling up. So at the top of the glass is
3 Parkinson's disease. So you have to fill all the
4 way up to the top. And, so, when you --

5 Inside that glass, you start off with a
6 little bit of water. That's your genetic risk.
7 Okay? And then everything that you put into that
8 glass are your environmental risks.

9 And, so, on an individual basis, you
10 might have a slight amount of water going in for
11 different reasons, but then if you sustain an
12 exposure that you believe is strongly implicated
13 or strongly influencing that Parkinson's risk,
14 that would represent the majority of the water in
15 that glass. And, so, without that water, you
16 would have never reached the top, which is the
17 Parkinson's diagnosis.

18 And, so, in the case of understanding
19 what other things someone is exposed to on an
20 individual basis, that can be important. But if
21 they sustain an exposure that's highly linked to
22 TCE or -- ah -- to Parkinson's disease or is
23 specific for -- in terms of a high amount of
24 risk, then that would represent the majority of
25 where that water is filling up in that glass.

1 And, so, on an individual basis, that's
2 kind of the way that you could imagine how these
3 multiple environmental contaminants can all be
4 related to what's ultimately Parkinson's risk but
5 that you could have more or less influence of an
6 individual factor based on a specific person's
7 exposure.

8 Q And so that glass could be filled with
9 exposure from pesticides; correct?

10 A It depends on -- on the context. But
11 in -- it could be -- a pesticide could be one of
12 the pieces that goes into that glass.

13 Q And, as we discussed today, you relied
14 on Goldman 2012, Goldman 2023, and Goldman 2024;
15 correct?

16 A Yes.

17 Q And these studies by Dr. Goldman did
18 not consider the exposure history of any
19 individuals; correct?

20 MR. TELAN:

21 Object to form.

22 A Not to my knowledge.

23 MS. PLATT:

24 Q Are you aware that up until 1977 TCE
25 was used as a general anesthetic, skin

1 disinfectant, grain fumigant and coffee
2 decaffeinating agent?

3 A Yes. Those are some of the uses of
4 TCE, yes.

5 Q Are you aware that TCE has been found
6 in up to 30 percent of US drinking water
7 supplies?

8 MR. TELAN:

9 Object to the form.

10 A So at one point it was found in up to
11 30 percent.

12 MS. PLATT:

13 Q Are you aware that PCE has been a
14 leading dry cleaning solvent since the 1950s?

15 A Yes.

16 MR. TELAN:

17 Form.

18 MS. PLATT:

19 Q Are you aware that PCE is found in many
20 household products?

21 A So at one point PCE was found in
22 household products. My understanding is that
23 most PCE is starting to -- either has been or is
24 being taken out of products that are sold in the
25 house, used in the house.

1 Q On page 4 of your report, the fourth
2 paragraph, second sentence starts "a primary
3 site."

4 Do you see that?

5 A Yes.

6 Q "A primary site of exposure for TCE
7 following inhalation is the brain and central
8 nervous system (CNS) which causes the immediate
9 neurological effects reported from acute TCE
10 exposure -- dizziness, sleepiness, and reduced
11 reaction time."

12 When you say "primary site for
13 exposure," do you mean primary site for effects?

14 A It says "a primary site for exposure
15 for TCE following inhalation." So it's -- you
16 know, effects and exposure are sort of --

17 In this case, I mean exposure; right?
18 So you're inhaling it, and then your brain's
19 getting exposed.

20 And, then, the effects, which I then
21 say in the next part of that sentence, "which
22 causes the neurological effects." So I -- I
23 guess, to answer your question, I do mean
24 exposure, but I also in that same sentence have
25 stated effects.

1 Q But the brain is not directly exposed
2 to TCE; correct?

3 MR. TELAN:

4 Object to the form.

5 A So -- so when you say "directly
6 exposed," I guess it sort of depends on what you
7 mean. So -- so in the sense that, like, if you
8 were dipping your hand in the TCE, your hand
9 would be directly exposed. But when you inhale
10 TCE, it would presumably go through the olfactory
11 bulb and into the brain.

12 So in that sense, it is sort of
13 directly exposed. But, you know, in the sense of
14 the inhalation, it would be maybe more forward in
15 the -- forward in the nasal cavity, and that
16 might be sort of the first site of exposure and
17 then through the olfactory bulb and into the
18 brain.

19 Q Thank you.

20 And you cite to --

21 Sorry. What levels of concentration
22 are associated with dizziness, sleepiness, and
23 reduced reaction time?

24 MR. TELAN:

25 Objection. Form.

1 A So I don't have a number for you on
2 that, but that was -- that's been cited in
3 studies that have actually shown the acute
4 neurologic effects of TCE, which you mentioned it
5 was used as an inhaled anesthetic at one point.
6 And, so, that's when -- when you use TCE for
7 those purposes, like as an anesthetic, or if
8 somebody's inhaling a really high dose of TCE,
9 those anesthetic effects happen acutely.

10 Which, again, the point of that is to
11 demonstrate that TCE has a neurologic effect.
12 But I -- I don't know specifically which doses
13 were -- are cited in that -- in that citation.
14 Like, I don't know which doses are being -- being
15 compared. But that's --

16 Yeah. That's from, like, a more
17 general effects of TCEs. That's just
18 demonstrating that TCE does have an effect in the
19 brain that produces neurologic effects when it's
20 inhaled.

21 Q And you cite to footnote 42; correct?

22 A Yes.

23 Q And is that an incomplete citation for
24 footnote 42?

25 A Well, that's a -- that's an -- I think

1 it's an OSHA website, Occupational Safety and
2 Health. So that's -- that's -- would be like an
3 OSHA website.

4 Q Would you be able to provide the
5 website address?

6 A Yes.

7 MS. PLATT:

8 We'd request that be provided after the
9 deposition.

10 MR. TELAN:

11 We'll agree, but not immediately after
12 the deposition.

13 MS. PLATT:

14 Q And, again, OSHA measures occupational
15 exposure; correct?

16 A That's correct.

17 And I can't say for sure that that's an
18 OSHA website. I'm sort of assuming it is, but I
19 can't say for sure.

20 But in your second context, question,
21 yes, OSHA measured occupational.

22 Q Did you write your reference list?

23 A I use a citation manager.

24 Q But you managed that citation manager?

25 A It's End -- it's EndNote. It's on my

1 copy of EndNote that's on my computer.

2 Q Page 10 of your report, the second
3 paragraph, about halfway down the paragraph
4 there's a sentence that starts "in addition."

5 Do you see that?

6 A Yes.

7 Q "In addition, inhalation is a more
8 direct route to the brain, whether through the
9 olfactory bulb or through circulatory perfusion
10 from the leg -- from the lungs."

11 Did I read that correctly?

12 A Yes.

13 Q And you cite to footnote 112; correct?

14 A Yes.

15 Q And that is an article by Lucchini;
16 correct?

17 A Yes.

18 Q That publication is about particles in
19 the air pollution; correct?

20 A It's pollutants and the nose-brain
21 connec- -- connection.

22 Q And it is not about solvent exposure;
23 correct?

24 A That's correct. And the -- the -- in
25 the sentence, I write I am writing about

1 inhalation specifically and not solvents.

2 Q And, then, continuing on page 10, the
3 last paragraph on that page, second sentence, it
4 begins with "human exposure."

5 Do you see that?

6 A Yes.

7 Q "Human exposure to chlorinated solvents
8 rarely occurs in isolation, and exposure to a
9 mixture of chlorinated solvents -- for example,
10 simultaneous TCE and PCE exposure -- could be
11 additive or synergistic neurotoxicity."

12 Did I read that correctly?

13 A Could cause additive or synergistic
14 neurotoxicity.

15 Q Could cause additive or synergistic
16 neurotoxicity.

17 A Yes.

18 Q Thank you.

19 And you cite to footnote 119; correct?

20 A Yes.

21 Q And that is an article by Roell?

22 A Yes.

23 Q Does this article by Roell mention TCE?

24 A I -- I'd have to look. I believe this
25 was more of a -- an overview article on the

1 principle of additive or synergistic toxicity.

2 Q So that article does not state whether
3 simultaneous exposure to TCE and PCE could cause
4 additive or synergistic neurotoxicity; correct?

5 A Right. So I'm talking about the
6 principles of additive or synergistic toxicity
7 there.

8 Q What is additive neurotoxicity?

9 A It's when the combined effect of two or
10 more chemicals is the sum of each neurotoxic
11 response individually.

12 Q To have additive neurotoxicity, each
13 chemical must independently cause the health
14 outcome at issue; correct?

15 MR. TELAN:

16 Object to the form.

17 A "Must each independently cause the
18 health" --

19 Sorry. Would you mind repeating that?

20 MS. PLATT:

21 Q To have additive neurotoxicity, each
22 chemical must independently cause the health
23 outcome at issue. Correct?

24 MR. TELAN:

25 Same objection.

1 A Each chemical must independently cause
2 the same.

3 Um, no, they don't have to cause the
4 same -- the same outcome independently. It could
5 be different. You could have two different toxic
6 effects, and they could still be additive in
7 terms of -- of how you sum the -- the -- the
8 total toxicity that you're measuring.

9 Q So if you have one chemical that causes
10 neurotoxicity in the liver and another chemical
11 that does not cause neurotoxicity in the liver,
12 would you see additive effects?

13 MR. TELAN:

14 Object to the form.

15 A So --

16 Sorry. I'm getting a little hung up.
17 So neurotoxicity wouldn't be in the liver. It
18 would be in the brain.

19 But -- but if you're referring to
20 toxicity, toxicity in the liver, so -- so
21 you're -- you're saying one does cause toxicity
22 in an organ and one doesn't cause toxicity in an
23 organ? Is that what you're referring to?

24 Q Yeah. Why don't I rephrase the
25 question?

1 A Okay. Okay.

2 Q If you have one chemical that causes
3 neurotoxicity in the brain and another chemical
4 that does not cause neurotoxicity in the brain,
5 would you have additive neurotoxicities?

6 MR. TELAN:

7 Form objection.

8 A So, broadly, that would -- broadly,
9 which I think is the point you're trying to get
10 at, the answer is no. You're using neurotoxicity
11 in some of the ways I use neurotoxicity. What we
12 should really be talking about is the specific
13 toxic effect that we're -- we're looking for.

14 So where this gets a little bit of a
15 gray area is, say -- say you have chemical X and
16 it causes mitochondrial dysfunction, and say you
17 have chemical Y and it causes lysosomal
18 dysfunction. They don't have to have the same
19 effects for them to each have an effect. Right?
20 And so that's kind of what I mean by they don't
21 have to have the same effect. One could be
22 causing mitochondrial toxicity, the other cause
23 lysosomal toxicity, and when you combine them,
24 you get an additive effect, meaning you get the
25 same amount of mitochondrial and lysosomal

1 toxicity that you would expect with two things in
2 the same system. That's what I'm referring to
3 when I talk about the different effects.

4 If you're talking about something
5 having no effect, then that's a different
6 principle of -- of additive toxicity. And, so,
7 that would not be -- that would not be the case.

8 So you -- you wouldn't necessarily
9 compare a chemical that has a specific, you know,
10 outcome measure of toxicity and one that doesn't
11 have toxicity, because -- to the point I think
12 you were trying to get at, you don't really
13 compare a chemical that doesn't cause toxicity to
14 a chemical that has toxicity.

15 But it's -- it's, you know, it's
16 obviously complex, because you can -- you have
17 many different types of things going on. So
18 neurotoxicity is sort of referring to the entire
19 toxic effect that's going on in the brain versus
20 the specific pathway that any given chemical is
21 interacting with.

22 Q Did you review ATSDR's public health
23 assessment for Camp Lejeune?

24 A I -- I have seen it. I have not
25 reviewed it in depth.

1 Q Are you aware that ATSDR's public
2 health assessment determined that the chemicals
3 at Camp Lejeune would not have an additive
4 effect?

5 MR. TELAN:

6 Object to the form of the question.

7 A I think I did see that in -- I may have
8 seen that in that report, yes.

9 MS. PLATT:

10 Q On page 11 of your report, the bottom
11 of the first paragraph, the sentence starts
12 "experimentally."

13 Do you see that?

14 A Uh-huh.

15 Q "Experimentally, PCE demonstrates
16 similar ability to induce oxidative stress in
17 cells as compared to TCE. Thus, it could be
18 predicted that in a system where TCE, PCE, and
19 benzene are present, a greater degree of toxicity
20 could arise from (1) elevated oxidative stress,
21 and (2), reduced antioxidant enzymes to minimize
22 oxidative damage to cellular macromolecules."

23 Did I read that correctly?

24 A Yes.

25 Q You noted that it could -- you noted

1 that it could be predicted; correct?

2 A Yes.

3 Q So it is not proven; correct?

4 A Correct.

5 Q And then you state, "thus, these
6 support" --

7 Sorry.

8 "Thus, these data support a premise
9 that exposure to multiple solvents simultaneously
10 would induce worsened neurotoxicity than exposure
11 to any one solvent in isolation."

12 Did I read that correctly?

13 A Yes.

14 Q And you noted that was a premise;
15 correct?

16 A Yes.

17 Q It has not been proven; correct?

18 MR. TELAN:

19 Object to the form.

20 A So -- so proven in the sense of -- of
21 a -- we have not peer-reviewed published
22 peer-review data on that, yes.

23 MS. PLATT:

24 Q I'm gonna go ahead and mark Exhibit
25 Number 15.

1 (DEPOSITION EXHIBIT NUMBER 15
2 WAS MARKED FOR IDENTIFICATION.)

3 MS. PLATT:

4 Q I'm showing you Exhibit Number 15,
5 which is your article titled "Preventing
6 Parkinson's Disease: An Environmental Agenda."
7 Do you see that?

8 A Yes.

9 Q And you published this in 2022;
10 correct?

11 A Yes.

12 Q And you published this with Dr. Samuel
13 Goldman; correct?

14 A He was a coauthor on the paper.

15 Q And he is the same Goldman we've been
16 discussing today; correct?

17 A Yes.

18 Q And you also have Dr. Gary W. Miller as
19 one of the authors on this article; correct?

20 A Yes. He's also a coauthor.

21 Q And he is one of plaintiffs' other
22 experts in this litigation; correct?

23 A Yes.

24 Q In this article, you discuss TCE
25 extensively, but you do not mention PCE; correct?

1 A Yes.

2 Q Has the chemical composition of PCE and
3 TCE changed since 2022, when you published this?

4 A Chemical compositions do not change,
5 so, no. That would be impossible.

6 Q Water and hydrogen peroxide are only
7 one atom different; correct?

8 A Yes.

9 Q Do you believe that water and hydrogen
10 peroxide are metabolized the same?

11 MR. TELAN:

12 Object to the form.

13 A So in this -- yes. So in this context,
14 no, water and hydrogen peroxide are not similarly
15 metabolized, although --

16 Is water metabolized? I think it's
17 just an electron receptor. I don't know that
18 it's metabolized.

19 Q You would be the one to know.

20 A Okay. Yeah. So -- so I don't think
21 water is metabolized.

22 Q Carbon dioxide and carbon monoxide are
23 only one atom different; correct?

24 A Carbon monoxide. So those have -- yes.
25 So that's CO and CO₂, so one additional oxygen.

1 Q Are they processed by the body in the
2 same way?

3 MR. TELAN:

4 Object to form.

5 A I mean, there's different health
6 effects. So -- so, no, we don't process those.
7 They have -- they have different -- different
8 effects in the body, yes.

9 MS. PLATT:

10 Q Okay. When considering routes of
11 exposure, do you consider all routes of exposure?

12 A Yes.

13 Q And that would be inhalation,
14 ingestion, and dermal exposure; correct?

15 A Yes.

16 Q The target organ toxicity is not the
17 same for exposures from ingestion, inhalation,
18 and dermal routes; correct?

19 MR. TELAN:

20 Object to the form.

21 A Based on what chemical or what
22 exposure, I guess would be the -- the necessary
23 information in order to understand target organ
24 toxicity.

25 MS. PLATT:

1 Q So between the different routes of
2 exposure, the target organ toxicity would be
3 different?

4 A It -- it can be the same, depending on
5 what the chemical is. So because most things
6 are -- are metabolized by the liver, there are
7 probably cases of -- or classes of chemicals
8 where maybe no matter how you're exposed, it's
9 mostly the target organ toxicity is in the liver.
10 So it could be the -- it could be the same,
11 depending on what chemical we're talking about.

12 Q And it could be different?

13 A And it could be different.

14 Q Are you aware of the National Academies
15 [sic] of Sciences?

16 A I know what that is, yes.

17 Q Are you a member?

18 A (Nods negatively.)

19 MS. PLATT:

20 Sorry. Did you get that?

21 THE COURT REPORTER:

22 Yes.

23 MS. PLATT:

24 Q Have you ever served on a committee for
25 the National Academy of Sciences?

1 A I don't believe so.

2 Q Have you ever served as a peer reviewer
3 for the National Academy of Sciences?

4 A I don't think I've peer-reviewed for
5 them. I peer-reviewed a meeting once that may
6 have been associated with National Academies.
7 But I don't -- I don't know that I peer-reviewed,
8 like, a document of theirs. I think it was more
9 of a review from, like, a meeting that occurred.
10 And I don't even know if it was National
11 Academies. But it sort of feels in my brain that
12 that may have been related.

13 Q Okay. How do you consider the National
14 Academy of Sciences' work, generally?

15 MR. TELAN:

16 Object to the form.

17 A How do I consider the work?

18 MS. PLATT:

19 Q Yes.

20 A In what context? Like, in my -- in
21 relationship to my research?

22 Q Do you consider it high-quality work?

23 MR. TELAN:

24 Object to the form of the question.

25 A Um, I think most people think it is.

1 To be honest with you, I don't really consider it
2 very much because it's not really in relation to
3 what I do. But I -- I believe that most people
4 would -- would agree that National Academy's
5 research is among the research that is considered
6 rigorous.

7 MS. PLATT:

8 Q And the National Academy of Sciences
9 has a committee on toxicology; correct?

10 A I believe so.

11 Q Are you familiar with Dr. David Savitz?

12 A I know who that is.

13 Q Are you familiar with his work?

14 A Not --

15 MR. TELAN:

16 Object to the form.

17 A Not really. I kind of know a little
18 bit about what he does. But I --

19 He's an epidemiologist, and I don't --
20 I don't follow his work very closely.

21 MS. PLATT:

22 Q In writing your report for this case,
23 did you consider the National Research Council's
24 report titled "Contaminated Water Supplies at
25 Camp Lejeune: Assessing Potential Health

1 Effects"?

2 A Um, if it's not in my citations, then
3 I'm not sure that I weighed it very heavily in
4 this report.

5 Q Does that mean you did not consider it?

6 MR. TELAN:

7 Object to form.

8 A The -- I can't say that I didn't
9 consider something, because, again, like I --
10 like I've mentioned, I -- you know, I read
11 thousands of papers a year. It probably is
12 something I've read. But it's not necessarily
13 some --

14 If I haven't cited it specifically,
15 it's not necessarily something that I am directly
16 speaking to in the report.

17 But if -- you know, amongst the body of
18 literature that I read, you know, and I -- and
19 come to my conclusions as an experimental
20 neurotoxicologist, that could -- could represent
21 some of the data that, you know, I built some of
22 my foundational data upon, like I mentioned
23 before with other -- other articles.

24 Q But you don't remember specifically
25 reviewing it in writing this report?

1 A I don't.

2 Q Are you aware that, under the Federal
3 Rules of Civil Procedure, your report should
4 include a list of all materials considered, not
5 simply just relied on?

6 MR. TELAN:

7 Object to the form of the question. I
8 think that's a legal question. Calls for a legal
9 answer.

10 You can answer.

11 MS. PLATT:

12 Thank you, Mr. Telan. But, as you
13 know, we're not allowed speaking objections.

14 MR. TELAN:

15 I understand. But your question
16 actually prefaced with the Rules of Civil
17 Procedure. So...

18 MS. PLATT:

19 Q You can answer.

20 A Okay. So -- so, no. So I don't -- I
21 think --

22 And -- and I -- I understand what
23 you're saying. So when you say "consider" in
24 terms of a report, I'm thinking of a different
25 meaning of that. I'm thinking of considering,

1 like, how I view the field of toxicology.

2 So if I do not specifically cite to it,
3 then it is not something that I have, in the
4 terminology that you just used, considered. So
5 I -- I --

6 Again, because I read a lot of
7 papers --

8 And I'm talking more generally about
9 how I come up with my understanding of -- of
10 neurotoxicity, of Parkinson's from exposures.
11 I'm not talking about considered in the way that
12 you -- that you just described.

13 And, so, when you clarified that, my
14 answer would be that, you know, no. If I haven't
15 listed it here, then it's not considered in my
16 report.

17 Q The National Research Council is a
18 branch of the National Academy of Sciences;
19 correct?

20 A I guess so. I don't -- I don't know.

21 Q Are you aware that Dr. David Savitz
22 chaired the National Research Council's committee
23 in writing the report titled "Contaminated Water
24 Supplies at Camp Lejeune: Assessing Potential
25 Health Effects"? Right?

1 A I didn't know that he chaired that.

2 Q Have you served as a testifying expert
3 in the past?

4 A Um, I've only provided depositions in
5 the past. I don't know if that's different than
6 a testifying expert in terms of your definition.

7 Q Have you provided a written report that
8 was disclosed in the litigation?

9 A Yes.

10 Q And how many times have you been a
11 testifying expert?

12 A Twice.

13 Q And what are those times?

14 A So --

15 Sorry. Let me back up. So if you're
16 just talking about reports, I provided three
17 reports on three different cases, and then I've
18 provided deposition on two. So that's where the
19 twice comes from.

20 So should I just say the ones I've
21 given depositions in first?

22 Q Do you know if that third report was
23 disclosed?

24 A It's in --

25 I don't know. I think it has been.

1 But it's in paraquat litigation. So I don't -- I
2 don't know.

3 Q Okay. Let's talk about the two in
4 which you gave deposition testimony.

5 A Okay.

6 Q What were those two matters?

7 A One, we -- I previously mentioned.
8 That was the O'Leary individual case of a person
9 who had Parkinson's disease from a suspected
10 exposure to TCE. And the other was from a case
11 out of West Virginia from a person who had
12 Parkinson's disease from exposure to TCE and
13 other solvents from paint cleaning products.

14 Q And you said the third, in which you
15 produced a report, was paraquat?

16 A Yes.

17 Q Turning to your CV, let's use the CV
18 that's part of your --

19 Well, one second.

20 -- the CV that's attached to your
21 report in this case.

22 A Okay.

23 Q Turning to page 8 --

24 A Uh-huh.

25 Q -- you have listed here "service to

1 legal cases involving Parkinson's disease risk,"
2 and then you list the law firms Hartley Law
3 Group, Metzger -- Metzger Law Group, Bell Legal,
4 and the Miller Firm; correct?

5 A Yes.

6 Q What matter did you work on for the
7 Hartley Law Group?

8 A That's the West Virginia case that I
9 just mentioned about a person with Parkinson's
10 from the paint thinner exposures, the solvent,
11 TCE exposure from some paint cleaners.

12 Q And the Metzger Law Group?

13 A That was the case of the O'Leary case
14 with a person who had Parkinson's disease from
15 TCE exposure. I believe he was a -- it
16 was involving Boeing and Dow were the defendants
17 in that case.

18 Q And Bell Legal Group?

19 A That would be this, pertaining to this
20 case.

21 Q And then the Miller firm?

22 A That's pertaining to the paraquat and
23 Parkinson's risk case.

24 Q This is titled "Legal Cases Involving
25 Parkinson's Disease Risk." Have you prepared any

1 other expert reports beyond this?

2 A Um, I think in the past I've provide --
3 I've provided nonlegal expert reports for people
4 who have had -- who are looking for sort of a
5 relationship between -- between TCE exposure and
6 Parkinson's disease for non- -- I assume nonlegal
7 cases, people who are looking for information for
8 their physicians, for example, who have been
9 contacted from over the years.

10 Q Okay. No expert report that's not
11 related to Parkinson's disease?

12 A No.

13 Q And you classify these as biological
14 plausibility reports. What does that mean?

15 A So, similar to the title of this
16 report, "Biological Mechanisms of Dopaminergic
17 Toxicity from Trichloroethylene Exposure," I deem
18 that to sort of be a biologic plausibility report
19 because I'm providing a biological basis for how,
20 in this case, TCE could be toxic to dopaminergic
21 neurons.

22 Q Do you see that as different than
23 causation?

24 A Um, I think it's -- it could be part
25 of -- of an overall causation in terms of -- of

1 how it's defined legally.

2 So, for example, in this, you know, the
3 report I was asked to provide in the report my
4 opinion on, if -- on whether it was equally
5 likely or more likely than not to be part of the
6 cause or to -- biological mechanism as part of
7 the causation.

8 But the biological plausibility which
9 I'm actually writing in there refers specifically
10 to those mechanisms that I talk about in the
11 report, so like the oxidative stress, the
12 mitochondrial dysfunction, those aspects of it.

13 Q Uh-huh.

14 When were you first contacted about to
15 work on this litigation?

16 A I think in 2023.

17 Q Do you know the month?

18 A It was before June. That's, I think,
19 the first time I have a record of a meeting on my
20 schedule.

21 Q And who first contacted you?

22 A I think it was David Miceli or
23 Pat Telan who contacted me. I don't -- I can't
24 remember who reached out with me first, but I
25 know I spoke with them very early on.

1 Q And when were you retained as an expert
2 in this litigation?

3 A Um, so I agreed to write a biological
4 plausibility report or this biological mechanisms
5 report. It would have been sometime in probably
6 2023. I don't know that I have a specific date
7 on the retained. I don't ask for a retainer fee,
8 so I don't have a date where I send, like, an
9 invoice for that.

10 Q Okay. Did you sign some sort of
11 engagement letter?

12 A I would have initially probably signed
13 an external activity request, where I submit that
14 to my university showing that I will -- what the
15 nature of the -- of what I'll provide is going to
16 be. And, so, that, I -- that could be a document
17 showing that's sort of when I was retained.

18 Q Uh-huh.

19 When did you first sign a document with
20 one of the plaintiffs' firms?

21 MR. TELAN:

22 Object to the form of the question.

23 A I would have to look for the exact
24 date. I believe it was in 2023.

25 MS. PLATT:

1 Q Okay. And who retained you?

2 A Bell Legal.

3 Q And sometime in 2023, you signed a
4 contract with Bell Legal?

5 A I've only signed the external
6 activity -- or we've only signed the external
7 activity. I don't -- I don't think I have a
8 contract that I've specifically signed with them.
9 I did --

10 I guess my fee -- I send my fee
11 schedule, and that has my signature on it. But
12 in terms of contract, I'd have to look to see if
13 there was a contract. It's been a couple of
14 years, and there may have been documents sent
15 over that I am forgetting about.

16 Q Did you receive any written directions
17 regarding the issues you should address?

18 MR. TELAN:

19 Object to the form.

20 A I haven't received any directives of --
21 of information. I received information.

22 MS. PLATT:

23 Q What information did you receive?

24 A I've received information on -- on
25 things like the legal standards in the

1 Camp Lejeune Justice Act, so showing -- to
2 explain to me that though -- like the CLJA exists
3 and to go read that and make sure I understand
4 it, so pointing to some of the resources that are
5 particular to this case as they pertain to
6 Camp Lejeune and not necessarily as my work on --
7 on neurotoxicity. So -- and kind of information
8 on -- on that. Information on --

9 Like, we -- we use -- saw a paper that
10 showed a citation or a paper that I -- you know,
11 would be good for me to look over, information
12 on, like, when -- when my report is due and that
13 sort of thing.

14 Q What specific studies were provided to
15 you?

16 A Um, so -- so the Camp Lejeune Justice
17 Act and the ATSDR report, the --

18 Those -- those are all in there. And,
19 then --

20 Sorry. I'm saying "um" a lot.

21 I'm trying to delineate, like, what I
22 already had in my body of literature versus what
23 was provided, so things that were pertaining
24 specifically to Camp Lejeune, like the
25 Camp Lejeune Justice Act.

1 Again, I -- I -- like, for example,
2 that Sallmén citation, that was sent over -- you
3 know, sent to me to consider. But I've already
4 read that paper, so, you know, having that
5 provided to me is sort of different than
6 having -- you know, it's a paper that exists, so
7 I already knew about.

8 What else? Is there specific -- a
9 specific --

10 No. I -- I have in my sort of, you
11 know, as you see, from my citation manager, most
12 of these citations are already something that I
13 already have. And, so, I don't know if there's a
14 lot beyond those -- those kind of specific
15 Camp Lejeune Justice Act, the ATSDR document that
16 was provided to me or that we sort of talk about
17 more broadly because it's the literature that I
18 use to do my everyday research.

19 Q Uh-huh.

20 You refer to an ATSDR report. Which
21 report are you referring to?

22 A The -- this -- this sort of thick one,
23 and then the water contamination level one that
24 we've -- we've talked about.

25 Q So that one is the Assessment of the

1 Evidence, and I believe that is Exhibit 6.

2 A That's right.

3 Q And then the water contaminant one
4 you're referring to, which is that?

5 A Um, so I think I --

6 It's -- there's the other ATSDR. Is
7 there another ATSDR one? I think I'm just
8 getting confused.

9 This is the water -- this is the water
10 one, and then the -- but this is also the
11 Camp Lejeune Justice Act one. So maybe I'm
12 thinking about the same. Maybe it's the same one
13 that I'm thinking of that has both the water
14 concentration and the Camp Lejeune Justice Act in
15 there.

16 Q Were you thinking of the Bove 2014
17 study?

18 A I may -- I may have been. But this is
19 something that --

20 So I think we even cited this in some
21 of our papers from --

22 Like, I think we cited this in 2022.
23 So, again, this is something I would have had in
24 my citation manager already, but it could have
25 also been something that was discussed in

1 relation to this. So --

2 Q And that's Exhibit Number 8; correct?

3 A Yes.

4 Q Were any other materials provided to
5 you?

6 A I don't --

7 Oh. I've seen a couple of the reports
8 from some of the experts. So I -- I have -- I've
9 seen Julie Goodman's report and -- is it
10 Lipscomb? Is it -- I don't know the first name
11 on that. And I've seen the draft deposition of
12 Gary Miller.

13 I also, I believe, have file folders of
14 the other expert witnesses that -- including from
15 Julie Goodman and John Lipscomb, but I can't
16 recall their names specifically.

17 Q Okay. Did you review the deposition
18 transcript of Dr. Gilbert?

19 A No.

20 Q Were any other materials provided to
21 you by counsel?

22 A I don't -- I don't think so. Again, I
23 can't completely rule out something that was
24 sent, you know, across a couple years of
25 communication. But I don't recall anything

1 offhand.

2 Q All right. Did you perform any work in
3 this matter prior to becoming retained?

4 A I have -- I have not done any work on
5 Camp Lejeune prior to being retained in terms
6 of -- of legal work, no.

7 Q Have you received any compensation in
8 connection with your work on this case?

9 A I've provided one invoice for my -- my
10 time in preparing my written report.

11 Q And that is the invoice that you
12 produced this morning; correct?

13 A Yes.

14 MR. TELAN:

15 Did you mark that, Elizabeth?

16 MS. PLATT:

17 I have not. I'd like to mark it right
18 now. I believe she has a copy of it.

19 MR. TELAN:

20 She has a copy, yes.

21 MS. PLATT:

22 And we have one copy on our side.

23 MR. TELAN:

24 Do you want to mark hers?

25 MS. PLATT:

1 Yes. Do you have a copy?

2 MR. TELAN:

3 I don't need one. It's okay.

4 MS. PLATT:

5 Okay.

6 MR. TELAN:

7 Just don't write on that one she's
8 marking.

9 (DEPOSITION EXHIBIT NUMBER 16
10 WAS MARKED FOR IDENTIFICATION.)

11 MS. PLATT:

12 Q I'm going to mark this as Exhibit
13 Number 16. Here you go.

14 I'm handing you Exhibit Number 16,
15 which is the invoice you produced this morning in
16 response to the subpoena. And you recognize this
17 document as what you produced this morning?

18 A Yes.

19 Q And this invoice is dated December
20 18th, 2024; correct?

21 A Yes.

22 Q Have you submitted another invoice
23 since December 18, 2024?

24 A No.

25 Q Have you billed any time since December

1 18, 2024, on this matter?

2 A So, as you can see from this, I tend to
3 collect my time and then send it over sort of in
4 bulk over many different months. I -- I just
5 keep track of what I work on, and then I bill for
6 time kind of all together once I've accumulated
7 what, I guess, sort of what I deem to be worth
8 sending over at any given time.

9 Q So are you right now accumulating time
10 to be billed in the future?

11 A When I work on matters related to this
12 case, I log the hours that I work.

13 Q Okay. I'm sure your counsel has spoken
14 to you about this, but you do have a duty to
15 supplement productions. So when you do bill for
16 the next accumulation, we would ask that that be
17 produced, in accordance with the subpoena.

18 A Sure.

19 Q And it looks like so far you have been
20 compensated \$7,500. Is that correct?

21 A That's correct.

22 Q And how much do you charge per hour?

23 A \$250 U.S. dollars per hour.

24 Q Do you have a minimum number of hours?

25 A I do not have a minimum number of

1 hours.

2 Q And how do you keep track of the time?

3 A On the day of, I probably -- typically
4 record it in my planner that sits next to my
5 computer, and then I transfer that into an Excel
6 spreadsheet, which has been copied and pasted
7 onto this invoice.

8 Q Are you reimbursed for expenses or
9 costs?

10 A I believe the only expense I ask for
11 reimbursement in my fee schedule is if I have to
12 travel for a deposition. And I have not had to
13 travel outside of Birmingham, so I have not been
14 reimbursed for any expenses or costs.

15 Q Is your compensation in this matter any
16 way dependent on the outcome of the case?

17 A No.

18 Q Do you have support staff with you on
19 this matter?

20 A No.

21 Q What percentage of your animal --
22 annual income is earned from performing expert
23 services?

24 A So for 2024, I would estimate all of my
25 external services would be probably between

1 8-and-a-half to 9 percent.

2 MS. PLATT:

3 Let's take a five-minute break.

4 MR. MICELI:

5 Okay.

6 VIDEOGRAPHER:

7 Going off the record. The time is 3:19
8 p.m.

9 (OFF THE RECORD.)

10 VIDEOGRAPHER:

11 We are back on the record, and the time
12 is 3:31 p.m.

13 MS. PLATT:

14 Q Dr. De Miranda, before we took a break,
15 we were discussing some of the documents that you
16 reviewed in this matter.

17 Did you review the deposition of
18 Dr. Bove?

19 A No.

20 Q Did you review the deposition of
21 Dr. Savitz?

22 A No.

23 Q Did you review the deposition of
24 Maslia?

25 A No.

1 Q Have you had any communications with
2 other experts in this case?

3 A Only my colleagues who I think we've
4 talked about earlier. So I speak with Gary
5 Miller occasionally. I speak with Jason Cannon
6 occasionally. I don't know --

7 Oh. Sorry. Sam Goldman's not an
8 expert in the case, but we -- we talked about him
9 earlier. So those -- I believe that's the limit
10 of any other expert that I know of that is
11 involved. Not Sam but Jason Cannon and Gary
12 Miller.

13 Q Do you have any communications, written
14 communications, with Dr. Miller, prior to him
15 becoming an expert in this matter, about
16 Camp Lejeune?

17 MR. TELAN:

18 Object to form.

19 A I don't think Gary Miller and I
20 conversed about Camp Lejeune prior to this case,
21 although we did write that review together, and
22 it -- there may have been --

23 As you know, there was TCE mentioned in
24 there. And, so, if, like, a paper was passed
25 back and forth between us during that writing of

1 that review, that would have been involving
2 Camp Lejeune potentially but not about this case.
3 So it would have been about the TCE
4 contamination. And that happened after.

5 Q Okay. Do you have any written
6 communications with Dr. Cannon prior -- prior to
7 him becoming an expert in this matter?

8 A I have written communications with him
9 about many things, but not about this case.

10 Q Okay. In rendering your opinion, did
11 you rely on any facts provided by another expert
12 in this case?

13 A No.

14 Q In rendering your opinion, did you rely
15 on any data provided by another expert in this
16 case?

17 MR. TELAN:

18 Object to form.

19 A I relied on published data. And I
20 think you -- some of the people you mentioned
21 were experts. But not from what they produced
22 for this case. Published -- peer-reviewed
23 published data.

24 MS. PLATT:

25 Q Okay.

1 A And it may have come from the -- the
2 people who are -- who could have been involved or
3 are adjacently involved, yes.

4 Q In rendering your opinion, did you
5 consider any facts considered --

6 Sorry.

7 Did you consider any facts provided by
8 another expert in this case?

9 A No.

10 Q In rendering your opinion, did you
11 consider any data provided by another expert in
12 this case?

13 MR. TELAN:

14 Same objection.

15 A No.

16 MS. PLATT:

17 Q Did you do anything to prepare for
18 today's deposition?

19 A Yes.

20 Q What did you do?

21 A I reread a few of the Camp Lejeune
22 documents that I am less familiar with, like what
23 we discussed, the Camp Lejeune Justice Act and
24 the water report that I seem to be struggling to
25 say the full name of occasionally. I reread my

1 report that I wrote. I skimmed through the
2 deposition of Gary Miller, the draft. And I
3 reread a few of the -- of the papers that I --
4 both that I've written as well as some of the
5 other TCE mechanism papers.

6 Q You said "water report." Did you
7 determine which one that is?

8 A So I think that the drinking water --
9 the ATSDR assessment for evidence for the
10 drinking water contaminants at Camp Lejeune and
11 specific cancers and other diseases, that's what
12 I'm referring to when I say "water report."

13 Q Okay. You're not referring to the
14 water modeling report?

15 A So that, the Bo- -- the Bove 2014 water
16 modeling, is that --

17 Sorry. The water mod- -- the Bove
18 water modeling report is also something that --
19 that I have -- that I've read, the evaluation of
20 mortality among Marines. And that has to do with
21 water modeling that's in the micrograms per liter
22 that we talked about earlier. And, so, that --
23 that modeling report is something that I've read.

24 Q And that's a cohort study; correct?

25 A Yes.

1 Q My question was about ATSDR's water
2 modeling for Camp Lejeune.

3 A I see.

4 Q Mr. Maslia, Robert Faye, those
5 individuals published that.

6 A Right. I have read that, yes.

7 Q Okay. And that is separate from the
8 document you're referring to by Dr. Bove.

9 A That's correct.

10 Q Okay. Did you reread the water
11 modeling by Mr. Maslia in preparation for today's
12 deposition?

13 A It was one of the reports I looked over
14 before today.

15 Q Okay. Did you meet with counsel to
16 prepare for this deposition?

17 A Yes.

18 Q How many times did you meet with
19 counsel?

20 A For this, maybe three, three to four
21 times.

22 Q Who did you meet with?

23 A Pat Telan, David Miceli, and I believe
24 Leslie La- -- sorry. I'm sorry, Leslie. But
25 LaMacchia? Sorry.

1 MR. MICELI:

2 Good. Yep.

3 A As a person whose name gets misspoken a
4 lot, I apologize. Those would have been the
5 three people who I've met with most frequently.

6 MS. PLATT:

7 Q And when did the first meeting occur?

8 A For preparation?

9 Q Yes.

10 A I'd have to look at my calendar, but I
11 think it was in -- it was in either late February
12 or early March.

13 Q And how long was that meeting?

14 A Few hours.

15 Q And the second meeting?

16 A How long was it?

17 Q Per --

18 Sorry. When did it occur?

19 A Oh. We met -- I'd have to look at my
20 calendar. I have them all saved in my calendar.
21 So we --

22 We would have met -- I think we met for
23 a few hours on the morning of April 2nd. But
24 I -- I may be a little wrong on the date on that
25 one.

1 Q Okay. But it was early April?

2 A I believe so, yes.

3 Q And, then, the third meeting, when did
4 that occur?

5 A So I -- I met briefly with David and
6 Pat yesterday.

7 Q And how long was that meeting?

8 A Probably an hour and a half.

9 Q And other than Mr. Telan, Mr. Miceli,
10 and Miss LaMacchia, was anybody else present at
11 these meetings?

12 A Um, I don't think so. Pat's paralegal,
13 Lori Merz, occasionally is on some of those Zoom
14 calls, but I don't recall if she was on every --
15 every Zoom meeting that we've had.

16 Q Did counsel instruct you to inspect any
17 specific documents?

18 MR. TELAN:

19 Object to the form.

20 A I reviewed --

21 Did they instruct me to review any
22 specific documents? They've given me information
23 on what would be good to review, like -- like
24 just talking about the Camp Lejeune Justice Act,
25 taking -- like, talking about different ATSDR

1 studies and things related to Camp Lejeune.

2 We -- we discussed already, but they
3 brought to my attention the Sallmén study. And I
4 think that's -- that's pretty much anything that
5 I -- that I've reviewed in terms of literature.
6 I don't --

7 I mean, I botched that a little bit
8 because I don't remember, frankly, what you asked
9 in that question. You asked for literature.

10 Q Just anything they instructed you to
11 review --

12 A Okay.

13 Q -- in preparation for this deposition.

14 A Okay. Yes. So reviewing would be
15 things like Camp Lejeune Justice Act and ATSDR
16 report.

17 Q Okay. You mentioned that you reviewed
18 the expert report for Dr. Goodman. Is that
19 correct?

20 A Yes.

21 Q And was that her Parkinson's disease
22 report?

23 A Yes.

24 Q Did you review her other reports?

25 A No.

1 Q Do you have a response to Dr. Goodman's
2 Parkinson's disease report?

3 MR. TELAN:

4 Object to the form.

5 A I don't have any specific response.

6 MS. PLATT:

7 Q And you said that you reviewed the
8 expert report of Dr. Lipscomb; correct?

9 A I've -- I've looked at it. I haven't
10 what you would consider reviewed it.

11 Q Would you say you skimmed it?

12 A I skimmed it.

13 Q Okay. Do you have a response to
14 Dr. Lipscomb's expert report?

15 A No.

16 MS. PLATT:

17 No further questions from me. Thank
18 you so much for your time.

19 MR. MICELI:

20 Let's take a break.

21 MR. TELAN:

22 Okay. Just a few minutes, and then
23 we'll jump back in.

24 VIDEOGRAPHER:

25 Going off the record. The time is

1 3:41.

2 (OFF THE RECORD.)

3 VIDEOGRAPHER:

4 Back on the record. The time is
5 3:55 p.m.

6 EXAMINATION

7 BY MR. TELAN:

8 Q Dr. De Miranda, let me -- let me start
9 with your background and training. And I'm not
10 gonna have you rehash what you've already told
11 us. But I'm interested in -- in hearing from you
12 how you got into the study of TCE and Parkinson's
13 specifically. So if you would take us, perhaps,
14 from your -- your postdoctorate through your --
15 your developing interest in trichloroethylene and
16 Parkinson's disease to where you are today,
17 that's kind of what I'm looking for.

18 A Sure.

19 So prior to my current work on TCE and
20 Parkinson's disease, I studied neurotoxic effects
21 of a number of different chemicals on the brain
22 in relation to Parkinson's neurodegeneration,
23 including pesticides, the pesticide rotenone in
24 particular. That's what I was focusing on in the
25 early part of my postdoc at the University of

1 Pittsburgh. And that's where a lot of the LRRK2
2 work comes out of is this understanding of, you
3 know, involvement of proteins that could
4 influence how these exogenous risks are eliciting
5 their effect on Parkinson's pathology in the
6 brain.

7 And I have a lot of different research
8 on rotenone and other -- other neurotoxic
9 elements that are associated with Parkinson's
10 disease, but TCE came up kind of organically when
11 I was having a conversation with my postdoc
12 mentor, Tim Greenamyre. We were talking about
13 how there's been this association with TCE and
14 Parkinson's risk, but no one's really looked at
15 it in depth in a model system beyond the initial
16 early 2010s papers that -- that treated rats and
17 mice with those high doses of TCE that looked at
18 neurodegeneration.

19 Our lab was really -- I don't want to
20 say better equipped, but we -- we -- we were. We
21 were sort of better equipped to build that system
22 out because I was at, you know, a highly
23 prestigious Parkinson's lab at University of
24 Pittsburgh. And, so, we used our kind of
25 expertise to develop a -- what we considered a

1 newer model system where we incorporated aged
2 rats into a TCE exposure study using a lower dose
3 than what had been reported.

4 And we were initially a bit surprised
5 that we saw dopaminergic neuron loss in that
6 initial study. And that's what's now published
7 in the 2021 De Miranda et al. paper in
8 Neurobiology of Disease.

9 We -- we had expected to maybe see
10 some -- some neurologic damage, but not
11 necessarily meet the threshold of dopaminergic
12 neuron loss. But when we saw there was neuron
13 loss, we recognized that what we were looking at
14 was a toxicant that has this association with
15 Parkinson's disease, similar to other model
16 systems.

17 Like rotenone can be exposed
18 systemically and then lead to the selective loss
19 of dopamine neurons, which is really kind of an
20 incredible feature. There's only a few, you
21 know, chemicals out there that you can expose an
22 animal systemically to a chemical and the result
23 is the loss of dopamine neurons.

24 So from there, both myself and my
25 postdoc mentor really realized that we had

1 something that we needed to expand on. And so we
2 started developing out, you know, writing grants
3 and writing papers on this.

4 And, then, from there, I was -- I had
5 applied for and was awarded a peer transition
6 grant by the NIH in order to study more on this
7 effect of TCE and these mechanisms of
8 neurotoxicity in relation to Parkinson's disease.

9 And so that career transition grant is
10 how I came to UAB, which is -- because of a large
11 concentration of Parkinson's researchers here.
12 And I started my lab here doing sort of the --
13 the next phase of that research that I'm doing
14 now.

15 Q So was it through this grant from the
16 National Institute [sic] of Health that actually
17 got you started in your what is now your lab at
18 UAB?

19 A It was one of the things that -- that
20 helped get me established. I had a couple of
21 grants, one from the Parkinson's Foundation, one
22 from NIH, that helped establish my independent
23 lab, yes.

24 Q And do you -- do you continue to
25 receive grants from both the NIH and the

1 Department of Defense to research Parkinson's
2 disease?

3 A Yes. Those are two of the sources
4 where I get some of my funding from.

5 Q And do you have an estimate over your
6 career as to how much funding money you've
7 received from the U. S. Government, either
8 through the Department of Defense, NIH, or its
9 agencies, for your work on Parkinson's?

10 A So I don't, added up, in totality. I
11 could probably estimate a couple of the larger
12 grants, I mean.

13 So the career transition grant that I
14 talked about, so that gave me about -- I think
15 it's just under a million dollars in direct costs
16 for the time that I had that grant. My R01 is, I
17 think, somewhere between, like, high one million
18 and two million.

19 And then I have a half-a-million-dollar
20 three-year DOD grant, and I just got another
21 half-a-million-dollar DOD grant. And those are
22 all focused on TCE and the neurotoxic mechanisms
23 involved in Parkinson's disease.

24 Q So the U.S. Government is actually
25 awarding you money to study TCE and Parkinson's

1 disease?

2 MS. PLATT:

3 Objection.

4 A Yes.

5 MR. TELAN:

6 Q And -- and -- and has that occurred
7 even in the last year?

8 A Yes.

9 Q And was that from the Department of
10 Defense?

11 A One of my -- one of the grants I hold
12 from the DOD was awarded in 2023, and the other
13 one that we just received a notice of award for
14 that I'm a coinvestigator on was awarded in 2025.

15 Q You had mentioned the transition grant.
16 When you came to the University of Alabama, did
17 you bring a lab with you? Did they build a lab
18 out for you here? How did your lab here get
19 formed?

20 MS. PLATT:

21 Objection.

22 A So I -- I brought equipment with me
23 from the University of Pittsburgh that I could
24 purchase from my grants, and then I purchased
25 more equipment and more resources when I got here

1 through my grants and through my startup funds
2 from UAB in order to build out the lab that I
3 have today.

4 MR. TELAN:

5 Q And what is --

6 Sorry. Go ahead.

7 A Sorry. I was gonna say if that answers
8 your question.

9 Q It does.

10 And what is the name of your lab here
11 at the University of Alabama?

12 A The De Miranda Lab.

13 Q And how many labs, if you know, around
14 the country study the relationship of TCE and
15 Parkinson's disease?

16 A From a -- experimental research?

17 Q Yes.

18 A Um, so I can only think of a couple --
19 one that it's not its main focus. It's sort of a
20 side project on TCE and Parkinson's, and it's
21 really to a metabolite of TCE, trichloroacetic
22 acid. There are probably others, but there's not
23 been -- there hasn't been published papers on
24 them, so I don't -- I can't say --

25 Someone might be working on something.

1 Whether or not that relates to a publication is
2 another story. So -- so that may -- whatever
3 they're working on may not result in a
4 publication. And, so, I -- until someone
5 publishes something else, I would say, you know,
6 very limited number of -- of TCE and Parkinson's
7 labs.

8 Q I know you are a humble person, but is
9 there another scientist in this country currently
10 that is -- is publishing the quantity of
11 literature on TCE and Parkinson's disease as a
12 result of literature -- liter- -- I'm sorry -- as
13 a result of scientific research being done in
14 labs like yours?

15 MS. PLATT:

16 Objection.

17 A Um, I'm not aware of anyone else that
18 meets that criteria. Again, you know, I'm a
19 basic scientist. I mentioned earlier I'm not an
20 epidemiologist. So I am --

21 In terms of the constraints that, you
22 know, talking about mechanisms or experimental
23 conditions looking at Parkinson's
24 neurodegeneration from TCE exposure, I'm not
25 aware of any others that are currently doing

1 that.

2 MR. TELAN:

3 Q The name Julie Goodman has come up
4 during the course of the deposition. Before we
5 sent you a report from Julie Goodman, had you
6 ever heard her name mentioned by any scientist
7 who studies Parkinson's disease, that you know
8 of?

9 MS. PLATT:

10 Objection.

11 A No.

12 MR. TELAN:

13 Q Have you ever met Dr. Goodman?

14 A Not to my knowledge.

15 Q Has she ever written you to ask to come
16 visit your lab?

17 A No, I don't believe so.

18 Q Now, you did review her report; right?

19 A Yes.

20 Q I think Miss Platt asked you if you had
21 any response to that. Let me ask you a little
22 bit more pointed of a question.

23 Do you have any disagreements that come
24 to the top of your mind about your review of her
25 report?

1 MS. PLATT:

2 Objection.

3 A Um, yes. I -- I disagree with a number
4 of points of what she wrote in that report in
5 terms of her -- how she evaluated the literature
6 and my work as well.

7 MR. TELAN:

8 Q As you read through her commentary on
9 your report, how would you gauge her level of
10 understanding of the molecular underpinnings of
11 how TCE causes Parkinson's disease?

12 MS. PLATT:

13 Objection.

14 A Um, so, you know, for me, it's -- I
15 guess what was most clear is that Dr. Goodman is
16 not a Parkinson's biologist, obviously more of a
17 general toxicologist, and, because of that, there
18 are several areas in the report where there is a
19 focus on things like number of dopamine neurons
20 lost that correlates more to, like, a person
21 who's coming to terms -- coming to autopsy with
22 Parkinson's versus how we calculate statistical
23 significance of dopaminergic neuron loss in an
24 experimental model.

25 I was surprised a little bit and, you

1 know, I guess a little bit --

2 One of the things I think was -- was
3 missed was that, you know, throughout my report,
4 I remark several times about other pathological
5 features of Parkinson's disease that TCE is
6 related to, and that really was not -- I don't
7 feel like that was -- was -- none of those
8 features were described in her report.

9 So, for example, alpha-synucleic
10 accumulation and talking about the loss or
11 endolysosomal function, things that are really
12 intrinsic to Parkinson's disease that I view, you
13 know, as a Parkinson's researcher to be really
14 critical; the Parkinson's synucleinopathy.

15 And it's really hard to just talk about
16 Parkinson's disease or Parkinson's neurotoxicity
17 and only talk about dopaminergic neuron loss. I
18 mean, I feel like that's only half the story.

19 And, so, I feel like not -- you know,
20 not acknowledging that there's a clear connection
21 there sort of does the same thing that, you know,
22 I think she was critical about, which is you're
23 not -- you know, she's only taking part of the
24 story and -- and, you know, not -- sort of
25 ignoring the rest. So that's --

1 You know, I think because I'm a
2 toxicologist and also Parkinson's biologist, you
3 know, I sort of have a foot in either side of
4 the -- of the field. I really think that she
5 missed a lot of what would be considered, you
6 know, really important Parkinson's biology points
7 in there.

8 Q Can you talk to us a little bit how the
9 thinking on Parkinson's has evolved even since,
10 let's say, 2010 in terms of the causality of
11 Parkinson's disease?

12 A So in the history of sort of
13 Parkinson's research, we've -- we've thought
14 Parkinson's was -- we went from having a disease
15 that initially was thought to be potentially this
16 idiopathic, or unknown, disease to a disease that
17 some people thought was gonna be completely
18 solved by genetics. So there was this big push
19 to do all the sequencing of all these people.
20 Even now there's billions of dollars of research
21 being funneled into genetic testing of people.

22 And, so, from 2010 till 2025, so 15
23 years, over that time, what we've found
24 remarkably with all these resources being poured
25 into that is that genetics do not well explain

1 Parkinson's disease. And, so, that reforming of
2 Parkinson's -- of Parkinson's heritability not
3 being a strong feature is now, I think, pretty
4 clearly, as I mentioned, you know -- now we're
5 defining idiopathic as a gene-environment
6 interaction. At the same time, we have a lot
7 more data showing that there's neurotoxic
8 mechanisms that can deeply contribute to -- to
9 this risk.

10 So I would say since -- in the last 15
11 years, we've really seen a shift from something
12 that thought it could be solved with genetics to
13 something that's definitely more a clear
14 gene-environment interaction.

15 Q And while you're on the subject of
16 genetics, the subject of Dr. Goldman's 2020 study
17 came up, the twin study. What was the import of
18 that particular study as it related to the
19 contribution of genetics or perhaps lack of
20 contribution of genetics toward Parkinson's
21 disease causality?

22 A So having PD-discordant monozygotic
23 twins suggests that individuals who share the
24 same DNA, when you have one person who has
25 Parkinson's and the other person doesn't, it's --

1 it tells you that Parkinson's is not a widely
2 inherited disease. So it gives a lot of strength
3 to the idea that exogenous factors or
4 environmental factors is playing a role in
5 Parkinson's risk outside of genetic or inherited
6 factors.

7 Q You were asked, I think, about the
8 National Academy of Science [sic] and a report
9 they generated in 2009. Has the National Academy
10 of Science [sic] sent any of its toxicologists
11 over to your lab?

12 MS. PLATT:

13 Objection.

14 A No.

15 MR. TELAN:

16 Q Did any of them reach out to you about
17 the causes of Parkinson's disease specifically or
18 generally?

19 MS. PLATT:

20 Objection.

21 A I don't recall ever being contacted
22 about it.

23 MR. TELAN:

24 Q Okay. In terms of Goodman, have you
25 ever seen any original research by Dr. Goodman

1 related to Parkinson's disease causality?

2 A Not that I recall.

3 Q You had mentioned -- there were several
4 folks that we've talked about today.

5 Tim Greenamyre, is he in the fraternity of people
6 that contribute regularly to Parkinson's --
7 Parkinson's disease research?

8 MS. PLATT:

9 Objection.

10 A Yes. I think, based on your
11 description, that would be accurate.

12 MR. TELAN:

13 Q Sam Goldman?

14 MS. PLATT:

15 Same objection.

16 MR. TELAN:

17 Q Is Dr. Goldman one as well?

18 MS. PLATT:

19 Objection.

20 A Yes.

21 MR. TELAN:

22 Q Dr. Miller?

23 MS. PLATT:

24 Objection.

25 A Yes.

1 MR. TELAN:

2 Q Are there any others that come to mind
3 in -- in the field of neurotoxicology that
4 regularly contribute to Parkinson's disease
5 causality?

6 MS. PLATT:

7 Objection.

8 A I mean, there are a number of
9 neurotoxicologists that are within my
10 professional network that either study
11 Parkinson's disease or Parkinson's neurotoxicity
12 that might be -- you know, might be considered
13 neurotoxicologists. I can list some names if
14 you'd like me to.

15 MR. TELAN:

16 Q Is Dr. Cannon one as well?

17 MS. PLATT:

18 Objection.

19 A Dr. Jason cannon would be on that list.

20 MR. TELAN:

21 Q Okay.

22 A Most of the people who I've published
23 with, if they're within the basic sciences, would
24 be on that list. I also publish with people who
25 are not in basic science or who are clinicians.

1 Q You were asked several questions about
2 your correspondence with Dr. Cannon and
3 Dr. Miller. Are -- and I think others, like
4 Dr. Greenamyre. Are these people that you
5 regularly speak to about Parkinson's disease and
6 causality outside of the context of -- of this
7 litigation?

8 MS. PLATT:

9 Objection.

10 A I mean, we talk more specifically
11 probably about, like, a paper or an experiment or
12 piece of data.

13 In the case of -- of Dr. Miller, we --
14 I'm on the committee of one of his graduate
15 students, and, so, we would talk more
16 specifically about that project and its
17 relationship to neurotoxicity, dopaminergic
18 neurotoxicity.

19 And, you know, so -- so sort of yes,
20 but more specifically, you know, when we're
21 talking about something, we talk about -- you
22 know, we sometimes share -- share interesting
23 papers or talk about pieces of data that we've
24 collected or are wanting advice on. So that's
25 one of the things we might talk about at

1 university.

2 MR. TELAN:

3 Q And I know in your report you -- you
4 speak to this, but in a -- in a very -- as lay
5 language as you possibly can, can you give us an
6 overview as to how TCE causes the hallmarks of
7 Parkinson's disease, as you've outlined in your
8 report?

9 A Yeah. So I guess to give an overview,
10 the way that I structured the report is that I
11 explain the Parkinson's hallmark pathology as the
12 loss of dopaminergic neurons and their terminal
13 projections from the substantia nigra to the
14 striatum, the accumulation of alpha-synuclein,
15 and neuroinflammation.

16 I then go into more specific details
17 about the mechanisms of how TCE contributes to
18 this, including mitochondrial dysfunction,
19 mitochondrial damage, oxidative damage and the
20 oxidative stresses involved in that, as well as
21 endolysosomal dysfunction, which is related to
22 what we talked to earlier with the LRRK2 kinase
23 activity, so endolysosomal trafficking,
24 endolysosomal dysfunction. And that's related
25 to -- can be related to the accumulation of

1 alpha-synuclein.

2 And, then, also, as we've previously
3 talked about, there are molecular mechanisms that
4 can be driving neuroinflammation; predominately
5 the activation of brain-resident immune cells,
6 microglia.

7 And, so, what we didn't talk about yet
8 is that there's also a correlation between the
9 adaptive immune system and TCE that's been
10 published previously. And, so, I -- I wrote my
11 report that, you know, you have to also consider
12 both the innate, which is microglia, innate
13 immune system, the adaptive immune system, which
14 involves things like T cells, and that can
15 contribute to the inflammation.

16 So those are sort of the molecular
17 mechanisms. And we have ongoing studies in the
18 lab right now to continue understanding that.

19 Q Okay. I'd like to talk to you about
20 several exhibits that Miss Platt went over with
21 you. And we'll start with the Goldman study of
22 2012, which is Exhibit 5.

23 And I'm not sure that there was this
24 suggestion made, but in terms of how the
25 cohort -- or how Dr. Goldman identified

1 Parkinson's disease within the cohort, if you
2 would go to page 777 in the top left paragraph
3 under "Subjects and Methods Case Ascertainments"
4 --

5 Do you see that paragraph?

6 A Yes.

7 Q I'm not sure that Miss Platt inferred
8 this, but when you look at this paragraph, does
9 this suggest that Dr. Goldman actually utilized a
10 medical records review process to identify those
11 twins with diagnosed Parkinson's disease and
12 suspected Parkinson's?

13 MS. PLATT:

14 Objection.

15 A So the question is does it -- does this
16 paragraph refer to the process?

17 MR. TELAN:

18 Q Yes.

19 A Um --

20 Q And I'm referring specifically to --
21 starting with the word "cases," about midway
22 through that first paragraph.

23 A Okay. Yes. So, in this paper, the
24 authors are outlining how cases from the twin
25 registry were collected, and it's talking about

1 the -- the medical databases of the Department of
2 Veterans Affairs, the Healthcare Financing
3 Administration and the National Death Index.

4 So then it says "twins with suspected
5 Parkinsonism and their brothers received an
6 in-person evaluation by a movement disorder
7 specialist that included a neurological exam and
8 scripted videotaping of the exam."

9 And, so, then it goes on to talk about
10 more medical -- medical records and then
11 diagnosis that's confirmed by two neurologists.

12 Q Does -- does that selection or
13 identification of the diagnosis reflect a robust
14 methodology in identifying the disease?

15 A Well, so, as you might remember, I am
16 not an epidemiologist.

17 Q True.

18 A But my understanding is that in -- in a
19 case where -- particularly when the -- when
20 number of participants is -- is lower, having a
21 movement disorder physician or a neurologist, I
22 guess, who goes in and actually confirms that a
23 person has Parkinson's disease is much stronger
24 than if someone just self-reports that they have
25 Parkinson's or especially if it comes up as just

1 like a -- like a code somewhere or if someone --
2 we talked about someone died with Parkinson's.
3 And, so, it's a stronger way to confirm that the
4 Parkinson's is real.

5 Q So to the extent that there was some
6 reliance on histories by proxies, certainly at
7 least as far as the identification of a diagnosis
8 is concerned, does this methodology reflect that
9 Dr. Goldman enlisted the assistance of -- of a
10 neurologist to make that confirmatory diagnosis?

11 MS. PLATT:

12 Objection.

13 A Yes, it looks like that was the case as
14 it was written here in this -- in this paper.

15 MR. TELAN:

16 Q Is Dr. Goldman a neurologist himself,
17 do you know?

18 A Yes.

19 Q And was that study --
20 Actually, let's go to the end of that.
21 I'm gonna ask you real quickly.

22 If you go to table 3 that Miss Platt
23 was asking you about, she spent a bit of time
24 talking about confidence intervals. I'd like for
25 you to look at the association, the level of

1 association. Do you see that 6.1?

2 A Yes.

3 Q Does that suggest to you that, at least
4 in this study, the -- the exposure to TCE
5 reflected a 610 percent increase in the risk of
6 Parkinson's disease?

7 A I think that is traditionally how you
8 can translate the odds ratio to percent risk is
9 you multiply by a hundred. So it will be 610 in
10 this case.

11 Q Okay. In terms of the -- the reliance
12 on the proxies, if you go to page 782 and if you
13 go in the first paragraph, about midway down -- I
14 want to say it's maybe ten lines down -- there's
15 a sentence beginning with "however."

16 A Okay.

17 Q Would you read those two sentences into
18 the record, please.

19 A "However, assigning exposures based on
20 job tasks rather than relying specifically on
21 self-reported chemical exposures should help
22 mitigate lack of knowledge on the part of the
23 proxy. Furthermore, because exposure estimates
24 were lower for proxy-provided histories than for
25 subject-provided histories, bias would be

1 expected to reduce rather than inflate apparent
2 disease association."

3 Q So does that suggest that there were
4 steps taken to mitigate, perhaps, what -- what
5 Miss Platt characterized as a bias from reliance
6 on proxy histories exclusively?

7 MS. PLATT:

8 Objection.

9 A It -- it does look like, at least in
10 the study that they took, they took that
11 limitation into consideration, and they state
12 that the exposure estimates were lower for the
13 proxy. So that would suggest that it would bias
14 it more towards the null.

15 And, so, that limitation might actually
16 make it harder to prove a connection rather than
17 easier, in the way that it's written here.

18 MR. TELAN:

19 Q Okay. And if you go down to the
20 acknowledgment, does it indicate who funded this
21 research?

22 A Yes.

23 Q And who was that?

24 A National Institute of Neurologic
25 Disorders and Stroke.

1 Q Is that a government agency?

2 A Yes. It's the NIH NINDS.

3 Q Would that have been -- would this have
4 been a peer-reviewed study as well?

5 A Yes, this was peer-reviewed.

6 Q Have you seen any criticisms that the
7 government NIH wrote about this study, if at all?

8 MS. PLATT:

9 Objection.

10 A I'm not aware of any. I'm not. I'm
11 not aware of any.

12 MR. TELAN:

13 Q Okay. If you go to the page -- the
14 first page of this study, Exhibit 5, kind of down
15 in the smaller font, do you see in the second
16 sentence it says "received June 23rd, 2011," and
17 "revised form August 18th, 2011; accepted for
18 publication September 2nd, 2011"?

19 A Yes.

20 Q Does that reflect the peer-review
21 process for this particular article?

22 MS. PLATT:

23 Objection.

24 A It very likely --

25 Or, typically, when you put that on a

1 paper and this is the journal article, so the
2 American Neurological Association would have been
3 the one to actually put that on the article
4 showing that that's when they submitted it,
5 that's when it was -- the peer review concluded,
6 and that's when it was actually accepted.

7 MR. TELAN:

8 Q You had made a statement that because
9 this study involved PD-discordant twins, that
10 there was more power to the study or something to
11 that effect. Did I get that right?

12 MS. PLATT:

13 Objection.

14 A Um, I --

15 So typically when we talk about a
16 disease that we're trying to --

17 We talked about it in the context of it
18 not being genetic. And, so, there's a lot of
19 power in the twin study because the two
20 individuals share genetics, and where disease
21 risk comes from is thought to largely be
22 exogenous in that case. And so a lot of power in
23 that study comes from the fact that they're
24 twins.

25 MR. TELAN:

1 Q Okay. In the -- the universe of -- of
2 literature out there on genetics and Parkinson's
3 disease, is there a study that is stronger than
4 this study on genetics and Parkinson's disease?

5 MS. PLATT:

6 Objection.

7 A Well, that's sort of a -- that --
8 that's a difficult question to ask.

9 So twin studies are very different than
10 other genetic studies that are conducted. So
11 we -- this is not a GWAS study. And there are a
12 plethora of GWAS studies on genetics and
13 Parkinson's.

14 MR. TELAN:

15 Q Okay.

16 A So, you know, genetic sequencing has
17 grown by leaps and bounds, and we now sequence
18 people regularly. And so we have a lot of people
19 on GWAS. We have a lot of data on coding and
20 noncoding variants and things like that.

21 So I don't know that I could
22 necessarily relate a twin study to other types of
23 genetic studies that are around just because
24 they're --

25 You know, that might sound nuanced to

1 you, but for the people who study that, they
2 might say "I do something completely different
3 because I do GWAS," you know. So a twin study is
4 a completely different study.

5 Q Okay. I know Miss Platt spent a bit of
6 time talking with you about a number of different
7 studies that you cited in your report and some of
8 the limitations of your studies. Do you recall
9 that generally?

10 MS. PLATT:

11 Objection.

12 A Yes.

13 MR. TELAN:

14 Q Do all epidemiological studies have
15 limitations?

16 A Yes.

17 Q The fact that a study has limitations,
18 does that result in -- in the study being
19 discarded or deemed to be irrelevant, basically,
20 or merely because of the fact it has a
21 limitation?

22 MS. PLATT:

23 Objection.

24 A So because all of these studies were
25 peer-reviewed and published, the limitations that

1 are -- are noted have been deemed to be, you
2 know, in -- at least in terms of the metrics of
3 publication of these peer-reviewed journals, that
4 it's deemed to be sufficient data, regardless of
5 those limitations.

6 You know, I -- I, again, as a person
7 who's not an epidemiologist, I can't comment
8 specifically on each one of those, only what I
9 sort of absorbed through being, you know, around
10 this field for -- for a number of years. But,
11 you know, it's --

12 If a paper is acknowledging their
13 limitations, which most of these do, and talking
14 about how they tried to overcome them or they
15 used, you know, modeling to overcome them,
16 then -- or they explain that modeling, then
17 that's one way that those limitations are put
18 into perspective.

19 But I would assume that, given the fact
20 that these were peer-reviewed and published in
21 reputable, you know, neurologic journals --

22 Many of these are not toxicology
23 journals. They're -- they're neurology journals.

24 -- that it would be deemed to be a
25 legitimate epidemiologic study.

1 Q Okay. I'd like to go to Exhibit 7,
2 which is the 2023 Goldman study. To the extent
3 that there were individuals who had Parkinson's
4 disease in the Camp Lejeune cohort that weren't
5 picked up because they were treated outside of
6 the -- the VA system or Medicare, would that
7 limitation potentially bias the result toward the
8 null?

9 MS. PLATT:

10 Objection.

11 A So -- so, presumably, if -- if you had
12 a Parkinson's case that was not filed in the
13 system but it was a person who was a veteran and
14 they could have been included in this, then I --
15 I would -- I think it would probably be harder to
16 prove that relationship from exposure to
17 Parkinson's disease because you're missing a case
18 that could have been connected. And that's --
19 tends to be, like, when you look at these tables,
20 that tends to be one of the problems with, you
21 know, studies like this, which is where you're --
22 you know, you wanted to make sure your n is high
23 enough to have proper power. And, so, if
24 anything is reducing that n, presumably it would
25 make it harder -- I think it could make it harder

1 to make that association.

2 MR. TELAN:

3 Q Biassing it toward the null, then.

4 MS. PLATT:

5 Objection.

6 A I think that that -- under those --
7 yeah. Under that terminology, that would be --
8 making it harder to prove the association would
9 be towards the null hypothesis, yes.

10 MR. TELAN:

11 Q So if you wouldn't mind going to page
12 677.

13 A Uh-huh.

14 Q You -- you spoke about the p-value of a
15 .001 in the first column or first row of table 3.
16 Do you recall the discussion with Miss Platt
17 about that?

18 A Yes.

19 MS. PLATT:

20 Objection.

21 MR. TELAN:

22 Q Let me ask you first about the strength
23 of association. You see, before the confidence
24 interval, it has a 1.70?

25 A Yes.

1 Q Does that indicate that, based upon
2 Dr. Goldman's analysis of the cohorts, those that
3 were at Camp Lejeune were found to have a 70
4 percent higher risk of Parkinson's disease than
5 in the Camp Pendleton cohort?

6 MS. PLATT:

7 Objection.

8 A Yes. So based on what we discussed
9 earlier, the way that we can translate odds ratio
10 to percent -- increased percent risk would be to
11 multiply it over -- over 10. So 1.7 would be 70.

12 MR. TELAN:

13 Q And the p-value of .001, what does that
14 tell you in terms of confidence in the -- the
15 association?

16 A So, typically, the lower the p-value,
17 the more statistical significance you have. So a
18 p-value of less than 0.001 would be, you know,
19 considered a -- a very strong p-value.

20 Q Okay. And if you go to page 679, under
21 the Strengths and Limitations section, I want to
22 read a few sentences in to you. I'll read them
23 one at a time just so that you -- I can ask you a
24 question about each.

25 Under that subheading, does it say "our

1 study has -- our study has many strengths. The
2 cohort -- cohorts were population-based,
3 including service members who resided at
4 Camp Lejeune or Camp Pendleton during a 10-year
5 period. We did not rely upon potentially biased
6 self-report to determine exposure. Rather, we
7 inferred exposure based on camp."

8 Did I read that correctly?

9 A Yes.

10 Q "We validated PD diagnosis by review of
11 medical record notes and applied science" --
12 sorry -- "applied accepted diagnostic criteria."

13 Did I read that correct?

14 A Yes.

15 Q If you go a couple sentences down, it
16 states "we explored potential confounding by a
17 range of variables and performed sensitivity
18 analyses that found consistent associations of
19 both PD and prodromal PD with residence at
20 Camp Lejeune."

21 Did I read that correct?

22 A Yes.

23 Q If you go down a few more sentences, it
24 says "we addressed this potential bias in
25 multiple ways. We performed analyses that

1 excluded PD ascertained after the 2017 federal
2 announcement of the diagnostic presumption or
3 that considered only cases that occurred in
4 veterans who were already receiving VA healthcare
5 at the same time of their diagnosis."

6 Did I read that correctly as well?

7 A Yes.

8 Q Finally, "importantly, we assessed risk
9 of PD prodromal features both individually and in
10 two cumulative risk models."

11 Did I read that --

12 A Yes.

13 Q -- correct as well?

14 So while there were a number of
15 limitations that Miss Platt had pointed out to
16 you, there were also a number of strengths that
17 are noted in the study as well?

18 MS. PLATT:

19 Objection.

20 A Yes. They -- they note and list the
21 strengths of the study and compare -- in
22 comparison to other epidemiological studies. And
23 I -- from what you read and, you know, from --
24 from what I understand for how these
25 epidemiological studies are run, they did try to

1 address the limitations. And they said "we
2 addressed this potential bias in multiple ways."
3 So it looks that they addressed the limitations
4 in multiple different ways.

5 Q Okay. Now, this study, was -- this was
6 Dr. Goldman's study. And is he with the -- an
7 academic institution like yourself?

8 A University of California San Francisco.

9 Q Okay. Were the authors from this study
10 also from the University of California
11 San Francisco?

12 A I know that Caroline Tanner is. And if
13 I look at the --

14 Let me look at the affiliations. It
15 does not look like --

16 So looks like there are several
17 affiliations, including Weaver, Stroupe, Cao,
18 Gonzalez and Colletta that are at the Center for
19 Innovation and Complex Chronic Healthcare, The
20 Hines Veterans Affairs Health Hospital in Hines,
21 Illinois.

22 And looks like there's a Loyola
23 University association for Weaver and Stroupe as
24 well.

25 And then -- and then Goldman and Tanner

1 are at UCSF, as well as Ethan Brown.

2 Q Okay. And was this also a
3 peer-reviewed study?

4 A Yes.

5 Q Published in JAMA, Neurology?

6 A Yeah. Yes.

7 Q I think you were asked a number of
8 questions about the Bove and Goldman studies and
9 whether or not they -- they used the same study
10 population. Do you recall those questions
11 generally?

12 MS. PLATT:

13 Objection.

14 A Yes. Excuse me. Yes.

15 MR. TELAN:

16 Q The studies that we're talking about
17 were published at different times; right? Bove
18 was -- there was one study in 2014. You had a
19 20- -- 2018 study and then a Goldman study in
20 2023.

21 MS. PLATT:

22 Objection.

23 MR. TELAN:

24 Q Do you recall that?

25 A Yes. We talked about the 2014 study

1 and I believe a 2024 study and then a 2023 study.

2 Q While the cohort may have been the
3 same, would the populations of the folks who had
4 been diagnosed with Parkinson's disease have
5 changed over that period of time?

6 MS. PLATT:

7 Objection.

8 A If you're asking if, over time, you can
9 have more Parkinson's disease, the answer is yes.

10 MR. TELAN:

11 Q So, in other words, it wouldn't have
12 been the exact same population.

13 MS. PLATT:

14 Objection.

15 A More Parkinson's cases could have --
16 could have popped up in that time. So the
17 population, I guess, could have shifted from
18 cases to controls. But it would be built off of
19 the same --

20 Q Cohort?

21 A -- demographic, same cohort.

22 Q Okay. So Miss Platt asked you a number
23 of questions about other exposures that
24 Dr. Goldman didn't account for in his studies.
25 Do you recall that, generally?

1 MS. PLATT:

2 Objection.

3 A I recall that, yes.

4 MR. TELAN:

5 Q Okay. Is there any reason to believe
6 that these other exposures would
7 disproportionately impact persons in the
8 TCE-exposed versus the unexposed group?

9 MS. PLATT:

10 Objection.

11 A So, you know, I can't necessarily make
12 any implications on what individuals were exposed
13 to outside of what's been reported here in
14 this -- in this paper. I would refer back to
15 that metaphor I used with filling up of the -- of
16 the glass, where if someone sustains a
17 significant exposure that's related to
18 Parkinson's risk, that would be like adding more
19 water to the glass in terms of it has more
20 influence on whether a person will develop
21 Parkinson's or not.

22 So if -- I don't know in this study --

23 There's -- they do have some criteria,
24 like, for example, I believe they account for
25 smoking, which is protective in -- in relation to

1 Parkinson's risk. So there was some element of
2 accounting for other risks. But, yeah, again,
3 kind of referring back to that metaphor of the
4 glass filling up.

5 Q And in the 2023 study, where the design
6 was that he would take Marines or a cohort of
7 Marines at Camp Lejeune and compare them with a
8 cohort of Marines at Camp Pendleton, would that
9 be a reasonable study design in order to offset a
10 confounder, let's say, as head trauma?

11 MS. PLATT:

12 Objection.

13 A Um --

14 MR. TELAN:

15 Q And let me strike that. Let me reask
16 that, in light of the objection.

17 The study design, as you understand it,
18 where you're looking at Marines on different
19 sides of the country, is it -- is it a reasonable
20 study design with the idea that the incidence of
21 traumatic brain injury would be similar at Camp
22 Pendleton as it would be to those at
23 Camp Lejeune?

24 MS. PLATT:

25 Objection.

1 A So the -- the data in the Parkinson's
2 field that relates to the question how you asked
3 it is that veterans or people who serve in the
4 military tend to be -- could tend to be at higher
5 risk for Parkinson's disease at baseline.

6 And, so, using a Camp Pendleton as a
7 control cohort would be stronger than using a
8 civilian cohort for people who have not undergone
9 the same conditions that an individual who has
10 served in the military would.

11 And, so, the idea of using Camp
12 Pendleton as a control in this case would --
13 would be stronger than just taking civilians
14 off -- you know, out of, say, a Medicare database
15 and comparing them to this -- this group of
16 individuals at Camp Lejeune.

17 Q Okay. And does that same analysis
18 apply to a confounder like genetics?

19 MS. PLATT:

20 Objection.

21 MR. TELAN:

22 Q The expectation would be that genetics
23 would have a similar contribution in the Marines
24 in California versus those in North Carolina?

25 MS. PLATT:

1 Objection.

2 A I can't think of a single reason why
3 there would be genetic diversity -- there
4 wouldn't be the same genetic diversity amongst
5 the two cohorts.

6 MR. TELAN:

7 Q If we go to Exhibit 9, which is the
8 2024 Goldman study, under Subjects and Methods,
9 do you see that this study, which, again, was
10 Dr. Goldman's study, was -- was approved by the
11 IRB at University of California San Francisco?

12 A Yes.

13 Q As well as the VA healthcare system?

14 A Yes.

15 Q And do you see that the funding support
16 on page 1 was through the U.S. Department of
17 Veterans Affairs?

18 MS. PLATT:

19 Objection.

20 MR. TELAN:

21 Q Do you see that, page 1? It says
22 "funding agencies"?

23 A Yes.

24 Q Could you read that in the record,
25 please?

1 A "Funding agencies. This work was
2 supported by the U.S. Department of Veterans
3 Affairs (VA) Clinical Science Research and
4 development Merit Award" --

5 Looks like -- is that I?

6 -- "I01 CX002040-01."

7 Would you like me to keep reading?

8 Q That's okay.

9 So this was a government-funded study?

10 MS. PLATT:

11 Objection.

12 A Yes.

13 MR. TELAN:

14 Q Have you seen any criticisms for --
15 published reports of criticisms from the
16 government, anyone in the government, about this
17 particular study?

18 MS. PLATT:

19 Objection.

20 A I can't recall anything that's been
21 published that specifically criticizes this
22 paper.

23 MR. TELAN:

24 Q Okay. There was a -- some discussion
25 about your -- your work in your published

1 studies. And we'll get into that a little bit.
2 But do you recall that generally, having the
3 conversation with Miss Platt about your research?

4 A Yes.

5 MS. PLATT:

6 Objection.

7 MR. TELAN:

8 Q In terms of your research, is it fair
9 that you have studied the effects of TCE on
10 rodents, both inhalationally, orally, and
11 peritoneally?

12 MS. PLATT:

13 Objection.

14 A I have not personally studied TCE
15 exposure via intraperitoneal injection, which is
16 what I think you're referring to with peritoneal.

17 MR. TELAN:

18 Q Yes.

19 So your studies have focused on the TCE
20 effects on rodents through oral and inhalation
21 routes of exposure.

22 MS. PLATT:

23 Objection.

24 A Yes.

25 MR. TELAN:

1 Q Have your studies consistently
2 demonstrated a -- that TCE, when administered to
3 rodents either orally or inhalationally, produce
4 a substantial significant decrease in dopamine
5 neurons within the substantia nigra?

6 MS. PLATT:

7 Objection.

8 A Every -- every study that we publish
9 shows, across the different exposure routes that
10 we measured, a statistically significant loss of
11 dopaminergic neurons in the substantia nigra.

12 MR. TELAN:

13 Q Whether it's through oral or
14 inhalation.

15 A Yes.

16 Q And have your studies also demonstrated
17 a consistent impact on the motor function of
18 those same rodents?

19 MS. PLATT:

20 Objection.

21 A We have published in two papers that
22 there were motor deficits observed in --

23 We published a paper in the oral
24 ingestion model showing open field motor
25 deficits, and in the inhalation model we

1 published only CatWalk motor deficits.

2 Q And, if you would, were all those
3 papers that we discussed in terms of your
4 literature, are those all peer-reviewed as well?

5 A Yes.

6 Q Okay. What is the significance of your
7 work when you speak about TCE degeneration of
8 dopamine-producing neurons in the substantia
9 nigra to the level that you've seen them?

10 MS. PLATT:

11 Objection.

12 MR. TELAN:

13 Q What is the significance of them?

14 MS. PLATT:

15 Objection.

16 A When you say "significance," are you
17 referring to a statistical value or are you
18 referring to, like, a -- like more broad --

19 MR. TELAN:

20 Q More broad.

21 A Okay.

22 Um, so I think I mentioned this
23 previously, but, you know, what --

24 When we were first starting to develop
25 this line of research, what we found in terms of

1 how -- how many dopamine neurons were lost in the
2 animals that we exposed to TCE when we didn't
3 think we were going to see a lesion was -- was
4 pretty remarkable. Many times as we've -- we've
5 begun running these studies -- for example, as we
6 began the inhalation line of research -- we sort
7 of tried to -- we tried to do this at doses that
8 are lower than what's been published. We went
9 through that. That's where those -- our
10 published papers are. And, so, we sort of set
11 a priori what our outcome measures are going to
12 be, and we say, you know, we're going to measure
13 dopamine neuron loss, whether it happens or not.
14 We're going to measure alpha-synuclein
15 accumulation, whether it happens or not.

16 So when we get to the study outcome
17 measure and we actually look inside the brain
18 tissue, particularly with inhalation, it's been
19 really remarkable how many neurons are gone in
20 those animal's brains.

21 And, so, you know, we started off
22 thinking, okay, well, maybe we'll just measure
23 precursors to dopaminergic degeneration, like
24 mitochondrial dysfunction. Then we'll say, you
25 know, over time these neurons would die.

1 But what we found was that, you know,
2 at our very first look, those neurons were gone.
3 It was -- it was highly significant. So, you
4 know, 50 percent of neurons with a p-value of
5 .0001.

6 And, so, you know, I think -- so when
7 you're talking about significance, for me, based
8 on what I just explained, the way that I view
9 this is that, you know, if you expose an entire
10 animal organ system systemically to TCE, it
11 causes specifically Parkinson's pathology in the
12 brain. There are so few things on the planet
13 that can do that that, to me, that feels like a,
14 you know, really remarkable effect in terms of --
15 of, you know, this -- this relationship with this
16 chemical to -- to -- specifically to Parkinson's
17 pathology. I highlighted and outlined, I guess,
18 what that pathology is already.

19 Q You did. And in terms of these
20 animals, these are animals that -- do they have
21 any prior evidence of degeneration within their
22 brain, the substantia nigra of their brain,
23 before you begin introducing the TCE to them?

24 A No. Every published study we've -- we
25 have to date is in wild-type animals, meaning

1 that they are not genetically predisposed to
2 Parkinson's disease or any -- or a model of
3 Parkinson's disease in -- in this case. So they
4 don't carry any Parkinson's mutations or harbor
5 any proteins that would cause them to lose
6 dopamine neurons on their own.

7 Q In terms of the human pathology of
8 Parkinson's disease, is there an understanding
9 that as we get older, we lose dopaminergic
10 neurons in our brain as we age?

11 A So the general consensus is that,
12 number 1, aging is a -- is the biggest risk
13 factor of Parkinson's because it's a disease of
14 aging. And, so, there is -- I think there's an
15 anecdote that, you know, sometimes I hear from
16 clinicians. People say if we all live long
17 enough, we'd all get Parkinson's disease. When
18 they say that, they mean like if we live to like
19 150 or 160. And the reason for that is because,
20 you know, we think that dopamine neurons, as I
21 mentioned, I think, several times, are
22 selectively vulnerable to pathologic processes
23 that cause their degeneration.

24 And, so, some of those pathologic
25 processes can stem from exogenous risks, like

1 what we've talked about today, and some of it can
2 be endogenous risks, like dopamine itself being
3 an oxidative -- oxidative compound or the long
4 and heavily branched axons of dopaminergic
5 neurons that make thousands of connections and
6 require a lot of energy to maintain. As we age,
7 our mitochondria stop functioning as -- as
8 correctly.

9 And, so, you know, the idea that we --
10 we sort of lose a small amount of neurons even in
11 a healthy brain over time is -- is generally
12 recognized as -- as a factor about dopamine
13 neurons.

14 Q Has your research established that the
15 substantia nigra of the brain and the dopamine
16 neurons in the substantia nigra are particularly
17 sensitive to TCE?

18 MS. PLATT:

19 Objection.

20 A So we showed in -- particularly in the
21 2021 Neurobiology of Disease paper, we -- we
22 showed several different markers of neurons that
23 did not degenerate in -- particularly in the
24 striatum, which is where we saw that initial and
25 most of your loss of dopamine terminals. And

1 so -- so the --

2 Again, I refer to the term of
3 "selective vulnerability." So dopaminergic
4 neurons in -- in that publication and in this
5 case are selectively vulnerable to TCE exposure.

6 Q So a human who is aging or going
7 through the normal aging process who is exposed
8 to TCE would have, using your -- your glass
9 analogy, would have additional water deposited
10 into the glass by virtue of the exposure to
11 the -- the TCE.

12 MS. PLATT:

13 Objection.

14 MR. TELAN:

15 Q Is that correct?

16 A Yes. And my analogy that I've given --
17 you know, I've said this a few times now -- that
18 TCE adds -- would be like adding a large amount
19 of water to the -- to the glass in order to reach
20 that threshold of -- of Parkinson's disease.

21 MR. TELAN:

22 Q I know you've talked a bit about LRRK2,
23 and you've kind of described that as a
24 complicated subject to -- to speak about in terms
25 of its impact on Parkinson's disease.

1 When you read Dr. Goodman's report, did
2 she demonstrate a good handle on -- on LRRK2 --

3 MS. PLATT:

4 Objection.

5 MR. TELAN:

6 Q -- as you recall?

7 A Um, what I recall -- what I recall, I
8 don't think that Dr. Goodman had --

9 I don't think she wrote --

10 I think she had a couple of mistakes
11 about -- about LRRK2. One is that the -- the
12 contribution of LRRK2, particularly in a case
13 of -- of my work and particularly in a case of
14 how LRRK2 interacts as a protein in a cell is
15 through its kinase activity as a protein. And I
16 believe there was a statement about it being
17 involved as the LRRK2 gene.

18 And, so, I think, you know, not
19 understanding the difference of a contribution
20 of -- of a -- of a genetic effect versus a -- you
21 know, at the protein level is -- is --

22 It would indicate that there's a lack
23 of understanding of that particular biology.

24 I'm trying to think back more
25 specifically, and I can't recall what exactly was

1 stated about LRRK2 other than, you know, I
2 think -- I think --

3 Again, I mentioned this earlier. You
4 know, I'm -- sometimes I guess I'm a little
5 surprised that there -- that, you know, that
6 Dr. Goodman didn't focus more on these, you know,
7 pathological effects beyond dopaminergic
8 neurodegeneration in terms of how TCE is
9 recapitulating certain types of Parkinson's
10 pathology in the brain, LRRK2 kinase activity
11 being one of them, and also its -- its deep
12 association with dysfunction in the endolysosomal
13 system and how that relates to alpha-synuclein
14 accumulation and -- you know, as a
15 synucleinopathy. I would think that, you know,
16 that should be something that, you know, you'd
17 want to understand and -- and think about when
18 you're talking about the totality of how, you
19 know, a disease mechanism is functioning.

20 Q Let me go, if I can, to Exhibit 11,
21 which is the paper that you published of 2023
22 with Dr. Adamson.

23 First of all, if you go to page 226,
24 who funded this research?

25 A This was funded by -- let me see if I

1 wrote. Yeah. So this was funded by my R00,
2 which is listed there, R00ES02996. That's --
3 that R00 grant was given to me by the National
4 Institute of Environmental Health Sciences.

5 Q So this was a government-funded study?

6 MS. PLATT:

7 Objection.

8 A Yes.

9 MR. TELAN:

10 Q And it was also peer-reviewed as well?

11 A Yes.

12 Q Did you -- have you ever read any
13 critical reviews of this particular study from
14 anyone within the government or outside the
15 government?

16 MS. PLATT:

17 Objection.

18 A I have not.

19 MR. TELAN:

20 Q When a paper like this is
21 peer-reviewed, do the reviewers also review the
22 methodology?

23 A They review the entire paper.

24 Q Including the methodology?

25 A Including methodology.

1 Q In terms of some of the epidemiological
2 literature on inhalation and occupational
3 studies -- and this is kind of a silly question,
4 but it's not ethical to expose humans
5 intentionally to TCE to determine what effects,
6 if any, it may have on their brains. Is that
7 correct?

8 MS. PLATT:

9 Objection.

10 A It would be considered unethical by
11 today's standards to expose humans to TCE.

12 MR. TELAN:

13 Q So other than the Goldman studies, that
14 to the extent that there are studies out there
15 like the Sallmén study, which I know was
16 referenced in the discussion, which, by the
17 way --

18 I know that was marked as an exhibit,
19 so let me refer to that. Or at least your -- I
20 think you referred to it. Might be Exhibit 2.
21 Let me see if I can find it.

22 While I'm looking for it, do you
23 recall --

24 That is Exhibit 4. You are familiar
25 with the Sallmén study in Exhibit 4?

1 A Yes.

2 Q And that was out of Finland, I believe?

3 MS. PLATT:

4 Objection.

5 MR. TELAN:

6 Q Was that study out of workers
7 exclusively located in Finland? Is that right?

8 MS. PLATT:

9 Objection.

10 A Yes.

11 MR. TELAN:

12 Q Were there limitations and weaknesses
13 of that study or anything that you can recall
14 that was unusual about that study, as you sit
15 here today?

16 MS. PLATT:

17 Objection.

18 MR. TELAN:

19 Q Let me reask it, in light of the
20 objection.

21 Do you recall any -- any limitations or
22 weaknesses of that study?

23 MS. PLATT:

24 Objection.

25 A So I -- when I read that study --

1 Again, I am not an epidemiologist.

2 MR. TELAN:

3 Q Understood.

4 A So when I read that study and -- and
5 read about the FINJEM cohort, the way that I've
6 been -- the way that I understand occupational
7 exposure of cohorts is that particularly when you
8 talk about occupation being the way that you
9 categorize exposure, if you just list, like, an
10 occupation, it's very difficult to understand how
11 the occupation is related to an exposure.

12 So in this case, in this study in
13 Finland, they used census data, and some of this
14 census data was missing from some of the decades
15 that was within that study period. And they took
16 a census data. Then they used some mathematical
17 modeling that they put in the supplementary
18 material, which is listed here as 124, and that
19 mathematical modeling was supposed to relate a
20 hundred parts per million dose of -- of -- of a
21 particular solvent to the occupation that that
22 person was in or that a person was in.

23 And where some of the limitations that,
24 you know, even I, as a nonepidemiologist, found
25 out was that it's very difficult to categorize

1 Parkinson's disease association with a -- with an
2 exposure based solely on census data of
3 occupation. The reason for this is because a
4 person who has an occupation as reported by a
5 census could be doing anything in that
6 occupation.

7 So if they worked at a specific
8 factory, we don't know if they were a manager or
9 the foreman, or we don't know if they were the
10 person actually doing something in the factory
11 that could have sustained an exposure. And, so,
12 that creates some concern.

13 In contrast, I have been made aware
14 that there are methods that people go in and they
15 do these surveys. They're actually, I think,
16 quite sophisticated now where people go in and
17 they'll -- they'll ask, like, you know, what are
18 the tasks that you did in a day? And they'll go
19 through really, like, clearly, and they'll even
20 ask, like, how many steps away were you from a
21 particular area? And that helps to generate more
22 granular data on what a person might be exposed
23 to in -- in an occupation.

24 And, so, that wasn't done in this
25 study, and so -- from the way I understand, the

1 way I interpret this is that, you know, going
2 through and taking census data and coming up with
3 the mathematical modeling of some, you know,
4 value of -- of solvent exposure to me doesn't --
5 it just felt very unclear how those numbers were
6 generated, and it didn't seem like it was a very
7 robust way in order to correlate that exposure to
8 a disease risk. So that's the way that I
9 interpret that -- that study.

10 Q Now, you were asked some questions
11 about your work in one in particular regarding
12 PCE and its impact on the brain. Could you tell
13 us generally again? That was a single published
14 study; right?

15 MS. PLATT:

16 Objection.

17 A Yes.

18 MR. TELAN:

19 Q Is that correct?

20 A Yes.

21 Q Explain to us again, generally
22 speaking, what your -- your research found with
23 regard to PCE and dopaminergic degeneration.

24 A So the one experiment we have with PCE
25 is published in the 2024 Ilieva et al. paper in

1 Biology of Disease -- it's in, I believe, figure
2 1 -- showing that PCE at the same concentration
3 as TCE causes the -- a similar statistically
4 significant amount of reactive oxygen species
5 that were measured in -- I think it was a cell
6 line that we did a test called dihydroethidium,
7 which binds to reactive oxygen species and a cell
8 fluoresces. That's how we measure. The
9 fluorescence intensity tells us how much reactive
10 oxygen is in that cell.

11 And, so, for the -- we use the same
12 dose and the same amount of time that we treated
13 cells with TCE. We use that same dose for PCE
14 and measured it the same way. And statistically,
15 those two increased the reactive oxygen species
16 over the control.

17 That same figure also shows that we --
18 that it was exacerbated with a LRRK2 kinase
19 mutation and then that treating with a LRRK2
20 kinase inhibitor or by knocking out LRRK2 in
21 those cells reduced that oxidative stress. And
22 so it would suggest that similar toxic mechanisms
23 along that pathway are mediating that oxidative
24 stress response.

25 Q You were asked by Miss Platt about

1 related chemicals generally. Do you recall that?
2 She gave you an example of water and hydrogen
3 peroxide, carbon monoxide and carbon dioxide. Do
4 you recall that?

5 MS. PLATT:

6 Objection.

7 A Yes.

8 MR. TELAN:

9 Q Do you have an understanding that PERC,
10 or PCE, can degrade into trichloroethylene;
11 correct?

12 A Yes. There are metabolic schemes
13 showing that it can be metabolized in the TCE
14 pathway.

15 Q Does carbon dioxide degrade into carbon
16 monoxide?

17 MS. PLATT:

18 Objection.

19 A I don't believe so.

20 MR. TELAN:

21 Q Does water degrade into hydrogen
22 peroxide?

23 MS. PLATT:

24 Objection.

25 A I don't believe it degrades into

1 hydrogen peroxide. It can get involved in
2 electron transfer where water becomes hydrogen
3 peroxide. But that's, again, not part of its
4 regular detoxification pathway.

5 MR. TELAN:

6 Q Do TCE and PCE share common
7 metabolites?

8 MS. PLATT:

9 Objection.

10 A Yes. There are metabolic schemes that
11 show that TCE and PCE can be metabolized through
12 similar -- similar metabolites and similar
13 metabolic pathways.

14 MR. TELAN:

15 Q Oh, yeah. So if you go to Exhibit 6,
16 this is 2017 ATSDR Health -- Public Health
17 Assessment.

18 Do you have that in front of you?

19 A Yes.

20 MS. PLATT:

21 Can I just clarify that this is the
22 assessment of the evidence, not the --

23 MR. TELAN:

24 Okay. I'm sorry. I stand corrected.

25 Q Exhibit 6 is the 2017 ATSDR Assessment

1 of Evidence. Do you have that in front of you
2 there?

3 A Yes.

4 Q If you would go to page 99 of that,
5 under the conclusion.

6 A Okay.

7 Q And this was in 2017, obviously, 8
8 years ago.

9 A Yes.

10 Q And, again, this probably goes without
11 saying, but has science evolved even over the
12 last 8 years in terms of TCE and Parkinson's
13 disease?

14 MS. PLATT:

15 Objection.

16 A Objectively, yes. We have more
17 information now.

18 MR. TELAN:

19 Q But as of 2017, if you look at the last
20 sentence of the conclusion paragraph of this
21 paper, does it state "however, given the strong
22 supporting mechanistic evidence for TCE, ATSDR
23 concludes that there's equipoise and above
24 evidence for causation for TCE and Parkinson's
25 disease?

1 A Yes.

2 Q And in terms of your opinions as you
3 sit here today, when you've offered the opinions
4 that you did in your report regarding TCE and
5 Parkinson's, is -- is your opinion that, to a
6 reasonable degree of medical certainty, that TCE
7 is more likely than not a cause of Parkinson's
8 disease?

9 MS. PLATT:

10 Objection.

11 A So I would amend that a little bit, and
12 I would say to a reasonable degree of scientific
13 certainty. Because I'm basing this off of
14 scientific evidence. And I'm not a clinician. I
15 don't look at medical evidence. But --

16 MR. TELAN:

17 Q My apologies. Let me rephrase that
18 question.

19 Is it your opinion, to a reasonable
20 degree of scientific certainty, that TCE is a
21 known cause of Parkinson's disease, based upon
22 the universe of research that exists
23 epidemiologically, mechanistically, and within
24 the animal science realm?

25 MS. PLATT:

1 Objection.

2 A That is the conclusion of my research
3 is that there is an association between TCE and
4 Parkinson's disease.

5 MR. TELAN:

6 Q That is causal, to a reasonable
7 degree -- sorry -- scientific certainty?

8 MS. PLATT:

9 Objection.

10 A Under the -- under the -- the legal
11 terminology of more likely than not, which I
12 talked about, you know, I define as being a lower
13 standard than having to apply statistical
14 significance. So saying that, you know, you're
15 tipping the balance even just, you know, one
16 point. So that could be 51 to 49. That's the
17 way that I -- I define those terms, and that's
18 what my conclusions were.

19 Q Okay. I think -- I think Miss Platt
20 had asked you earlier in the deposition if you'd
21 received any correspondence outside of the
22 specific people enumerated in Exhibit 1. Have
23 you received any emails or have a folder of
24 emails from persons that may have called you over
25 the years about Camp Lejeune?

1 MS. PLATT:

2 Objection.

3 A Um, I have a couple of -- I'm sure
4 there are probably emails in my inbox of people
5 who have -- who I've talked to about TCE,
6 Parkinson's disease, and Camp Lejeune that are
7 outside of the case in terms of -- of things I've
8 discussed with people or people have contacted
9 me.

10 MR. TELAN:

11 Q Okay. But, as you sit here today, do
12 you know if any of the people who contacted you
13 are actual claimants in the Camp Lejeune Justice
14 Act litigation?

15 MS. PLATT:

16 Objection.

17 A I have not been made aware of any
18 claimants that are involved in the Camp Lejeune
19 Justice Act. I, to my knowledge, don't know of a
20 single name of who those people are. So I
21 wouldn't be able to cross-reference that with
22 anybody who's contacted me over the years.

23 Q Okay. Those are all the questions that
24 I have for you. Thank you for your time.

25 MS. PLATT:

1 I have no further questions. I would
2 ask that we keep the deposition open, in light of
3 the productions that were requested today.

4 And, also, we are ordering a rough
5 draft.

6 MR. TELAN:

7 And just so that the record is clear, I
8 don't want my silence to be construed as an
9 agreement to keep the deposition open. That's
10 something that we can chat about later.

11 MR. MICELI:

12 We want a rough draft as well.

13 MR. TELAN:

14 Okay.

15 VIDEOGRAPHER:

16 Going off the record. The time is 5:12
17 p.m.

18 (Deposition concluded at 5:12 p.m.)
19
20
21
22
23
24
25

C E R T I F I C A T E

I do hereby certify that the above and foregoing transcript of proceedings in the matter aforementioned was taken down by me in machine shorthand, and the questions and answers thereto were reduced to writing under my personal supervision, and that the foregoing represents a true and correct transcript of the proceedings given by said witness upon said hearing.

I further certify that I am neither of counsel nor of kin to the parties to the action, nor am I in anywise interested in the result of said cause.



LOIS ANNE ROBINSON, RPR, RMR
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Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY.

THE ABOVE RULES ARE CURRENT AS OF APRIL 1, 2019. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

VERITEXT LEGAL SOLUTIONS

COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

Veritext Legal Solutions represents that the foregoing transcript is a true, correct and complete transcript of the colloquies, questions and answers as submitted by the court reporter. Veritext Legal Solutions further represents that the attached exhibits, if any, are true, correct and complete documents as submitted by the court reporter and/or attorneys in relation to this deposition and that the documents were processed in accordance with our litigation support and production standards.

Veritext Legal Solutions is committed to maintaining the confidentiality of client and witness information, in accordance with the regulations promulgated under the Health Insurance Portability and Accountability Act (HIPAA), as amended with respect to protected health information and the Gramm-Leach-Bliley Act, as amended, with respect to Personally Identifiable Information (PII). Physical transcripts and exhibits are managed under strict facility and personnel access controls. Electronic files of documents are stored in encrypted form and are transmitted in an encrypted

fashion to authenticated parties who are permitted to access the material. Our data is hosted in a Tier 4 SSAE 16 certified facility.

Veritext Legal Solutions complies with all federal and State regulations with respect to the provision of court reporting services, and maintains its neutrality and independence regardless of relationship or the financial outcome of any litigation. Veritext requires adherence to the foregoing professional and ethical standards from all of its subcontractors in their independent contractor agreements.

Inquiries about Veritext Legal Solutions' confidentiality and security policies and practices should be directed to Veritext's Client Services Associates indicated on the cover of this document or at www.veritext.com.