Exhibit 609

1 UNITED STATES DISTRICT COURT
2 FOR THE EASTERN DISTRICT OF NORTH CAROLINA
3 SOUTHERN DISTRICT
4
5 IN RE:
6 CAMP LEJEUNE WATER LITIGATION No. 7:23-CV-00897
7 THIS DOCUMENT RELATES TO:
8 ALL CASES.
____
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11
12 VIDEOTAPED DEPOSITION OF
13 KRISTIN ANDRUSKA, M.D., Ph.D.
14 Tuesday, July 8, 2025
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23
Reported By:
24 KATHLEEN A. MALTBIE, STENOGRAPHIC REPORTER
California CSR 10068, Nevada CCR 995, Texas CSR
25 12212, RPR-RMR-CRR-CCRR-CLR-CRC-RDR

1 VIDEOTAPED DEPOSITION OF
2 KRISTIN ANDRUSKA, M.D., Ph.D.
3 BE IT REMEMBERED that on Tuesday, July 8,
4 2025, commencing at the hour of 9:48 a.m. thereof,
5 before me, Kathleen A. Maltbie,
6 RPR-RMR-CRR-CCRR-CLR-CRC-RDR, a Certified
7 Stenographic Shorthand Reporter, in and for the
8 State of California, Nevada and Texas, personally
9 appeared KRISTIN ANDRUSKA, M.D., Ph.D., a witness in
10 the above-entitled court and cause, who, being by me
11 first duly sworn, was thereupon examined as a
12 witness in said action.
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15 Cory Boyer, Intern
16
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1 INDEX

2 INDEX OF EXAMINATIONS

		PAGE
4	Morning Session	8
	Examination By Mr. Gibbons	10
5	Examination By Mr. Telan	242

6 INDEX OF EXHIBITS

	EXHIBIT	DESCRIPTION	PAGE
8-11	Exhibit 1	Document entitled, "Notice of Videotaped Deposition of Kristin Andruska Pursuant to Subpoena With Requests For Production of Documents"	13
12-15	Exhibit 2	Document entitled, "Retention Agreement, Kristin Andruska, MD, PhD, PC, Diplomate of the American Board of Psychiatry and Neurology"	22
15-19	Exhibit 3	Document entitled, "Expert Report of Kristin Andruska, MD, PhD, Rothchild v. United States, No. 7:23-cv-00858-D-KS, February 6, 2025"	23
19-21	Exhibit 4	Document entitled, "Curriculum Vitae: Kristin Andruska, MD, PhD"	28
22-23	Exhibit 5	Document entitled, "X. Appendix A: Reliance Materials"	93

24

25

INDEX OF EXHIBITS (Continued)

EXHIBIT	DESCRIPTION	PAGE
Exhibit 6	Document entitled, "Amended Materials Considered List For Dr. Kristin Andruska's Report On Plaintiff Diane Rothchild"	93
Exhibit 7	Document entitled, "ATSDR Assessment of the Evidence for the Drinking Water Contaminants at Camp Lejeune and Specific Cancers and Other Diseases, January 13, 2017"	97
Exhibit 8	Document entitled, "Evaluation of mortality among Marines, Navy Personnel, and civilian workers exposed to contaminated drinking water at USMC base Camp Lejeune: a cohort study"	115
Exhibit 9	Document entitled, "Evaluation of mortality among marines and navy personnel exposed to contaminated drinking water at USMC base Camp Lejeune: a retrospective cohort study"	123
Exhibit 10	Document entitled, "Mortality study of civilian employees exposed to contaminated drinking water at USMC Base Camp Lejeune: a retrospective cohort study"	125

INDEX OF EXHIBITS (Continued)

EXHIBIT	DESCRIPTION	PAGE
Exhibit 11	Document entitled, "Solvent Exposures and Parkinson Disease Risk in Twins"	132
Exhibit 12	Document entitled, "Risk of Parkinson Disease Among Service Members at Marine Corps Base Camp Lejeune"	150
Exhibit 13	Document entitled, "Parkinson's Disease Progression and Exposure to Contaminated Water at Camp Lejeune"	157
Exhibit 14	Document entitled, "Trichloroethylene and Parkinson's Disease: Risk Assessment"	161
Exhibit 15	Document entitled, "Comparative Analysis of Metabolism of Trichloroethylene and Tetrachloroethylene Among Mouse Tissues and Strains"	174
Exhibit 16	Document entitled, "Cumulative Exposure Expert Report, Kelly A. Reynolds, MSPH, PhD"	188
Exhibit 17	Document entitled, "Appendix 23, Diane Rothchild (Parkinson's Disease)"	189

1 JULY 8, 2025 9:08 A.M. PACIFIC TIME
2 P R O C E E D I N G S
3
4 MORNING SESSION
5
6 THE VIDEOGRAPHER: Good morning. We are
7 now on the record. My name is Douglas Stock. I am
8 the legal videographer for Golkow, a Veritext
9 division. Today's date is July 8th, 2025, and the
10 time is 9:08 a.m.
11 This video deposition is being held at
12 1333 Old Bayshore Highway, in Burlingame,
13 California, in the matter of Diane L. Rothschild
14 versus United States of America. This case is being
15 heard in the United States District Court, Eastern
16 District of North Carolina. The deponent today is
17 Kristin Andruska, M.D. and Ph.D.
18 And, counsel, if you would please identify
19 yourselves for the record and state whom you
20 represent.
21 MR. TELAN: My name is Pat Telan. I'm
22 here for Miss Rothchild on behalf of the plaintiff's
23 leadership group.
24 MR. MICELI: David Miceli on Zoom, and I'm
25 here for the plaintiff's leadership group as well.

1 MR. DOWLING: Mike Dowling on behalf of
2 Ms. Rothschild, co-lead counsel and plaintiff's
3 leadership group.
4 MR. GIBBONS: Hanley Gibbons for the
5 United States.
6 MS. PLATT: Elizabeth Platt for the United
7 States.
8 MR. GIBBONS: We're also joined by
9 Cindy Hurt of the Department of Justice for the
10 United States, as well as Joey turner for the United
11 States. We're also being observed by Cory Boyer,
12 one of our interns.
13 THE VIDEOGRAPHER: Thank you all, and
14 Madam Court Reporter, if you would please announce
15 yourself, state your CSR number and swear in the
16 witness. And then counsel, you may proceed.
17 THE REPORTER: Good morning. My name is
18 Kathleen Maltbie. I am a certified stenographic
19 court reporter, license number CSR 10068. Licensed
20 in California, Nevada and Texas.
21 Would you raise your right hand, please?
22 KRISTIN ANDRUSKA, M.D., PH.D.,
23 having been duly sworn,
24 was examined and testified as follows:
25 / /

1 EXAMINATION BY MR. GIBBONS
2 BY MR. GIBBONS:
3 Q. Good morning, Dr. Andruska.
4 A. Good morning.
5 Q. Can you please state your full name for
6 the record again?
7 A. Kristin Andruska.
8 Q. Okay. No middle name?
9 A. Marie.
10 Q. Marie. Okay.
11 Can you please state your current business
12 address?
13 A. 2495 Hospital Drive, Suite 660, Mountain
14 View, California 94039.
15 Q. Thank you.
16 As you just heard, my name is
17 Hanley Gibbons. I'm an attorney for the United
18 States Department of Justice, and I represent the
19 United States in the Camp Lejeune Water Litigation
20 in the Eastern District of North Carolina.
21 Have you ever been deposed before?
22 A. Yes.
23 Q. So even though you're probably familiar
24 with the procedures, I'm just going to go through a
25 few ground rules this morning just to make sure

1 we're on the same page and maintain the record.
2 Do you understand that a few moments ago
3 you took an oath to tell the truth?
4 A. Yes.
5 Q. Do you understand this is the same oath
6 you would take in court subject to the same
7 penalties for perjury?
8 A. Yes.
9 Q. So the purpose for our time together is to
10 discover information related to the claims and
11 defenses in this lawsuit. To do that, I'm just
12 going to ask you some questions. All I ask is that
13 you answer them to the best of your ability.
14 Is that fair?
15 A. Yes.
16 Q. During this deposition, the court reporter
17 is going to record and transcribe everything we say
18 while we're on the record. Just to make sure that
19 everything is transcribed properly, I'm going to ask
20 that you always answer my questions clearly,
21 verbally and at a reasonable pace.
22 So, for example, if I ask you a yes-or-no
23 question, please respond with an affirmative "yes"
24 or a "no" rather than an uh-huh or a nod.
25 Is that fair?

1 A. Yes.
2 Q. Thank you.
3 If you didn't hear or understand my
4 question, please tell me and I will clarify it or
5 say it again. If you answer one of my questions,
6 I'm going to assume that you understood it.
7 Is that fair?
8 A. Yes.
9 Q. For clarity, please let me finish my
10 question before beginning your answer. Okay?
11 A. Okay.
12 Q. As I said earlier, before we were on the
13 record, I plan to take breaks about every hour. You
14 can take a break at any time if you wish. However,
15 if I've asked a question, I would request that you
16 please answer the question before we go on break.
17 Is that fair?
18 A. Yes.
19 Q. Okay. And just to be clear, you can
20 correct your testimony at any time while we're here
21 today.
22 Do you understand?
23 A. Yes.
24 Q. Are you currently taking any medications
25 that might affect your memory or testimony?

1 A. No.
2 Q. Are there any medical conditions you have
3 that might affect your memory or ability to testify?
4 A. No.
5 Q. Is there any reason that you wouldn't be
6 able to testify or give your most truthful and
7 accurate answers today?
8 A. No.
9 Q. Okay. During the deposition, your
10 attorney may object, but unless he specifically
11 tells you not to answer, I am going to request that
12 you let him finish his objection and then answer my
13 question.
14 A. Okay.
15 Q. Before we get started, I'm just going to
16 go over a few common abbreviations for the record to
17 make sure that we're on the same page.
18 The agency for toxic substances and
19 disease registry is the ATSDR. Trichloroethylene is
20 TCE. Tetrachloroethylene, also known as
21 perchloroethylene, is PCE or PERC, and Parkinson's
22 disease is PD.
23 (Whereupon, Deposition Exhibit 1 was
24 marked for identification.)
25 / /

1 BY MR. GIBBONS:
2 Q. Dr. Andruska, we're introducing what's
3 going to be Andruska Exhibit 1.
4 Do you recognize this document?
5 A. Yes.
6 Q. What is it?
7 A. The notice of today's deposition.
8 Q. Okay. And you've seen this document
9 before, correct?
10 A. Yes.
11 Q. Okay. Have you read through Attachment A,
12 the requested productions with the subpoena?
13 A. I'm sure I've looked at it. I don't know
14 that I've read every word. Okay. I might have
15 skimmed it. I don't know if I read the whole thing.
16 Q. Okay. Do you know if you have any
17 responsive materials that are appropriate to produce
18 under this request for production?
19 MR. TELAN: Hanley, what I would say
20 before she answers, we've responded to it with
21 responses and objections, and so you all have that.
22 But she doesn't have anything additional to add to
23 what we've already responded.
24 MR. GIBBONS: Okay.
25 / /

1 BY MR. GIBBONS:
2 Q. If you'll turn to page 2 of Attachment A,
3 there is an ongoing list of all -- several
4 individuals in the case.
5 Have you had any communications with
6 either Dr. Samuel Goldman or Dr. Richard Dorsey?
7 A. No.
8 Do you mean -- okay. I also know him as
9 Ray Dorsey, but --
10 Q. Sorry, Ray Dorsi.
11 A. But that may be his full legal.
12 No, I have not had any conversations.
13 Q. Even outside the context of this
14 litigation?
15 A. Ray Dorsey and I see each other at
16 professional conferences. We are friendly, but we
17 have not discussed anything specific to this
18 litigation.
19 Q. Okay. What about Dr. Goldman?
20 A. Same, we might be on an airplane together
21 on the way to a conference and say hello, but have
22 not discussed anything particular to this
23 litigation.
24 Q. Okay. If you had any communications with
25 them at these conferences, what were the general

1 substance to the conversations that you had with
2 them?
3 Are they pertinent to Parkinson's disease
4 in particular?
5 A. For Dr. Goldman, very -- for example, at
6 last year's International Movement Disorders
7 Conference, we were on the same plane together. We
8 said hello in the -- in the line at the terminal.
9 We reminisced that we were both employed at the
10 Parkinson's Institute in Sunnyvale, California.
11 That was it.
12 Q. Okay.
13 A. Dr. Dorsey and I collaborate with a group
14 of other movement disorder specialists on a clinical
15 trial in Parkinson's disease, and we met in that --
16 in that research context.
17 Q. Have you had any communications with
18 anyone other than an attorney to prepare for your
19 deposition today?
20 A. Not to prepare for deposition.
21 You might ask me this later. The only
22 person in this litigation that I've spoken to is
23 Dr. Reynolds. We can talk about that now or later
24 if you like. But that was not related to preparing
25 for this deposition in particular.

1 Q. Okay. And that's the same Dr. Reynolds
2 that authored the general causation report in this
3 litigation, correct?
4 A. Correct.
5 Q. Okay. We'll come back to that.
6 Outside of communications, did you do
7 anything to prepare for today's deposition?
8 A. I read my report, I re-reviewed the
9 literature that I cited to in my report.
10 Q. Okay. Did you meet with attorneys at any
11 point to prepare for your deposition?
12 A. Yes. I met with Mr. Telan for about an
13 hour last week and about two hours yesterday just to
14 review my report.
15 Q. Okay. Did you meet in person or via Zoom
16 or telephone?
17 A. We met in person yesterday and over
18 telephone last week.
19 Q. Okay. Were you provided any documents
20 during those discussions?
21 A. No.
22 Q. Other than the independent medical
23 evaluation, have you had any communications with
24 plaintiffs in the Camp Lejeune Water Litigation?
25 A. No.

1 Q. Okay. Have you had any communications
2 with any of Miss Rothschild's treating physicians?
3 A. No.
4 Q. Do you know Dr. Joel Perlmutter?
5 A. I do.
6 Q. Okay.
7 A. Yes. He was my mentor at Washington
8 University in St. Louis.
9 Q. When you say "mentor," can you describe
10 that relationship a little bit more?
11 A. Sure.
12 He was and still is a well-regarded
13 attending physician in the department of movement
14 disorders and someone who kind of taught and trained
15 me during my neurology residency.
16 Q. Okay. Great.
17 And how long did you work with
18 Dr. Perlmutter when you were at Wash U?
19 A. Residency for neurology is four years. So
20 for at least two or three of those years.
21 Q. Okay. Dr. Perlmutter completed a
22 questionnaire in support of your report; is that
23 correct?
24 A. I'm not sure what you mean.
25 Q. Okay. We'll come back to that when we

1 talk about your report in more detail, then.
2 A. I might add, I'm aware that he and I both
3 contributed to the life care plan and that -- that
4 Dr. Fryar uses a questionnaire for the life care
5 plan. So if that's the reference, then I am aware.
6 Q. So you did not personally send anything
7 from your office to Dr. Perlmutter then, correct?
8 A. Correct.
9 Q. Okay. About going back to your meeting
10 with Mr. Telan, did you take any notes during that
11 meeting?
12 A. No.
13 Q. Okay. Was anybody else present with you?
14 A. Mr. Miceli called for a few minutes during
15 the meeting.
16 Q. Okay. Transitioning a bit, how did you
17 first become aware of the Camp Lejeune Water
18 Litigation?
19 A. I was contacted by a member of the
20 leadership team, perhaps Mrs. LaMacchia, around
21 November 2024 to discuss this case.
22 Q. Okay.
23 A. Or maybe Mr. Miceli. I should give him
24 credit since he's on the meeting with us today.
25 Q. That's fine. Just what you remember.

1 What, if any, information were you
2 provided during that initial contact?
3 A. I don't recall exactly. Likely a brief
4 background of the case and likely a timeline for the
5 milestones in the case and, again, a brief
6 discussion of would I consider the -- would I review
7 the materials in the case and consider
8 participating.
9 Q. Okay. Were you already generally aware of
10 the alleged issues with the Camp Lejeune water by
11 the time you met with Ms. LaMacchia?
12 A. Not in any significant context.
13 Q. Okay.
14 A. Had I heard the -- the term
15 "Camp Lejeune," yes. But I didn't know any
16 specifics of the case.
17 Q. Okay. Generally, you were aware that
18 there was alleged toxic exposure at Camp Lejeune,
19 though?
20 A. Yes.
21 Q. To the best of your ability, when did you
22 first hear about Camp Lejeune in conjunction with
23 toxic exposures?
24 MR. TELAN: Object to the form. The only
25 cautionary instruction I give you is that if you're

1 going to guess, let him know it's a guess. If you
2 remember, then absolutely you can tell him by
3 memory, but we don't want you to speculate or guess
4 if you don't know.
5 MR. GIBBONS: I'm just going to remind you
6 for the record that your objections are limited to
7 form and foundation, so please reframe from making
8 any speaking objections.
9 MR. TELAN: Understood. But I don't
10 think -- I think that's a fair cautionary
11 instruction because I don't think you want her to
12 guess at anything today. And so I wanted to make
13 that known at the outset.
14 MR. GIBBONS: Okay.
15 BY MR. GIBBONS:
16 A. I really couldn't say for sure. Of
17 course, I'm a Parkinson's doctor. There's --
18 there's mention in the lay media of do you have
19 Parkinson's, have you been at Camp Lejeune. There
20 is, you know, of course, some literature about it in
21 professional journals that I read, but I have no
22 specific memory other than perhaps years before
23 2024.
24 Q. Okay. And at some point after that
25 initial meeting with Ms. LaMacchia, you were

1 retained as an expert witness in this matter,
2 correct?
3 A. Yes.
4 Q. When were you formally retained?
5 A. My -- my best recollection is around
6 November 2024.
7 Q. Okay. How many times did you speak with
8 plaintiffs' counsel before being retained?
9 A. I believe it was the one meeting.
10 Q. Okay. Did you execute a retainer
11 agreement?
12 A. Yes.
13 Q. Okay.
14 (Whereupon, Deposition Exhibit 2 was
15 marked for identification.)
16 BY MR. GIBBONS:
17 Q. Dr. Andruska, I'm introducing Exhibit 2.
18 Is this the retainer agreement that you
19 completed?
20 A. This is my standard retainer. I have a
21 copy that's -- that's fully executed. I see this
22 copy only has my signature, but the content is the
23 same.
24 Q. Did you perform any work in connection
25 with this case prior to executing that retainer

1 agreement?
2 A. No.
3 Q. Can you generally describe what you were
4 retained to opine on in this case?
5 A. Generally, to opine on whether toxic
6 exposure to chemicals, TCE and PCE, by
7 Mrs. Rothschild at Camp Lejeune were a factor in
8 causing her Parkinson disease.
9 Q. Okay. A minute ago you just went over
10 this, but you were requested to complete a report
11 specifically for Diane Rothschild in this case,
12 then?
13 A. Correct.
14 Q. Have you ever written any other reports in
15 this litigation?
16 A. No.
17 Q. Okay.
18 (Whereupon, Deposition Exhibit 3 was
19 marked for identification.)
20 BY MR. GIBBONS:
21 Q. Dr. Andruska, I've just handed you what's
22 going to be Exhibit 3.
23 Is this the report that you entered in
24 this litigation for Diane Rothschild?
25 A. It looks like it, yes.

1 Q. Okay. Did anyone else assist you in
2 writing this report?
3 A. No.
4 Q. Did you have any supporting staff?
5 A. No.
6 Q. And on page 64, that's your signature,
7 correct?
8 A. I just looked at it, but I'll turn there
9 again.
10 Yes.
11 Q. Does this report contain all the opinions
12 you've formed in this case?
13 MR. TELAN: Object to the form.
14 THE WITNESS: Yes.
15 BY MR. GIBBONS:
16 Q. For clarity, I will rephrase.
17 Does this report contain all the opinions
18 that you discuss -- that you plan to offer in this
19 case?
20 MR. TELAN: Object to form.
21 THE WITNESS: Yes. And if other opinions
22 come up today, I'm sure we'll discuss it.
23 BY MR. GIBBONS:
24 Q. Okay.
25 A. Of course, I also note that if new

1 information comes into the case, I'm happy to review
2 that and -- and adjust my opinions as needed.
3 Q. Okay. Have you discussed your work in
4 this case with anyone else in your practice?
5 A. No.
6 Q. Have you received any compensation in
7 connection with your work on this case?
8 A. Yes.
9 Q. How much do you charge for your services
10 as an expert?
11 A. 850 per hour. 950 for deposition and
12 testimony.
13 Q. And how much have you billed to date in
14 this case?
15 A. I believe it's approximately 90,000.
16 Q. Okay. Does $97,000 sound about right?
17 A. Yes.
18 Q. Does your payment depend on the outcome of
19 this case?
20 A. No.
21 Q. Are you reimbursed for any expenses or
22 costs?
23 A. Yes.
24 Q. What expenses or costs are you reimbursed
25 for?

1 A. For example, travel, postage.
2 Q. Any others?
3 A. Not that have come up-to-date in this
4 case, but my retainer agreement also mentions food
5 and reasonable expenses.
6 Q. Okay. And you had a flat retainer
7 agreement or a flat fee attached to your retainer
8 agreement, correct?
9 A. Correct.
10 Q. That was $10,000, correct?
11 A. Correct.
12 Q. Okay. What percentage of your annual
13 income is earned from serving as an expert witness?
14 A. That's hard to say. I would estimate my
15 work as an expert is 5 -- approximately 5 percent of
16 my time. But the amount of work I do is not
17 constant, varies from year to year, and, you know, I
18 only take projects as time allows. So there's not a
19 consistent amount or percentage.
20 Q. Okay.
21 A. It is a -- it's a small fraction; leave it
22 at that.
23 Q. Okay. If you had received all $97,000 for
24 your work in this case this year, roughly what
25 percentage of your income would that constitute?

1 A. Between 10 and 15 to 20 percent.
2 Q. Okay. Are you currently serving as an
3 expert witness in any other cases?
4 A. I have been retained in other cases, yes.
5 Q. Okay. Is your fee schedule in this case
6 the same as the fee schedules you have in other
7 cases?
8 A. Approximately, yes.
9 Q. How many times have you served as an
10 expert witness before?
11 A. I've been deposed five times. And there
12 have been some other cases over the years where I --
13 where I have only written a report.
14 Q. How many reports have you written in
15 connection with litigation?
16 A. I'm just guessing here. Maybe an
17 additional five.
18 Q. Okay. Did any of those have to do with
19 Parkinson's disease?
20 A. Yes.
21 Q. Were those on behalf of plaintiffs,
22 defendants or both?
23 A. So far, these have been for plaintiffs.
24 But I have considered cases for both and happy to
25 work for either side.

1 Q. Okay. Of those five cases, how many of
2 those dealt with Parkinson's?
3 A. The five that went to deposition or trial?
4 I'm sorry, I'm asking.
5 Q. Oh, sorry, the five that went to
6 deposition.
7 A. Three.
8 (Whereupon, Deposition Exhibit 4 was
9 marked for identification.)
10 BY MR. GIBBONS:
11 Q. Dr. Andruska, you were just handed what's
12 going to be marked as Exhibit 3.
13 MR. TELAN: We are on 4, I think.
14 MR. GIBBONS: I'm sorry, correction,
15 Exhibit 4.
16 BY MR. GIBBONS:
17 Q. Is this your CV?
18 A. Yes.
19 Q. And you drafted this document yourself,
20 correct?
21 A. Yes.
22 Q. When did you draft this document?
23 A. This would have been in November 2024.
24 Q. Is this document a complete representation
25 of your educational and employment background?

1 A. It is with the exception that I am now
2 also the chief medical officer of SpinMed, which is
3 listed at the top of my employment.
4 Q. And when did that change occur?
5 A. Around February of 2025.
6 Q. That was after you finalized your report
7 for Ms. Rothschild?
8 A. Correct.
9 Q. Is there any -- is there information that
10 you did not include in your CV?
11 A. No.
12 Q. Okay. And you hold yourself out to be an
13 expert in Parkinson's disease, correct?
14 A. Yes.
15 Q. I know this sounds reductive, but what is
16 the basis of your expertise in Parkinson's?
17 A. My background, my education, my training
18 as a neurologist and movement disorder specialist,
19 as a scientist and my experience.
20 Q. Okay. Are you a geneticist?
21 A. I have a background in genetics. I
22 don't -- I'm not currently employed as a geneticist.
23 Q. Would you be qualified to testify as an
24 expert on the subject of genetics?
25 MR. TELAN: Object to the form.

1 THE WITNESS: I have a Ph.D. in the
2 biochemistry and genetics of movement disorders, so
3 I feel very comfortable and well versed.
4 BY MR. GIBBONS:
5 Q. Okay. You're not an attorney, correct?
6 A. Correct.
7 Q. And you're not an epidemiologist, correct?
8 A. Correct.
9 Q. You don't hold yourself out to be an
10 expert in epidemiology, correct?
11 A. Correct.
12 Q. You don't have any certifications or
13 licenses in epidemiology, right?
14 A. Correct.
15 Q. It's safe to say you don't have a
16 specialty in epidemiology in Parkinson's disease
17 specifically, correct?
18 MR. TELAN: Object to the form.
19 THE WITNESS: Again, as a movement
20 disorders specialist, I am well versed and well
21 aware of the epidemiology of Parkinson's, but I am
22 not an epidemiologist.
23 BY MR. GIBBONS:
24 Q. You've never been the principal
25 investigator for an epidemiological study, correct?

1 A. Correct.
2 Q. You've never published any peer-reviewed
3 literature on epidemiology, right?
4 A. Correct.
5 Q. Have you taught any sources on
6 epidemiology?
7 A. No.
8 Q. Do you have any formal training as an
9 epidemiologist?
10 A. No.
11 Q. Have you ever conducted a literature
12 review of epidemiological studies and publications
13 in part of your clinical practice?
14 A. Yes.
15 Q. What did you do to conduct those searches?
16 Sorry. Reviews.
17 A. If I'm understanding your question
18 correctly, I have searched the parkins [sic]
19 literature. I'm just thinking about how to address
20 your question specifically. And perhaps you can
21 redirect me if I'm -- if I'm off.
22 As a movement disorders and Parkinson's
23 specialist, I keep abreast of the literature, I read
24 the literature when specific questions come up,
25 including in the course of my standard clinical

1 practice. I search the literature. Certainly, many
2 of the articles that come up are epidemiologic
3 studies. I think that's the better answer to your
4 question.
5 Q. Okay. When you said when questions come
6 up, what did you mean?
7 A. Myriad questions. Could be when I'm
8 reading an article that opens the door to another
9 question, either that I personally have or the
10 author's posed, I might dig deeper into the
11 literature, or oftentimes my patients will ask me
12 questions about their disease and about Parkinson's
13 in general, their research in Parkinson's, and we
14 might look at the literature together if it's a --
15 if it's a question that I don't already know the
16 answer to.
17 And -- and I like to stay abreast of just
18 current topics in Parkinson's. So I might search
19 for things that are of interest --
20 Q. Okay.
21 A. -- on my own.
22 Q. What kinds of things are of interest to
23 you in researching Parkinson's disease?
24 A. Everything, at the risk of being too
25 broad.

1 Q. Okay. Do you conduct independent analysis
2 of the studies or publications that you review
3 during this process?
4 A. Can you word that differently?
5 Q. Sure.
6 Let's say that a study that you find in
7 connection with a question that a patient has asked
8 you, do you go through and independently verify the
9 methods used in the studies that you've reviewed?
10 A. Yes. I would look at the quality of the
11 paper, at the authors, at conflicts of interests, at
12 their methods and so on.
13 Q. And what kinds of things would you look
14 for to determine quality of a paper?
15 A. All of the things I just mentioned, the
16 methods, the impact factor of the journal, the
17 reputation of the institution, any conflicts of
18 interest, the -- the strengths and limitations of
19 the study and so forth.
20 Q. Relatedly, have you ever conducted a
21 literature review for epidemiological studies as
22 part of your expert witness practice?
23 A. As part of my expert witness practice, I,
24 of course, mine the literature for relevant
25 articles. Many of those are epidemiologic studies,

1 but I'm not sure that I'm answering your question,
2 meaning --
3 Q. Do you have any different practices that
4 you employ when you're reviewing studies for expert
5 witness work versus in your clinical practice?
6 A. No.
7 Q. You're not a toxicologist, correct?
8 A. Correct.
9 Q. You don't have any certifications in
10 toxicology?
11 A. Correct. I do not.
12 Q. Do you have any training or experience as
13 a toxicologist?
14 A. No.
15 Q. You've never been a principal investigator
16 of a toxicological study then, correct?
17 A. No.
18 Q. You've never published peer-reviewed
19 literature on toxicology?
20 A. No.
21 Q. Have you taught any courses on toxicology?
22 A. No.
23 Q. You're not a risk assessor either,
24 correct?
25 A. A risk assessor.

1 Q. A risk assessor?
2 A. No.
3 Q. Okay. You don't have any certifications
4 in risk assessment?
5 A. No.
6 Q. Similar question.
7 You've never published any peer-reviewed
8 literature on risk assessments?
9 A. No.
10 Q. You've never taught any courses on risk
11 assessment?
12 A. No.
13 Q. Have you taken any college or graduate
14 level courses that give you scientific expertise in
15 risk assessment?
16 A. No.
17 Q. You've never published any peer-reviewed
18 literature regarding the connection between TCE and
19 Parkinson's disease, correct?
20 A. Correct.
21 Q. You've never published any peer-reviewed
22 literature between PCE and Parkinson's, correct?
23 A. Correct.
24 Q. You've never published any peer-reviewed
25 literature between literature on the connection

1 between vinyl chloride and Parkinson's, correct?
2 A. Correct.
3 Q. And you've never published any
4 peer-reviewed literature on the connection between
5 Benzene and Parkinson's disease, correct?
6 A. Correct.
7 Q. In your clinical practice, you've never
8 treated any individuals with Parkinson's that were
9 exposed to water at Camp Lejeune, correct?
10 A. Not to my knowledge. I certainly have
11 veterans in my practice, but we don't usually get
12 into the specifics of where they were stationed.
13 Q. Okay. What kinds of information do you
14 get about their background, then?
15 MR. TELAN: Object to the form.
16 THE WITNESS: I usually, in my social
17 history, ascertain whether they've had military
18 service and which branch. But don't probe into
19 which bases they were stationed at.
20 BY MR. GIBBONS:
21 Q. Okay. Is there a reason that you request
22 what branch of service they were in but not the
23 location?
24 A. I think it's in part to get to know them a
25 little bit better and to understand their background

1 and -- because sometimes it's relevant to exposures
2 or injuries or other, you know, relevant parts of
3 their history. It's the way I was taught to take a
4 social history.
5 Q. Okay. Is there a reason that knowing the
6 branch of service gives you an idea of general
7 exposure that they may have, but that wouldn't
8 necessarily necessitate asking for specific
9 locations?
10 A. For example, in Parkinson's, we know that
11 Agent Orange has a connection to Parkinson's. So
12 people who served in Vietnam in the Army were more
13 likely to know that they were exposed to
14 Agent Orange than someone who wasn't on the ground
15 in Vietnam.
16 Q. Is Ms. Rothschild the first patient that
17 you have examined after knowing that she was at
18 Camp Lejeune?
19 A. Yes.
20 Q. Okay. In your practice, have you ever
21 evaluated the cause of a patient's Parkinson's
22 disease?
23 A. It does come up from time to time,
24 especially -- well, it comes up certainly when
25 patients ask about what might have caused their

1 Parkinson's. And that happens quite a bit.
2 Patients are interested in whether their occupations
3 or their historic exposures may have caused or
4 contributed to their Parkinson's, and we'll have a
5 thoughtful discussion about that.
6 I also, as I mentioned, take a thorough
7 social history in -- you know, in keeping with my --
8 my training, and a good social history also includes
9 exposures and whether we know, for a particular
10 patient in a clinical setting, exactly which
11 exposures they had and can give patients a
12 satisfying answer about what caused their
13 Parkinson's. Of course, it is a different question,
14 but it is something that I typically think about.
15 Q. What kinds of exposures are you
16 specifically concerned with when you are going
17 through a social history of a patient?
18 A. This is not --
19 MR. TELAN: Object to the form.
20 THE WITNESS: And this is not a
21 comprehensive list, but a social history in movement
22 disorders will often include smoking, alcohol, drug
23 use, former occupations, current occupation,
24 military service, pesticide exposure,
25 anti-dopaminergic, medication use, certain types of

1 head injury, things like that.
2 BY MR. GIBBONS:
3 Q. To your knowledge, have any of your
4 clinical patients ever been exposed to TCP?
5 A. That's not something that we discuss in
6 detail, so I wouldn't know.
7 Q. Is that also true of PCE?
8 A. Yes, that's correct.
9 Q. Same is true for vinyl chloride?
10 A. Yes. It's true for vinyl chloride. To go
11 back to your PCE comment, I do have patients who
12 have a history of working in the dry cleaning
13 industry, and that has come up in the conversations
14 that I referenced earlier, could those exposures be
15 relevant to their Parkinson's, and we'll discuss
16 that.
17 Q. How many clinical patients have you seen
18 in your career with Parkinson's disease?
19 A. Oh, thousands.
20 Q. And how many of them have prior experience
21 working at a dry cleaners?
22 MR. TELAN: Object to the form.
23 THE WITNESS: I -- I can say that I have
24 two patients in my current practice with that
25 exposure. I can remember that easily because it's a

1 conversation we had in the last few months. Whether
2 other patients have mentioned that over, you know,
3 the last 15 years, I don't remember.
4 BY MR. GIBBONS:
5 Q. Is there a reason that you remember those
6 two patients in particular?
7 A. I would remember most of the conversations
8 that I had with -- with all my patients in the last
9 few months just because of the short -- of the -- of
10 the closeness of the conversations.
11 Q. Okay. But out of the thousands of
12 patients you've seen, you're only aware of two that
13 have potential exposure from work in dry cleaning?
14 A. Yes. And, of course, we take that
15 thousands in context of having different levels of
16 relationships with all those patients. These could
17 be patients I see in consultation one time. These
18 could be patients I saw a long time ago for -- for
19 shorter or longer periods of time.
20 The patients that I currently take care
21 of, I know very well, I see them often, I spent a
22 lot of time with them. Because they know I'm a
23 scientist, they'll come to me with their concerns
24 and questions about exposures. So those are more --
25 those are deeper relationships and patients that I

1 know on a much more granular level.
2 Q. Okay. To the best or your ability, could
3 you approximate how many of your thousands of
4 patients are one-off consultations versus routine
5 patients that you see clinically?
6 MR. TELAN: Object to the form.
7 THE WITNESS: It's almost impossible to
8 say. In my current practice, I have very few
9 consultations because my goal is to provide
10 longitudinal personal care for all these patients.
11 In former institutions where, you know, I may be
12 covering for colleagues, seeing patients once,
13 patients come in and out more quickly, we may not
14 call it a consultation in that sense, but it would
15 be more common to see a patient just once or twice,
16 for example. Certainly in my training, that those
17 relationships were quite a mix of -- of short and
18 longer-term relationships. So it's just hard to
19 say.
20 BY MR. GIBBONS:
21 Q. Okay. Well, using your distinction,
22 short- and long-term relationships, how many
23 patients do you currently see that you have a
24 long-term relationship with?
25 A. Approximately 50.

1 Q. Okay. In your practice, have you ever
2 determined that the cause of a patient's Parkinson's
3 disease was TCE?
4 A. No. That's not within the standard goal
5 of a -- of a clinical practice.
6 Q. Okay. Same question for PCE.
7 Have you ever determined that the cause of
8 one of your patient's Parkinson's disease was PCE?
9 A. No. And for the same reason.
10 Q. Okay. Do you regularly perform
11 differential diagnosis on your patients?
12 A. Yes, I do.
13 Q. Okay. How do you go about conducting a
14 differential diagnosis for your patients?
15 A. I consider all of the possible diagnoses
16 that are relevant to a particular patient's case. I
17 integrate their history, their exam. And then I
18 determine a priority list of which diagnoses I think
19 are more likely versus less likely given their
20 history, their exam and their clinical context.
21 Q. Okay. Do you specifically rule out risk
22 factors as you select the most likely causes?
23 MR. TELAN: Object to the form.
24 THE WITNESS: I consider their risk
25 factors. I would specify that when we say "rule

1 out," it's hard to say with 100 percent certainty
2 that something is or is not either on the
3 differential or -- or a risk factor. But we do the
4 best we can with the information we have.
5 BY MR. GIBBONS:
6 Q. Other than this case, have you ever
7 offered an expert opinion in a case involving toxic
8 exposures?
9 A. I have, yes.
10 Q. And how many?
11 A. Let me think.
12 About three.
13 Q. Were any of those the Parkinson's cases
14 that you referred to earlier?
15 A. Yes.
16 Q. Okay. How many?
17 A. I have been involved in the -- in a case
18 of toxic exposures. This is one of the depositions
19 that I referenced earlier, and another kind of one
20 report that I referenced earlier.
21 Q. Was that deposition and report in the same
22 case?
23 A. I'm referencing two plaintiffs, but, of
24 course, the plaintiff that went to deposition also
25 had a report, and then another only had a report.

1 Q. Okay. So just to clarify, earlier when
2 you specified the difference between cases in which
3 you have been deposed versus cases in which you had
4 offered a report, the cases in which you were
5 deposed, you had also offered reports?
6 A. Correct.
7 Q. Okay. So you've offered -- you've offered
8 two reports on Parkinson's disease in conjunction
9 with toxic exposure, correct?
10 A. Correct.
11 Q. You've been deposed for one of them?
12 A. Correct.
13 Q. Not including this case?
14 A. Correct.
15 Q. Okay. Were either of those cases the ones
16 that you've disclosed in your report on
17 Ms. Rothschild?
18 A. I'm not following.
19 Q. Let's see.
20 A. Oh.
21 Q. Go to -- I believe it's page 2 in your
22 report.
23 A. Yes. The answer to your question is yes.
24 Q. Okay. And other than this case, have you
25 ever offered an opinion on the etiology of

1 Parkinson's disease?
2 A. Are you asking have I commented on
3 differential etiology in a -- in a report --
4 Q. Correct?
5 A. -- like this?
6 Yes.
7 Q. Okay. How many times?
8 A. I believe one other time.
9 Q. Was that in either of the two Parkinson's
10 cases that we discussed?
11 A. Yes.
12 And now remembering that, the number is
13 three.
14 Q. Three total reports?
15 A. Three reports.
16 Q. Okay. Do you remember the case names that
17 were associated with those three reports?
18 A. Yes. Referencing page 2 in my report,
19 this is Evans, et al. This is multiple plaintiffs
20 versus Syngenta. And in a separate case under the
21 Paraquat products liability litigation.
22 Q. Do you know the specific case that was
23 part of the Paraquat litigation?
24 A. Are you asking for the specific
25 plaintiff's --

1 Q. Correct.
2 A. -- name?
3 I remember one, yes.
4 Q. Okay. What was that name?
5 A. This was -- I forget his first name, but
6 this was a case where the plaintiff was Mr. Krause.
7 Q. Can you spell that?
8 A. K-R-A-U-S-E.
9 Q. Have you ever been subject to any
10 disciplinary action or censure by any licensing
11 body?
12 A. No.
13 Q. Okay. Have you ever been the subject of
14 any disciplinary action by a court or tribunal?
15 A. No.
16 Q. All right. I think we're about an hour,
17 so we can take a break, go off the record.
18 THE VIDEOGRAPHER: Going off the record at
19 10:04 a.m.
20 (Whereupon, a recess was taken from
21 10:04 a.m. to 10:15 a.m.)
22 THE VIDEOGRAPHER: We are back on the
23 record at 10:15 a.m.
24 BY MR. GIBBONS:
25 Q. Dr. Andruska, is there anything from your

1 previous testimony that you'd like to correct before
2 we get started again?
3 A. No, thank you.
4 Q. Okay. In your report for Ms. Rothschild,
5 you applied the as likely as not standard in your
6 report, correct?
7 A. Correct.
8 Q. Have you ever used the as likely as not
9 standard in your medical professional work?
10 A. Yes, I have.
11 Q. When did you use it?
12 A. The -- I'm sorry, did you say at least as
13 likely as not?
14 Q. Correct.
15 A. At least as likely as not is the
16 Department of Veterans Affairs or VA's hospitals'
17 standard for evaluating medical disability. And I
18 have written these nexus letters for my patients in
19 support of disability claims, and this is the
20 standard used.
21 Q. Okay. These nexus letters are more of,
22 like, a legal document for the VA to cover
23 presumptive diseases, correct?
24 MR. TELAN: Object to the form.
25 THE WITNESS: A nexus letter is a

1 document -- is a letter written by a qualified
2 medical professional to discuss a patient's disease
3 and educate the VA about the disease so the VA can
4 use the information in the letter when evaluating
5 these disability claims.
6 BY MR. GIBBONS:
7 Q. Understood.
8 Have you ever used the as likely as not
9 standard in evaluating medical studies?
10 A. Again, do you mean at least as likely as
11 not?
12 Q. Correct. Sorry. At least as likely as
13 not.
14 A. In evaluating medical studies?
15 Q. Have you ever seen a medical study that
16 uses the at least as likely as not standard?
17 A. I've seen studies from ATSDR and others
18 using the term "clinical equipoise," which, to me,
19 means at least as likely as not.
20 Q. Okay.
21 A. Otherwise, it's not a particularly common
22 term outside of the -- the medical examples that I
23 just gave.
24 Q. Okay. Let's talk about the equipoise
25 standard for a second.

1 Is that above, below or the same as the
2 reasonable degree of medical certainty?
3 MR. TELAN: Object to the form of the
4 question.
5 THE WITNESS: I define clinical equipoise
6 as as likely as not, or equally likely. You're
7 asking about to a reasonable degree of medical and
8 scientific certainty.
9 In -- in my view, I am opining on these
10 topics to a reasonable degree of medical and
11 scientific certainty. Meaning, to the best of my
12 ability and to the best that I can say as a
13 physician and scientist based on available data,
14 there is a distinction, however, between to a
15 reasonable degree and clinical equipoise.
16 BY MR. GIBBONS:
17 Q. Okay. I think I understand the
18 distinction that you're making.
19 Have you ever used the at least as likely
20 as not standard in any of your reports for
21 litigation before?
22 A. No.
23 Q. Okay. Where did the at least as likely as
24 not language come from in Ms. Rothschild's report?
25 MR. TELAN: Object to the form.

1 THE WITNESS: I have seen it in the
2 Camp Lejeune Justice Act statute.
3 BY MR. GIBBONS:
4 Q. Okay. Did you find that language on your
5 own, then?
6 A. I believe it was shared in the context of
7 a meeting with counsel.
8 Q. Okay. So the Camp Lejeune Justice Act was
9 provided to you?
10 A. Yes.
11 Q. Okay. Outside of these nexus letters for
12 the VA, have you ever used the at least as likely as
13 not standard in your clinical practice?
14 A. No.
15 Q. Okay. And apart from the ATSDR reports
16 that use clinical equipoise, have you ever seen the
17 at least as likely as not standard used in
18 literature that you reviewed for your report?
19 A. I don't believe so.
20 Q. Okay.
21 A. I might add that, in coming up with a
22 differential diagnosis, of course, I'm considering
23 which diagnoses are likely. And, of course, as we
24 discussed earlier, in making a ranked order list or
25 a high priority list, I'm considering which

1 diagnoses are likely or more likely than others, are
2 at least as likely as not, although I might not use
3 that wording in my day-to-day life.
4 Q. Okay. So when you're making this kind of
5 prioritized or we'll call it, like, hierarchical
6 list of the different causes, the at least as likely
7 as not causes would be ruled in, ruled out? I'm
8 just trying to understand how you would use it in
9 making your list.
10 MR. TELAN: Object to the form.
11 THE WITNESS: If I am considering a
12 differential diagnosis or a differential etiology in
13 my clinical practice, I focus on diagnoses that
14 are -- that are likely. And I wouldn't distinguish
15 between as likely as not or at least as likely as
16 not in my standard clinical practice, but, again,
17 I'm considering diagnoses that are most likely or
18 more likely than others.
19 BY MR. GIBBONS:
20 Q. Okay. So the at least as likely as not
21 causes would be ranked lower than the more likely
22 than not, correct?
23 A. I think you mean the opposite.
24 Q. It would be ranked below -- you would use
25 causes that are at least as likely as not would be

1 ranked lower than the most prevalent causes that you
2 think are more likely the causes, correct?
3 A. Correct.
4 Q. But you wouldn't rule them out completely?
5 A. At least as likely as not is a more
6 relaxed standard, yes, I agree with you.
7 Q. Okay. Earlier you said that you reviewed
8 a copy of the Camp Lejeune Justice Act.
9 Have you ever previously reviewed
10 statutory language in rendering an expert opinion?
11 A. I don't know.
12 Q. Have you ever previously addressed a legal
13 burden of proof in rendering an opinion?
14 A. By "legal burden of proof," do you mean a
15 legal standard, such as more likely than not or a
16 substantial contributing factor?
17 Q. Yes.
18 A. Yes.
19 Q. Okay. Was that in any of the three cases
20 that we've discussed previously?
21 A. Yes.
22 Q. Okay. Which ones?
23 A. Each case had a legal standard, but, of
24 course, sitting here today, I couldn't tell you
25 which standard for which case.

1 Q. You wrote an expert report in Feindt
2 versus United States, correct?
3 A. Correct.
4 Q. And that's related to the Red Hill
5 litigation, correct?
6 A. Correct.
7 Q. And in that report, you used the more
8 likely than not standard, correct?
9 A. I don't recall.
10 Q. Okay. Assuming for a moment that was
11 true, why would you choose to opine on the more
12 likely than not standard versus as at least as
13 likely than not standard?
14 MR. TELAN: Object to the form.
15 THE WITNESS: Similar to what we discussed
16 for the Camp Lejeune Justice Act, in a -- in a legal
17 setting, I apply the appropriate legal standard when
18 I perform my scientific and medical assessment.
19 BY MR. GIBBONS:
20 Q. Okay. Feindt versus United States was a
21 case brought under the Federal Tort Claims Act,
22 correct?
23 A. Correct.
24 Q. Did you ever review the statutory language
25 of the Federal Tort Claims Act?

1 A. I don't remember.
2 Q. Okay. Were you instructed to opine on the
3 Camp Lejeune Justice Act legal causation standard?
4 MR. TELAN: Object to the form. And don't
5 answer any questions in which he asks you to reveal
6 discussions that you and I have had.
7 BY MR. GIBBONS:
8 Q. Okay. Do you remember which attorney
9 provided you with a copy of Camp Lejeune Justice
10 Act?
11 A. No.
12 Q. Did you conduct any independent research
13 on the Camp Lejeune Justice Act when you were
14 preparing your report for Ms. Rothschild?
15 A. No.
16 Q. Does your background qualify you to review
17 and interpret legal causation standards?
18 MR. TELAN: Object to the form.
19 THE WITNESS: Not as a lawyer, no.
20 BY MR. GIBBONS:
21 Q. Okay. The Camp Lejeune Justice Act
22 contains two standards for the burden of proof,
23 correct?
24 MR. TELAN: Object to the form.
25 / /

1 BY MR. GIBBONS:
2 Q. The first one is sufficient to conclude
3 that a causal relationship exists, and the second
4 one is sufficient to conclude that a causal
5 relationship is at least as likely as not, correct?
6 MR. TELAN: I'll object to the form of the
7 question. Hanley, if you're going to ask her a
8 question about a document, if you'd show her the
9 document. That way she can answer the question
10 since she hasn't memorized that.
11 MR. GIBBONS: That's fine. We'll skip
12 that part.
13 BY MR. GIBBONS:
14 Q. Is it your understanding that the
15 Camp Lejeune Justice Act requires evidence
16 sufficient to conclude that there is at least as
17 likely as not a causal standard?
18 A. Yes.
19 Q. Okay. Have you ever told one of your
20 patients in clinical practice that a specific cause
21 was at least as likely as not the cause of their
22 Parkinson's disease?
23 A. Probably not.
24 Q. Okay. What is your understanding of the
25 phrase "to a reasonable degree of medical and

1 scientific certainty"?
2 MR. TELAN: I'll object to the form as
3 asked and answered.
4 But you can answer it again.
5 THE WITNESS: In science and medicine,
6 it's rare that we can be 100 percent certain about
7 something. But a reasonable degree means that to
8 the best of our ability with available knowledge, we
9 have certainty.
10 BY MR. GIBBONS:
11 Q. Okay. What percentage would you peg as
12 certainty in that definition?
13 MR. TELAN: Object to the form.
14 THE WITNESS: That's hard to say.
15 BY MR. GIBBONS:
16 Q. Does it depend on the literature that
17 you've reviewed?
18 MR. TELAN: Object to the form.
19 THE WITNESS: I wouldn't think of it as a
20 number. Again, just to the best of our ability.
21 BY MR. GIBBONS:
22 Q. So like a gut feeling, then?
23 MR. TELAN: Object to form.
24 THE WITNESS: Science shouldn't be a gut
25 feeling. Of course, we're basing this on evidence

1 and data, but the amount of data, the type of data
2 and evidence will vary case by case.
3 BY MR. GIBBONS:
4 Q. Okay. Have you used the phrase "to a
5 reasonable degree of medical and scientific
6 certainty" in your academic publications?
7 A. No.
8 Q. What about in your clinical practice?
9 A. Again, probably not.
10 Q. In any context outside of litigation?
11 A. Probably not.
12 Q. Are you aware of any published guidance on
13 how to apply the at least as likely as not standard?
14 A. I am generally aware there is literature
15 that describes the standard. I'm not sure what you
16 mean by how to apply the standard.
17 Q. For instance, when evaluating studies or
18 causes of a disease, is there any guidance on how to
19 determine something whether it is at least as likely
20 as not the cause?
21 A. ATSDR 2017 describes clinical equipoise
22 and gives options for what types of data would meet
23 clinical equipoise. I think that's the best example
24 that I can think of sitting here today.
25 Q. Okay. You've opined that TCE can be a

1 cause of Parkinson's disease, correct?
2 A. Are you referring to my opinions on
3 page 64?
4 Q. Correct. And throughout the report.
5 A. Broadly speaking, yes. I would just
6 clarify that my specific opinion is that exposure to
7 water at Camp Lejeune contaminated with PCE and TCE
8 can cause Parkinson's disease.
9 Q. So you're not specifically saying that TCE
10 is the cause of Parkinson's disease?
11 MR. TELAN: Object to the form.
12 THE WITNESS: As I said, I discuss in my
13 report how and why TCE can cause Parkinson disease,
14 but if we're going to reference specifically my
15 opinions from Mrs. Rothschild, I'm also including
16 her PCE exposure.
17 BY MR. GIBBONS:
18 Q. Fair. So I'll rephrase.
19 You've opined that TCE and/or PCE is the
20 cause of Parkinson's disease, correct?
21 MR. TELAN: Object to the form.
22 THE WITNESS: Can be a cause of Parkinson
23 disease.
24 BY MR. GIBBONS:
25 Q. Okay. Is it your opinion that TCE, either

1 by itself or with PCE, in the Camp Lejeune water was
2 sufficient by itself to cause Parkinson's disease?
3 MR. TELAN: Object to the form. And
4 you're -- I don't want to interrupt, Hanley, but
5 you're talking about Ms. Rothschild and not
6 generally? Because she's here to speak on specific
7 causation as to Ms. Rothschild.
8 Was your question more broadly?
9 MR. GIBBONS: Broadly. She offered two
10 opinions in this case.
11 MR. TELAN: She's a specific causation
12 expert.
13 MR. GIBBONS: She has general causation
14 conclusions.
15 MR. TELAN: Understood. We're offering
16 her on specific causation.
17 MR. GIBBONS: Let me ask the question
18 because it's fundamental to her specific causation
19 opinion.
20 MR. TELAN: Okay. You can ask.
21 MR. GIBBONS: Okay. So I'll rephrase --
22 or I'll restate the question.
23 BY MR. GIBBONS:
24 Q. Is it your opinion that TCE and/or PCE in
25 the Camp Lejeune water was sufficient by itself to

1 cause Parkinson's disease?
2 MR. TELAN: Object to the form.
3 THE WITNESS: Yes.
4 BY MR. GIBBONS:
5 Q. Okay. Are you then saying that TCE and
6 PCE, in absence of any other risk factors, could be
7 a cause of Parkinson's disease?
8 MR. TELAN: Object to the form of the
9 question.
10 THE WITNESS: Can you restate the
11 question?
12 BY MR. GIBBONS:
13 Q. Are TCE and PCE, either together or
14 separately, sufficient for an independent cause of
15 Parkinson's disease or are they merely a
16 contributing factor to the cause of Parkinson's
17 disease?
18 MR. TELAN: Object to the form. Compound.
19 THE WITNESS: My answer would depend on
20 the specific data in a -- in a particular exposure
21 case. To answer your question, that, again,
22 depending on the specifics, they can be a cause of
23 Parkinson disease.
24 BY MR. GIBBONS:
25 Q. Okay. So in your opinion, general

1 causation for TCE and/or PCE, either independently
2 or together, is sufficient to cause Parkinson's
3 disease in some circumstances, and your role then
4 would be to apply these specific factors of a case
5 to that -- to arrive at your conclusions, correct?
6 A. Correct.
7 Q. Okay. You would agree with the principle
8 that the dose makes the poison, correct?
9 MR. TELAN: Object to the form.
10 THE WITNESS: Generally, yes, I agree that
11 dose is a relevant consideration.
12 BY MR. GIBBONS:
13 Q. Okay. You're aware that it's a
14 fundamental principle of toxicology, correct?
15 A. Correct.
16 MR. TELAN: Object to the form.
17 BY MR. GIBBONS:
18 Q. In general, the risk for developing a
19 disease from a chemical exposure increases with
20 dose, correct?
21 MR. TELAN: Object to the form.
22 THE WITNESS: Not necessarily.
23 BY MR. GIBBONS:
24 Q. Can you offer an example?
25 MR. TELAN: Object to the form of the

1 question.
2 THE WITNESS: I don't have any specific
3 examples today.
4 BY MR. GIBBONS:
5 Q. Okay. What's the level of exposure to TCE
6 that's necessary to cause Parkinson's disease?
7 MR. TELAN: Form objection.
8 THE WITNESS: What we know about TCE is
9 based on retrospective studies because the types of
10 experiments that would allow us to answer your
11 question with a number would be inhumane and
12 unethical. We can't give people titrating doses of
13 a poison in order to simply say that we know exactly
14 which number would answer your question.
15 BY MR. GIBBONS:
16 Q. Okay.
17 A. But we have guidance from the EPA, with
18 MCLs of five parts per billion, and we have a large
19 body of -- of retrospective cohort literature
20 looking at populations where median concentrations
21 are known.
22 Q. You indicated that the EPA uses maximum
23 contaminant levels to evaluate potential risks to
24 human health, correct?
25 A. The EPA sets goal maximum contaminant

1 levels in order to protect populations from toxic
2 exposures.
3 Q. Okay. Are you aware of how the EPA
4 establishes an MCL?
5 A. I'm not aware of their methodology.
6 Q. Okay. Were you aware the MCLs were
7 designed to be acceptable drinking water
8 concentrations for a lifetime of exposure?
9 MR. TELAN: Object to the form.
10 THE WITNESS: I'm aware that there are
11 goals for water concentrations. I'm not sure what
12 you mean by "for a lifetime of exposure."
13 BY MR. GIBBONS:
14 Q. Meaning that they are designed to be the
15 acceptable threshold for someone that's exposed to
16 that same concentration of a chemical for their
17 entire lifetime --
18 MR. TELAN: Object --
19 BY MR. GIBBONS:
20 Q. -- which is, under the EPA, approximately
21 70 years.
22 MR. TELAN: Object to the form of the
23 question.
24 THE WITNESS: I am generally aware of
25 that.

1 BY MR. GIBBONS:
2 Q. Would you agree that Ms. Rothschild was
3 not exposed to the Camp Lejeune water for 70 years?
4 A. She was not exposed for 70 years. But she
5 was exposed for far longer than cohorts of people
6 who were included in relevant studies that I cited
7 where associations were shown between Parkinson's
8 and PCE or TCE exposure.
9 Q. You indicated that we wouldn't have
10 specificity because of the types of studies that
11 would be needed to determine the specific level of
12 causation -- or the specific level of TCE that's
13 causative for Parkinson's disease for humans.
14 But you do have studies from animals,
15 correct?
16 A. We have studies from animals, from
17 cellular models and from humans that address what is
18 known about certain levels. As I said, we have
19 human studies that can demonstrate median
20 concentrations that can look retrospectively at the
21 Camp Lejeune water sources and measure the TCE and
22 PCE in the water. This is, of course, far more
23 specific and -- and illustrative than we have for
24 most epidemiologic studies.
25 Q. Okay. Is there a specific level of

1 exposure to PCE that's necessary to cause
2 Parkinson's disease?
3 MR. TELAN: Object to the form of the
4 question.
5 THE WITNESS: Again, I don't doubt that
6 that level exists, but it would not be ethical to
7 conduct the studies to tell us what that number is.
8 BY MR. GIBBONS:
9 Q. Okay. If you don't know what the specific
10 number is, how can you offer an opinion that the
11 exposure that Ms. Rothchild had was at least as
12 likely as not the cause of her Parkinson's disease?
13 MR. TELAN: Objection. Argumentative.
14 THE WITNESS: Because we have multiple
15 studies looking at the same population of people as
16 Ms. Rothschild is included in, the same years on
17 base as Ms. Rothschild was at Camp Lejeune and
18 exposure data for the same water that she drank at
19 Camp Lejeune. And we can model what those exposures
20 were, and we can show that those exposures cause
21 Parkinson's. We can calculate the risk of
22 Parkinson's, the increased risk of Parkinson's in
23 those populations, and we can compare those levels
24 that we know and were studied to Ms. Rothschild's
25 levels and say she's part of that population, she

1 had at least that exposure, if not more, in
2 concentration and duration.
3 And that satisfies the opinion. It
4 doesn't necessitate knowing what the minimum
5 threshold number is exactly.
6 BY MR. GIBBONS:
7 Q. Okay. Is it your opinion that any amount
8 of exposure to TCE is sufficient to cause
9 Parkinson's disease?
10 MR. TELAN: Object to the form.
11 THE WITNESS: That's a very hypothetical
12 question. We know TCE is toxic. It's a chemical
13 that's been banned completely by the EPA. I would
14 need to know more specifics in order to answer your
15 question.
16 BY MR. GIBBONS:
17 Q. Okay. Would exposure to a single molecule
18 of TCE be sufficient to cause Parkinson's disease?
19 MR. TELAN: Objection. Overbroad.
20 Incomplete hypothetical.
21 THE WITNESS: I don't know.
22 BY MR. GIBBONS:
23 Q. Is it your opinion that exposure to any
24 amount of PCE is sufficient to cause Parkinson's
25 disease?

1 MR. TELAN: Same objection.
2 THE WITNESS: Same answer.
3 BY MR. GIBBONS:
4 Q. Okay. Same question.
5 Would an exposure to a single molecule of
6 TCE be enough to cause Parkinson's disease?
7 MR. TELAN: Same objection.
8 THE WITNESS: Didn't you just ask that
9 same question or did I miss something?
10 BY MR. GIBBONS:
11 Q. That was the previous question. Any level
12 of exposure. I'm asking now about a specific single
13 molecule.
14 A. Ah, I don't know.
15 Q. Okay. Are you aware of what the lowest
16 level of exposure to TCE that's been shown in a
17 well-conducted epidemiological study to increase the
18 risk of Parkinson's disease is?
19 MR. TELAN: Form.
20 THE WITNESS: Of TCE?
21 BY MR. GIBBONS:
22 Q. Correct.
23 A. We have Goldman 2023 with a median
24 micrograms per liter monthly concentration of 366.
25 And we have several other epidemiologic studies that

1 included persons with so-called ever exposure to TCE
2 that was unquantified. And other studies that
3 included persons exposed to -- exposed to TCE for as
4 little as less than one hour a week for less than
5 one year.
6 Q. Okay. Same question with PCE.
7 What is the lowest level of PCE exposure
8 that's been shown in a well-conducted
9 epidemiological study to increase the risk of
10 Parkinson's disease to make it at least as likely as
11 not that the disease will develop?
12 MR. TELAN: Form objection.
13 THE WITNESS: Similar answers. The
14 distinctions are, for Goldman 2023, the median
15 monthly concentration was approximately
16 15 micrograms per liter. And, again, in these ever
17 exposures and ever epidemiologic studies, sometimes
18 chlorinated solvents as a class were looked at,
19 which included both TCE and PCE, but did not
20 distinguish between them.
21 BY MR. GIBBONS:
22 Q. These ever exposure studies, do you recall
23 the names of any of these studies?
24 A. We have -- we have Pizoli, et al. 2012.
25 We have Nielsen 2021. I believe at least one of

1 those was an ever exposure.
2 Q. Okay.
3 A. And at least one was a study that relied
4 on self-report of exposure. So, of course, less
5 specific and more prone to confounding, but still
6 showed a significant relationship between these
7 solvents and Parkinson's.
8 Q. Were all those studies included in your
9 materials considered list?
10 A. Yes.
11 Q. Okay. Do you recall whether those studies
12 were cohort studies or case control studies?
13 A. I believe they were cohort studies.
14 Q. Okay. All of the ones that you just
15 described?
16 A. I believe so, but I don't recall
17 specifically.
18 Q. Okay.
19 A. Of course, Goldman 2012 is case control,
20 and we can cite to others that -- that had even more
21 rigorous methods.
22 Q. Apart from Goldman being a case control
23 study, those cohort studies, did they deal with
24 actual exposure data?
25 A. Can you expand on the meaning of "actual"?

1 Q. For instance, you said that they were --
2 some of them were self-reported, correct?
3 A. Correct.
4 Q. Were they corroborated by any readings of
5 the concentrations of chemicals to corroborate?
6 A. I don't recall.
7 Q. Okay. Do you know if all of those studies
8 found statistically significant risks?
9 MR. TELAN: Object to the form.
10 THE WITNESS: It's an important
11 distinction. Sometimes we might define
12 statistically significant as a P value less than a
13 certain amount, such as 0.05, or look at confidence
14 intervals that don't include one, for example. And,
15 of course, in some of these studies, we're looking
16 at small populations. And so we may have a
17 practically notable odds ratio or hazard ratio. But
18 because the population was small, those -- a
19 confidence interval or a P value may not exactly fit
20 within that threshold. That doesn't mean that the
21 statistic is not meaningful or doesn't have
22 practical relevance. It just needs to be put in
23 context of what those tools mean.
24 For example, if a P value less than 0.05
25 means that there's a 95 percent chance that the

1 relationship studied is not due to random chance, is
2 it still valuable to know if there's a 94.5 percent
3 chance of the same thing, yes.
4 BY MR. GIBBONS:
5 Q. I think I understand your meaning.
6 Do you know if any of those studies
7 observed dose response gradients?
8 A. I have cited to two studies in my report
9 that observed dose response gradients. I don't
10 recall without looking at the articles now whether
11 the ones I just mentioned do.
12 Q. Okay. Can you identify any studies where
13 TCE exposure was found to cause Parkinson's disease
14 at a dose similar to what's alleged in this case?
15 MR. TELAN: Object to the form.
16 Overbroad. Incomplete hypothetical.
17 THE WITNESS: That's hard to say. We
18 don't always have the -- the measured TCE
19 concentrations, especially in the majority of
20 retrospective cases. For Mrs. Rothschild, her PCE
21 exposure was likely more relevant.
22 BY MR. GIBBONS:
23 Q. Okay. So same question with PCE exposure,
24 then.
25 A. Can you restate the question?

1 Q. Sure.
2 Can you identify any studies where PCE
3 exposure was found to have caused Parkinson's
4 disease at a similar dose to what's alleged in this
5 case?
6 MR. TELAN: Object to the form.
7 THE WITNESS: Again, we have at least
8 Goldman 2023 that studied a population of people who
9 were on base for at least three months and had
10 median concentrations of 15 micrograms per liter,
11 whereas Mrs. Rothschild's modeled exposure to PCE
12 was almost triple that at greater than 40 micrograms
13 per liter. And, of course, she was on base for far
14 longer than three months.
15 BY MR. GIBBONS:
16 Q. We'll come back to this later, but that
17 exposure was based on the report of
18 Dr. Kelly Reynolds, correct?
19 A. Yes. Dr. Reynolds modeled the exposure.
20 Q. Okay. People without exposure to TCE can
21 still develop Parkinson's disease, correct?
22 A. Correct.
23 Q. And people without exposure to PCE can
24 still develop Parkinson's, correct?
25 A. Correct.

1 Q. Okay. In your neurology practice, how
2 many of your Parkinson's disease patients have
3 indicated that they have a history of exposure to
4 TCE?
5 MR. TELAN: Object to the form. Asked and
6 answered.
7 THE WITNESS: I think we discussed earlier
8 that sometimes patients will share with me what
9 occupations they had, what broad exposures they --
10 they're curious about. But we rarely get as
11 granular to discuss specific chemicals.
12 BY MR. GIBBONS:
13 Q. Okay. Same question for PCE.
14 A. Yes. Same answer.
15 Q. Same answer? Okay.
16 You evaluated the effects of the
17 chemicals, meaning PCE and TCE, individually,
18 correct?
19 A. I considered them separately and together,
20 yes.
21 Q. Okay. That was my next question.
22 So you also considered them together as a
23 mixture?
24 A. Meaning that in some studies, TCE, PCE
25 and/or other chlorinated solvents in the same class

1 are studied together when they cannot be parsed
2 apart, and in other cases, they are studded
3 separately when that data exists.
4 Q. Okay. We talked about this a little bit
5 earlier, but you framed your opinion as you believe
6 that exposure to the water at Camp Lejeune was at
7 least as likely as not the cause of Ms. Rothschild's
8 Parkinson's disease, correct?
9 A. You are correct that that is how I phrased
10 it. And, of course, I also mentioned other relevant
11 routes of exposure for TCE and PCE, such as dermal
12 and inhalation routes, and any exposure that
13 Mrs. Rothschild had through those additional routes
14 would be additive to the -- the calculated water
15 ingestion levels.
16 Q. Okay. But the phrasing of the exposure to
17 the water at Camp Lejeune, that would be a mix of
18 PCE and TCE, correct?
19 A. That would depend on the water source, and
20 yes, for Mrs. Rothschild, it's a mix.
21 Q. Okay. Well, let me clarify, then.
22 What did you mean by exposure to the water
23 at Camp Lejeune?
24 A. To water at Camp Lejeune contaminated with
25 PCE and TCE.

1 Q. Okay.
2 A. And I'm just reading.
3 Q. That's fine.
4 A. I'm quoting from page 64.
5 Q. So is it your understanding that different
6 parts of the base had different combinations of PCE
7 and TCE?
8 A. Had different concentrations, yes.
9 Q. Okay. What years did you look at the
10 Camp Lejeune water in reaching your conclusions?
11 A. Two things to note. One, Mrs. Rothschild
12 was on base between 1973 and 1975, I believe. And
13 so those were the relevant years where Dr. Reynolds'
14 modeled water exposures specifically for
15 Mrs. Rothschild.
16 Q. Okay. So you didn't consider any
17 concentration levels from years outside of the years
18 relevant to Ms. Rothschild's exposure?
19 A. I'm aware of them. I've read about them.
20 I've considered them. But they don't play directly
21 into Mrs. Rothschild personal exposure because she
22 didn't consume water on base outside of those years.
23 Q. Right.
24 And you are only offering an opinion that
25 the specific water at Camp Lejeune that

1 Ms. Rothschild was exposed to was sufficient to
2 cause her Parkinson's disease, correct?
3 A. Correct.
4 MR. TELAN: I was going to ask you to
5 repeat that again.
6 MR. GIBBONS: Reporter, can you read that
7 back, please?
8 (Record read by the reporter
9 as follows:
10 QUESTION: And you are only
11 offering an opinion that the
12 specific water at Camp Lejeune
13 that Ms. Rothschild was exposed to
14 was sufficient to cause her
15 Parkinson's disease, correct?
16 ANSWER: Correct.)
17 THE WITNESS: I would say my opinion,
18 again, is that her exposure was at least as likely
19 as not a cause of her Parkinson disease.
20 BY MR. GIBBONS:
21 Q. Okay. Based on the literature that you've
22 reviewed, would you be comfortable saying that it
23 was more likely than not the cause of her
24 Parkinson's disease?
25 MR. TELAN: Object to the form.

1 THE WITNESS: That is outside of the scope
2 of what I'm prepared to discuss today.
3 BY MR. GIBBONS:
4 Q. Okay. Well, but during the course of your
5 review, did you get a sense that if the standard was
6 more likely than not, that you thought it would have
7 reached that level?
8 MR. TELAN: Object to the form.
9 THE WITNESS: Yes.
10 BY MR. GIBBONS:
11 Q. Okay. Did you account for the differences
12 in the mixture of PCE and TCE in the water during
13 Ms. Rothschild's exposure?
14 A. What do you mean by "the differences in
15 the mixture"?
16 Q. Well, the relative concentrations of PCE
17 and TCE were not static the entire time that
18 Ms. Rothschild was at Camp Lejeune, correct?
19 A. Correct.
20 Q. Okay. And when you were making your
21 assessment, did you account for any of the
22 fluctuations in these concentrations during her time
23 there?
24 MR. TELAN: Object to the form.
25 THE WITNESS: Dr. Reynolds accounted for

1 those fluctuations. I've included Dr. Reynolds'
2 model exposure levels in my report. And so in that
3 sense, yes, I have considered them and accounted for
4 them.
5 BY MR. GIBBONS:
6 Q. Okay. So you defer to Dr. Reynolds'
7 exposure concentration -- or exposure models, then,
8 correct?
9 A. Yes.
10 Q. Okay. Have any regulatory agencies
11 recognized the mixtures -- sorry -- the specific
12 mixture of PCE and TCE that Ms. Rothschild was
13 exposed to as a known or probable cause of
14 Parkinson's disease?
15 MR. TELAN: Object to the form.
16 THE WITNESS: We have the Institute of
17 Medicine 2015 report, which considered TCE and PCE
18 in the water at Camp Lejeune in making their
19 designation for service connection for subjects at
20 the VA and, of course, we have ATSDR considering TCE
21 and PCE. I think what you're asking is about the
22 specific levels, and those institutional analyses
23 would have been in a broader context rather than
24 specific to, of course, one plaintiff in a -- in a
25 case that hadn't been brought at the time those

1 reports were written.
2 BY MR. GIBBONS:
3 Q. What Institute of Medicine study were you
4 talking about? I believe it was from 2015.
5 A. Yes. I've cited to it in my report. It's
6 the -- it's the document that the VA used to base
7 service connection for veterans who served at
8 Camp Lejeune for 30 days or more and later developed
9 Parkinson disease.
10 Q. Do you recall what you specifically cited
11 to that in the report or in the materials
12 considered?
13 (Reporter clarification.)
14 BY MR. GIBBONS:
15 Q. Do you recall whether you specifically
16 cited to that in the body of the report, or was that
17 only in your materials considered?
18 A. In the body of the report.
19 Q. Okay. Do you know of any scientific
20 literature that specifically studied the effects of
21 the mixture on the development of Parkinson's
22 disease? When I say "mixture," I mean mixture of
23 TCE and PCE that Ms. Rothschild was exposed to.
24 A. Yes. We have an excellent example in
25 Goldman 2012, which is the well-controlled twin

1 study in veterans that looked at odds ratios for TCE
2 and PCE individually, and also, for the combination
3 and found a very high odds ratio of nine, greater
4 than nine, for the combination of TCE and PCE to
5 cause Parkinson's.
6 Q. Okay. We'll come back to that one.
7 Was the Goldman 2012 twin study -- sorry.
8 Did the Goldman 2012 twin study contain
9 any dose response assessment?
10 A. I don't remember.
11 Q. Would you agree that the exact
12 relationship between the interactions of PCE, TCE,
13 Benzene and vinyl chloride is not known?
14 MR. TELAN: Object to the form.
15 THE WITNESS: As the question is stated,
16 the exact relationship is perhaps not well
17 characterized, but each of those chlorinated
18 solvents have been studied individually and as a
19 group both in cohort studies and well-designed
20 meta-analyses, animal studies, et cetera.
21 BY MR. GIBBONS:
22 Q. Okay.
23 A. If I may, I would go back to your prior
24 question about dose response in 2012, Goldman, and
25 the answer is yes, and I describe it on page 52 of

1 my report. Describing there is evidence of an
2 exposure response relationship in this study between
3 duration of exposure and cumulative exposure.
4 Q. Okay. Thank you.
5 A. Mm-hmm.
6 Q. Based on what we've just discussed, the
7 Goldman 2012 studies and any other studies you've
8 referenced, would you agree that there is a
9 synergistic effect of TCE and PCE in terms of
10 causation for Parkinson's?
11 MR. TELAN: Object to the form.
12 THE WITNESS: Can you define or explain
13 how you used the term "synergistic"?
14 BY MR. GIBBONS:
15 Q. Would you -- is it your understanding that
16 TCE and PCE exposure together is more likely to
17 cause Parkinson's disease than pure exposure to TCE
18 or pure exposure to PCE?
19 MR. TELAN: Object to the form of the
20 question.
21 THE WITNESS: I'm not aware of studies
22 that demonstrate a more than additive response as
23 you define "synergy." We do have the odds ratios in
24 Goldman 2012, which show, for example, odds ratio of
25 PCE to be greater than ten, the odds ratio of PCE

1 and TCE to be about the same, greater than nine.
2 And I think what's relevant to understand here is
3 that PCE and TCE are structurally very similar
4 molecules. They have the same downstream
5 metabolite, they are metabolized to TaClo. In
6 regions of the brain, such as the substantia nigra,
7 that are very relevant for Parkinson's by enzymes
8 that are specifically in the substantia nigra and
9 that the downstream common metabolite is also very
10 analogous to MPTP, the molecule that we know causes
11 Parkinson's disease in other models.
12 So based on that totality of evidence,
13 again, while I can't rule out the synergistic
14 relationship, I think it's at least clear that their
15 additive, that they're similar, that their
16 downstream metabolites are the same.
17 BY MR. GIBBONS:
18 Q. Okay.
19 THE VIDEOGRAPHER: Sorry to interrupt.
20 Touching the cable is causing a little bit of
21 interference. Thank you.
22 BY MR. GIBBONS:
23 Q. Would you agree that association is not
24 the same thing as causation?
25 A. Yes.

1 Q. Okay. Generating an opinion on causation
2 is not as simple as just having a study that shows
3 statistically significant association between a
4 substance and a disease, correct?
5 MR. TELAN: Object to the form of the
6 question.
7 And just for purposes, you're -- are you
8 asking general causation or specific with your
9 question there?
10 MR. GIBBONS: Generally.
11 THE WITNESS: Again, I would go back to
12 ATSDR 2017 that talks about -- that provides
13 examples of how clinical equipoise could be
14 established, and sometimes it is mechanistic data,
15 animal data and a well-designed meta-analysis, for
16 example.
17 BY MR. GIBBONS:
18 Q. Okay. For example, would it be possible
19 to have a study showing a statistically significant
20 association between a substance and a disease and
21 still not be able to conclude that the substance can
22 cause the disease?
23 MR. TELAN: Object to the form of the
24 question.
25 THE WITNESS: I can't answer that without

1 knowing more specifics.
2 BY MR. GIBBONS:
3 Q. Okay. You wouldn't normally draw a
4 conclusion about causation from a single study,
5 right?
6 MR. TELAN: Object to the form of the
7 question.
8 THE WITNESS: That would depend on the
9 study and the totality of the evidence in that
10 particular hypothetical.
11 BY MR. GIBBONS:
12 Q. Even if it was just a single study, it
13 would depend?
14 MR. TELAN: Object to form.
15 THE WITNESS: It's hard to say.
16 BY MR. GIBBONS:
17 Q. Okay. But you would agree that typically
18 you want to see a study's result replicated to some
19 degree, correct?
20 MR. TELAN: Object to the form.
21 THE WITNESS: When a result is replicated,
22 it -- it adds to the weight of the evidence.
23 BY MR. GIBBONS:
24 Q. Okay.
25 A. That's not always possible.

1 Q. If there's only a single study, that would
2 introduce the possibility that its results were
3 chance, correct?
4 MR. TELAN: Object to the form.
5 THE WITNESS: I disagree.
6 BY MR. GIBBONS:
7 Q. Would you agree that seeing a study's
8 results replicated would reduce the odds that its
9 findings were purely based on chance?
10 A. Perhaps. But, again, dependent on the
11 specifics.
12 Q. Okay. Generally, the authors of
13 epidemiological studies provide statistical results
14 that indicate their level of risks observed,
15 correct?
16 A. Generally, yes.
17 Q. But it's generally not possible -- or
18 strike that.
19 But it's not possible that from this
20 information alone to say whether a particular case
21 of a disease in a study was caused by exposure
22 versus another risk factor, right?
23 MR. TELAN: Object to the form.
24 THE WITNESS: When you phrase it as "was
25 caused by," again, if it means 100 percent cause can

1 be opposite of ruling out, I agree that's generally
2 hard to say.
3 BY MR. GIBBONS:
4 Q. Okay.
5 A. And so we're back to a reasonable degree
6 of certainty.
7 Q. You would agree it's important to analyze
8 risk ratios in studies results, correct?
9 MR. TELAN: Object to the form.
10 THE WITNESS: A risk ratio. Do you mean a
11 relative risk?
12 BY MR. GIBBONS:
13 Q. Correct.
14 A. Or an odds ratio or a hazard ratio or
15 whatever was the appropriate statistical application
16 germane to a particular study.
17 Q. It would be important to risk -- to
18 analyze the risk or odds ratio or whatever the
19 general assessment is for the study, then, correct?
20 A. Yes.
21 Q. Okay. And a risk ratio indicates the
22 level of association that's observed, correct?
23 A. Can you restate that?
24 Q. The risk ratio indicates the level of
25 association observed?

1 A. Do you mean a relative risk?
2 Q. Yes. Sorry. Relative risk ratio.
3 A. Generally, yes.
4 Q. Okay. And 1.0 would indicate no
5 association, correct?
6 MR. TELAN: Object to the form.
7 THE WITNESS: In this case, we mostly have
8 odds ratios, and an odds ratio of 1 would suggest
9 that there's no increased risk.
10 BY MR. GIBBONS:
11 Q. What level of increased risk would reflect
12 a modest association in an odds ratio?
13 MR. TELAN: Object to the form of the
14 question.
15 THE WITNESS: We don't define deltas in
16 risk as mild, moderate or severe necessarily, but we
17 could look to ATSDR as defining odds ratios greater
18 than 1.1 as relevant for a disease and other studies
19 using 1.2 as a cutoff.
20 BY MR. GIBBONS:
21 Q. Okay. When you say, "1.2 as a cutoff,"
22 what do you mean exactly?
23 A. I think the ATSDR definition is the best
24 example, but I recognize that I cited to a study in
25 my report that commented that -- that an odds ratio

1 of greater than 1.2 was relevant or meaningful.
2 Q. Okay. So greater than 1.2 would be
3 relevant or meaningful.
4 What about 2.0?
5 MR. TELAN: I'll object to the form and
6 move to strike the statement at the front of the
7 question.
8 THE WITNESS: All I can say is that an
9 odds ratio of two represents a doubling of the risk.
10 BY MR. GIBBONS:
11 Q. Okay. And then odds ratio of 3.0 would be
12 a tripling of the risk, then?
13 A. Correct.
14 Q. Would you agree it's important to analyze
15 confidence intervals in a study's results?
16 MR. TELAN: Object to the form.
17 THE WITNESS: As I mentioned before, we
18 take them into consideration in the context of the
19 study and its strengths and limitations, sometimes
20 the confidence intervals don't include one,
21 especially when there's a small population studied,
22 as is often the case in Parkinson's.
23 BY MR. GIBBONS:
24 Q. Okay. Confidence intervals evaluate how
25 precise the risk assessment is, correct?

1 MR. TELAN: Object to the form.
2 THE WITNESS: I can agree with that.
3 MR. GIBBONS: Okay. I think this is a
4 good time for a break. We've been going for about
5 an hour.
6 THE VIDEOGRAPHER: We're going off the
7 record at 11:24 a.m.
8 (Whereupon, a recess was taken from
9 11:24 a.m. to 11:36 a.m.)
10 THE VIDEOGRAPHER: We are back on the
11 record at 11:36 a.m.
12 BY MR. GIBBONS:
13 Q. Dr. Andruska, would you agree that there's
14 a hierarchy of epidemiological evidence generally
15 where meta-analysis in systematic reviews are
16 considered more reliable than randomized controlled
17 studies?
18 MR. TELAN: Object to the form of the
19 question.
20 THE WITNESS: Reliable is an interesting
21 word. Often meta-analyses, depending on the
22 specifics, can give a broader picture and at first
23 synthesis the results of multiple smaller trials.
24 And this case is very unique in that we have
25 specific studies that look at Camp Lejeune and the

1 actual water that is in question here. That's
2 not -- that's very rare. And so it gives us even
3 extra insight and very specific and informative data
4 about Mrs. Rothschild in particular.
5 BY MR. GIBBONS:
6 Q. Okay. Would you agree that randomized
7 controlled studies are generally more reliable than
8 cohort studies?
9 A. They can be informative, yes. This also
10 gets to that point I made earlier about
11 administering toxins to innocent people. We
12 wouldn't do that in a toxic exposure question. So
13 given that, we wouldn't say you get placebo and you
14 get toxic chemical. We -- we rely more on the
15 cohort studies.
16 Q. Okay. Do you rely more on cohort studies
17 than case control studies?
18 A. Rely was a poor choice of words on my
19 part. I might say instead we have more cohort
20 and/or case control studies in a case like this
21 where there's a toxic exposure at question.
22 Q. Okay. If there was a disagreement between
23 a cohort study and a case control study, which one
24 would you go with?
25 MR. TELAN: Object to the form of the

1 question.
2 THE WITNESS: It's an interesting
3 question. As you know, so many particulars about
4 the design of a study, and a cohort study may be
5 more well designed in many aspects, and thus, in a
6 particular head-to-head comparison may be the quote,
7 unquote better study. Where we have case control
8 design, like in twin studies, we can confound -- we
9 can control for many confounders, like genetic
10 modifications and environmental exposures. And we
11 have to consider the pros and cons of each study in
12 their own context.
13 BY MR. GIBBONS:
14 Q. So it would just depend on the particulars
15 of each of those respective cases, then?
16 A. Yes.
17 Q. Okay. Would case control studies
18 generally be more favorable than cross-sectional
19 studies?
20 MR. TELAN: Object to the form of the
21 question.
22 THE WITNESS: Same answer as for cohort.
23 BY MR. GIBBONS:
24 Q. Okay. What about case control studies
25 compared to animal studies?

1 MR. TELAN: Object to the form of the
2 question.
3 THE WITNESS: Where we can model human
4 exposures in relevant and comparable cohorts, that's
5 useful. But, of course, in science and medicine, we
6 consider animal data, cellular data, mechanistic
7 data, it's all an important part of the equation.
8 BY MR. GIBBONS:
9 Q. Okay. Would you agree that generally,
10 when analyzing causation, cohort studies should
11 carry more weight in a case control study?
12 MR. TELAN: Object to the form.
13 THE WITNESS: Did you ask if cohort
14 studies should carry more weight than a case control
15 study?
16 BY MR. GIBBONS:
17 Q. Correct.
18 A. I can't agree to that.
19 Q. Okay. Would you disagree with that?
20 MR. TELAN: Object to the form.
21 THE WITNESS: Again, I just couldn't say
22 depending on the particulars.
23 BY MR. GIBBONS:
24 Q. Okay. Should a cohort study carry more
25 weight than an ecological study?

1 MR. TELAN: Object to the form of the
2 question.
3 THE WITNESS: I'm not sure what you mean
4 by "ecological study."
5 BY MR. GIBBONS:
6 Q. Fair enough.
7 (Whereupon, Deposition Exhibit 5 and
8 Exhibit 6 were marked for
9 identification.)
10 THE WITNESS: Thank you.
11 BY MR. GIBBONS:
12 Q. Dr. Andruska, I'm showing you what's been
13 marked for identification as Exhibits 5 and 6.
14 Do you recognize these documents?
15 A. Yes, I do.
16 Q. Okay. What are these documents?
17 A. Exhibit 5 is my Appendix A of reliance
18 materials, and Appendix 6 is an amended materials
19 considered list.
20 Q. Okay. Great.
21 MR. GIBBONS: And I'll just record for the
22 record that we received another supplemental
23 materials considered list last night. However, I
24 didn't have a chance to review it or print it out
25 for today. So we are going to leave the deposition

1 open for any questions that I might need to ask
2 regarding new materials that were disclosed last
3 night.
4 MR. TELAN: I would object to that.
5 That's something we can chat about, but, at this
6 point, I'll object to that.
7 MR. GIBBONS: All right. We can discuss
8 more off the record.
9 BY MR. GIBBONS:
10 Q. Dr. Andruska, between appendix -- or
11 Exhibits 5 and 6, excluding anything that might have
12 been produced last night, are these a complete and
13 accurate list of the works that you relied upon for
14 your report?
15 A. To the best of my knowledge --
16 Q. Okay.
17 A. -- yes.
18 Q. Were there any documents that you
19 reviewed, but decided not to rely on when writing
20 your report?
21 A. No.
22 Q. Okay. You didn't perform a systematic
23 review in finding these results, did you?
24 A. What do you mean by "systematic"?
25 Q. Would you agree that a search should be

1 calibrated to have both positive and negative
2 results?
3 MR. TELAN: Object to the form of the
4 question.
5 THE WITNESS: As I described in my report,
6 I performed a search for literature that was
7 relevant to Parkinson's and TCE and PCE and the
8 other search terms that I mentioned, and I did not
9 intentionally exclude literature.
10 BY MR. GIBBONS:
11 Q. Okay. And that's on Exhibit 4, I believe,
12 your initial report, right?
13 A. My report is Exhibit 3.
14 Q. 3. Sorry.
15 A. And Section 4 is my methodology.
16 Q. All right. And you performed a search
17 using PubMed, correct?
18 A. Correct.
19 Q. And Google Scholar? It's on page 6 of
20 your report.
21 A. Yes.
22 Q. Okay. Did you use any other research
23 databases?
24 A. No.
25 Q. What was your search criteria in these

1 databases?
2 A. Again, reading from page 6 (as read):
3 TCE, PCE, PERC,
4 trichloroethylene,
5 tetrachloroethylene
6 perchloroethylene, Parkinson,
7 dopamine, neurologic,
8 neurotoxicity.
9 Q. Okay. Sitting here today, are there any
10 other search terms that you can remember that you
11 used?
12 A. Not specifically, although I may have
13 searched for things that were germane to the terms
14 listed, like chlorinated solvents or similar, but I
15 don't recall. Nothing outside of this domain.
16 Q. Do you believe that this search criteria
17 would produce existing contrary results, for
18 instance, studies that found that there was no
19 causal link between TCE and Parkinson's?
20 MR. TELAN: Object to the form.
21 THE WITNESS: They could, yes.
22 BY MR. GIBBONS:
23 Q. And same question for PCE.
24 A. Yes.
25 Q. Were you provided with any studies that

1 you relied on for your report?
2 A. Not that I hadn't already found on my own.
3 Q. Okay. You found the ATSDR study on your
4 own, then?
5 A. Yes.
6 (Whereupon, Deposition Exhibit 7 was
7 marked for identification.)
8 BY MR. GIBBONS:
9 Q. Dr. Andruska, I've just handed you what's
10 going to be Exhibit 7, the ATSDR 2017 assessment of
11 the evidence.
12 Is this the study that you're referring
13 to?
14 A. Yes.
15 MR. TELAN: Object to the form.
16 BY MR. GIBBONS:
17 Q. Okay. Are you aware that this literature
18 review found equipoise for TCE causing Parkinson's?
19 A. I believe so, yes.
20 Q. Okay.
21 A. We can look at it together if you like.
22 Q. Okay. Sure.
23 Can you turn to page 99, please?
24 MR. TELAN: Did you already mark this,
25 Hanley, by the way?

1 MR. GIBBONS: Yes.
2 MR. TELAN: You did, okay.
3 THE WITNESS: Yes.
4 BY MR. GIBBONS:
5 Q. Okay.
6 A. I agree.
7 Q. On page 99, under the conclusion
8 paragraph, about third sentence in, it reads (as
9 read):
10 Because only two studies have
11 focused on TCE exposure, Goldman et
12 al. 2012, and Bove, et al. 2014B,
13 the epidemiological evidence for
14 causation for PCE and Parkinson's
15 disease is very limited --
16 correction -- for TCE Parkinson's
17 disease is very limited.
18 Did I read that correctly?
19 A. Yes.
20 Q. Okay. And then the last sentence of that
21 paragraph says (as read):
22 However, given the strong
23 supporting mechanistic evidence for
24 TCE, the ATSDR concludes that there
25 is equipoise and above evidence for

1 causation for TCE in Parkinson's
2 disease.
3 Correct?
4 A. That's correct.
5 Q. Okay.
6 A. And, of course, I'm just calling out that
7 2017 predates the last eight years of literature
8 that we've had since then that would impact these
9 assessments.
10 Q. Okay. And you're aware that the ATSDR
11 report found that there was below equipoise for PCE
12 and Parkinson's disease, correct?
13 A. Yes. In 2017.
14 Q. Okay. And that's the last paragraph on
15 page 99, which reads (as read):
16 For PCE, the epidemiological
17 evidence is very limited and there
18 is no available information on the
19 plausible mechanism as there is for
20 TCE.
21 Did I read that correctly?
22 A. You read that correctly.
23 Q. Okay. And then the last sentence is (as
24 read):
25 Given what is presently known,

1 ATSDR concludes that there is below
2 equipoise evidence for causation of
3 PCE in Parkinson's disease.
4 Correct?
5 A. You read that --
6 MR. TELAN: Object to the form.
7 THE WITNESS: You read that correctly.
8 BY MR. GIBBONS:
9 Q. Okay. Are you aware of how long an
10 epidemiological study takes to plan and perform?
11 MR. TELAN: Object to the form.
12 THE WITNESS: I'm sure it varies, but well
13 conducted science can take some time.
14 BY MR. GIBBONS:
15 Q. Okay. Were you aware that Dr. Frank Bove
16 was the sole author of this review?
17 A. I'm aware that he is a leading
18 epidemiologist at the ATSDR.
19 You asked if he was the solo author of
20 this?
21 Q. Correct.
22 Were you aware of that?
23 A. I'd have to look.
24 Q. I'll represent to you that he admitted as
25 much in his deposition.

1 MR. TELAN: So then there's no question,
2 then?
3 MR. GIBBONS: I was asking if she was
4 aware of it.
5 MR. TELAN: I know. I'm just messing with
6 you.
7 BY MR. GIBBONS:
8 Q. Okay. Would you agree that cumulative
9 exposure to a chemical by itself does not provide
10 full insight into the risk associated with that
11 exposure?
12 MR. TELAN: Object to the form of the
13 question.
14 THE WITNESS: Could you repeat the
15 question?
16 BY MR. GIBBONS:
17 Q. Sure.
18 Would you agree that cumulative exposure
19 to a chemical by itself does not provide full
20 insight into the risk associated with that exposure?
21 MR. TELAN: Same objection.
22 THE WITNESS: No.
23 BY MR. GIBBONS:
24 Q. So you believe that cumulative exposure to
25 a chemical does provide full insight into the risks

1 associated with exposure?
2 MR. TELAN: Object to the form.
3 THE WITNESS: I need more information.
4 Are you asking if -- are you suggesting that
5 cumulative exposure is not the full picture or data
6 about the exposure?
7 BY MR. GIBBONS:
8 Q. Correct.
9 A. If there was more data or exposure
10 information outside of cumulative exposure, then
11 yes, I would want to know all of the available data
12 in order to make a conclusion.
13 Sometimes that's not available.
14 Q. Regardless of whether or not it's
15 available, you sometimes need more information
16 before you can have a full understanding of the
17 risks associated with exposure, correct?
18 A. I'm struggling with the question because
19 "cumulative" to me means all exposure. And that
20 sounds sufficient.
21 Q. Okay. So, for instance, total dosage,
22 irrespective of time, would be enough to accurately
23 assess the risks of a certain exposure?
24 MR. TELAN: Object to the form.
25 THE WITNESS: It would depend on the

1 particulars.
2 BY MR. GIBBONS:
3 Q. Would you agree that a high dose in a
4 single moment in time would have a different risk
5 profile than a lower level of exposure over a
6 prolonged period of time?
7 MR. TELAN: Object to the form of the
8 question.
9 THE WITNESS: It's possible they could
10 have the same risk, period, or the same odds ratio,
11 the same increased risk of disease. I'm not sure
12 what you mean by "risk profile," though.
13 BY MR. GIBBONS:
14 Q. Okay. Fair enough.
15 You would agree, though, that they could
16 have the same level of risk, but that they don't
17 necessarily have the same level of risk, correct?
18 MR. TELAN: Object to the form of the
19 question.
20 THE WITNESS: Could the dose and the
21 duration be titrated to a point where their risk was
22 equal? Yes, I suppose that's possible.
23 BY MR. GIBBONS:
24 Q. Okay. Would you agree that intensity and
25 duration of exposure to a chemical are important in

1 determining the risk associated with that exposure?
2 MR. TELAN: Object to the form of the
3 question.
4 THE WITNESS: If by "intensity" you mean
5 concentration.
6 BY MR. GIBBONS:
7 Q. Right.
8 A. Concentration is a relevant consideration.
9 Duration is also relevant.
10 Q. Okay. Would you agree that frequency of
11 exposure to a chemical is important for determining
12 the risks associated with the exposure?
13 A. It can be, yes.
14 Q. Okay. Are you aware that the studies that
15 were conducted by the ATSDR regarding exposure to
16 contaminated water at Camp Lejeune failed to account
17 for Marines' deployment off base?
18 MR. TELAN: Object to the form.
19 THE WITNESS: I'm generally aware that the
20 studies around Camp Lejeune, including studies
21 outside of ATSDR, that I couldn't turn to the
22 specific page within ATSDR, had variables that
23 couldn't be accounted for, but generally studied,
24 you know, populations, to the best of their ability.
25 / /

1 BY MR. GIBBONS:
2 Q. Okay. Were you aware that no
3 toxicologists were involved in peer reviewing the
4 2017 ATSDR assessment of the evidence?
5 A. I'm not aware of who peer reviewed it.
6 Q. Okay. Were you aware that the purpose of
7 the 2017 ATSDR assessment was to help the Department
8 of Veterans Affairs develop a presumption list to
9 compensate individuals that may have been exposed at
10 Camp Lejeune?
11 MR. TELAN: Object to the form.
12 THE WITNESS: Are you referring to a
13 presumptive service connection for Parkinson's and
14 Camp Lejeune?
15 BY MR. GIBBONS:
16 Q. (Nods head.)
17 A. I'm aware that, generally, it was related,
18 yes.
19 Q. Were you aware that the specific purpose
20 of the ATSDR report was to build a presumption list
21 for the Department of Veterans Affairs?
22 MR. TELAN: Object to the form.
23 THE WITNESS: I'm not sure.
24 BY MR. GIBBONS:
25 Q. Okay. Were you aware that Dr. Bove

1 testified that the 2017 assessment was specifically
2 designed to add diseases to the VA's presumption
3 list?
4 MR. TELAN: Object to the form.
5 THE WITNESS: No.
6 BY MR. GIBBONS:
7 Q. Okay. Were you aware that the community
8 action panel and several senators had pushed the VA
9 to add more diseases to their presumption list
10 because they wanted more diseases?
11 MR. TELAN: Object to the form.
12 THE WITNESS: No.
13 BY MR. GIBBONS:
14 Q. The 2017 ATSDR assessment didn't use
15 significance testing to assess the evidence for
16 causality, correct?
17 A. I couldn't say for the totality of the
18 report.
19 Q. Okay.
20 A. I see they acknowledged, as we've spoken
21 about, that when looking at mortality, there's a
22 small number of deaths due to Parkinson's disease.
23 We can talk about that later if you like.
24 Meaning we don't expect people to die from
25 Parkinson disease and that can limit significance

1 testing when we're looking at mortality.
2 Q. If you'll turn to page 8 of the ATSDR
3 assessment.
4 A. Which page?
5 Q. Page 8, please.
6 A. Yes.
7 Q. Last paragraph on page 8 reads (as read):
8 In our assessment, we did not
9 use confidence intervals to
10 determine whether a finding was
11 statistically significant, nor did
12 we use significance testing to
13 assess the evidence for causality.
14 Did I read that correctly?
15 A. Yes. And, of course, they go on to
16 explain that's because there's limitations to such
17 testing.
18 Q. Okay.
19 A. And that, quoting from page 8 (as read):
20 The finding that does not
21 achieve statistical significance
22 nonetheless can provide important
23 evidence for a causal association.
24 Q. Okay. But instead, the ATSDR looked at
25 the ratio of upper end of the confidence interval to

1 the lower end of the confidence interval, correct?
2 MR. TELAN: Object to the form.
3 THE WITNESS: Some studies in this work
4 have used confidence interval ratios.
5 BY MR. GIBBONS:
6 Q. Mm-hmm.
7 A. Is that what you're referencing?
8 Q. I'm referencing the paragraph above. It
9 says (as read):
10 In the disease-specific
11 tables, 95 percent confidence
12 intervals were provided in order to
13 solely indicate the level of
14 precision or uncertainty in the
15 effect estimates. An effect
16 estimate, e.g., risk ratio, odds
17 ratio or standardized mortality
18 ratio, was considered to have good
19 precision or less uncertainty if
20 the ratio of the upper limit to the
21 lower limit of its 95 percent
22 confidence interval was less than
23 or equal to two.
24 Did I read that correctly?
25 A. Yes, you did.

1 Q. Okay. Have you ever used a confidence
2 interval ratio in any of your work?
3 A. My personal research is not epidemiologic;
4 it's clinical research. So I'm well aware of it and
5 familiar with it from my reading of the literature,
6 but it's not germane to the type of research that I
7 personally perform.
8 Q. Okay. Are you aware of whether it's
9 generally acceptable in epidemiology to use a
10 confidence interval ratio?
11 MR. TELAN: Object to the form.
12 THE WITNESS: I would defer to an
13 epidemiologist.
14 BY MR. GIBBONS:
15 Q. Okay.
16 A. Would you mind if we revisited page 99 for
17 a minute?
18 Q. Sure. Just a few more questions and then
19 we'll turn back.
20 A. Okay. Thank you.
21 Q. The assessment stated that a CIR of less
22 than or equal to two indicated good precision,
23 correct?
24 That was the last sentence of the second
25 paragraph -- second-to-last paragraph, right?

1 A. Correct.
2 Q. Okay. There's no authority cited to
3 support that assertion, correct?
4 A. There's no citation in that paragraph.
5 Q. Okay. Have you ever used -- as you said,
6 you don't typically use confidence interval ratios,
7 correct?
8 A. Correct.
9 Q. You've never used that parameter before at
10 your work?
11 A. Correct.
12 Q. Okay. In your own report on page 55, you
13 wrote, midway through the first paragraph (as read):
14 A CIR of less than or equal to
15 three is determined the highest
16 level of precision.
17 Correct?
18 A. Correct. And I'm summarizing Bove and
19 colleagues' interpretation or opinion on this as
20 they published in the paper.
21 Q. Okay. So that was based on your reliance
22 on the literature review, not from your own use of
23 CIRs in your work, correct?
24 A. Correct.
25 Q. Okay. So there's no specific source or --

1 sorry, so there's no specific source for that
2 statement other than the general understanding that
3 you gleaned from the ATSDR reports?
4 A. Are you referring to my statement on
5 page 55?
6 Q. Correct.
7 A. Disagree.
8 The source for summarizing that a CIR less
9 than or equal to three determine the highest level
10 of precision, and other reasons why the authors used
11 the confidence interval ratio was -- the source was
12 the authors of the paper that I cited here, Bove, et
13 al.
14 Q. And you are deferring to them because they
15 are -- they are epidemiologists?
16 A. Yes. And I'm explaining using their
17 rationale, how they analyzed, interpreted and
18 presented their data in this paper.
19 Q. Okay. And real quick, going back to
20 page 9 in the ATSDR report (as read):
21 The assessment stated that a
22 risk result at or above 1.10 was
23 considered elevated, and anything
24 below that was considered null.
25 Correct? That's the bottom of the first

1 paragraph of page 9.
2 A. I would correct what you said by
3 clarifying, a value less than or equal to 1.1 was
4 considered, quote, near the null value, end quote,
5 but not null.
6 Q. Okay. And, again, have you ever used
7 these parameters in your own work?
8 A. Which parameters?
9 Q. The -- is it hazard ratio of 1.1 or
10 higher?
11 A. No. And, again, for the same reason.
12 Q. Okay. Sorry, we will go back to page 99,
13 but first go to page 6 of the ATSDR report, please.
14 In the assessment, the ATSDR considered
15 what it described as epidemiological studies
16 considered to be of high utility, correct? That's
17 in page 6, the fifth paragraph from the bottom.
18 Just under point 2.
19 A. I'm with you now.
20 Q. It says (as read):
21 Sufficient evidence from human
22 studies can be provided by a
23 meta-analysis and/or by several
24 studies considered to have high
25 utility.

1 Correct?
2 A. Correct.
3 Q. The ATSDR never defined the term "high
4 utility," correct?
5 A. I don't know.
6 Q. Okay. What does the phrase "high utility
7 epidemiologic study" mean to you?
8 MR. TELAN: Object to the form.
9 THE WITNESS: I don't see the phrase "high
10 utility epidemiologic study" in the ATSDR report. I
11 simply see human studies considered to have high
12 utility, and I'm paraphrasing.
13 BY MR. GIBBONS:
14 Q. Okay.
15 A. To closely answer your question, I
16 interpret the term "high utility" to mean high
17 usefulness or relevance.
18 Q. Okay. And I'll just point out on page 7,
19 in paragraph 2 and 3, they did specify epidemiologic
20 study of high utility. I think it was just a typo
21 on page 6.
22 A. I see. Thank you.
23 Q. Using that same definition of high utility
24 that you just gave me, are those the same parameters
25 that you use to define studies for your reliance

1 materials in your report?
2 MR. TELAN: Object to the form.
3 THE WITNESS: Again, I considered all
4 studies, but studies that are of higher utility may
5 be more informative, may be more useful, may be more
6 directly applicable to a particular case. But I
7 consider the totality of the evidence.
8 BY MR. GIBBONS:
9 Q. Okay. And I believe you wanted to go back
10 to page 99 of the report.
11 A. Thank you.
12 Q. Was there something that you wish to
13 correct about your testimony?
14 A. I wanted to add an important part of the
15 paragraph that wasn't mentioned, which is in the
16 last paragraph (as read):
17 The ATSDR notes that even
18 though in 2017, there was more
19 limited epidemiologic evidence,
20 they, quote, say, however, this may
21 change if a metabolite of TCE that
22 is common to PCE is found to be the
23 agent causing damage to the
24 dopaminergic neurons.
25 And I would call your attention to

1 pages 45 through 47 of my report where I discuss the
2 principles that I mentioned earlier about TaClo
3 being a common metabolite for PCE and TCE and, of
4 course, their biologic plausibility and relevance in
5 that specific enzyme that catalyzes their conversion
6 is found in the substantia nigra, causing the same
7 loss of dopaminergic neurons and with analogy to
8 MPPE plus.
9 Q. You also referenced other literature that
10 Dr. Bove authored in your report, correct?
11 A. Correct.
12 Q. And you relied on Bove's 2024 study titled
13 "Evaluation of Mortality Among Marines, Navy
14 Personnel and Civilian Workers Exposed to
15 Contaminated Drinking Water at USMC Base
16 Camp Lejeune, a cohort study," correct?
17 A. Correct.
18 (Whereupon, Deposition Exhibit 8 was
19 marked for identification.)
20 BY MR. GIBBONS:
21 Q. Dr. Andruska, I'm marking that study as
22 Exhibit 8.
23 This study compared the Camp Lejeune
24 cohort to the Camp Pendleton cohort, correct?
25 A. Correct.

1 Q. The exposures for those at Camp Lejeune
2 were not directly measured, correct?
3 MR. TELAN: Object to the form.
4 THE WITNESS: Correct. I believe they
5 included people who were stationed at Camp Lejeune.
6 BY MR. GIBBONS:
7 Q. Okay. The exposures were assumed based on
8 the assignment and employment of tertials of
9 duration of assignment to/employment at
10 Camp Lejeune, correct?
11 A. Where are you reading?
12 Q. Give me one moment.
13 Okay. Sorry about that. That's on page 5
14 on the second paragraph from the bottom.
15 It says (as read):
16 Results of duration analysis
17 should be interpreted with caution
18 because monthly contamination
19 levels fluctuated considerably
20 between 1972 and 1985. Duration
21 stationed or employed at
22 Camp Lejeune was categorized into
23 tertials of the quarterly data
24 after removal of the Camp Pendleton
25 reference group.

1 Did I read that correctly?
2 A. Yes.
3 Q. Okay. Are you aware that Camp Pendleton
4 is a super-fund site?
5 A. Not specifically. I'm aware that
6 Camp Pendleton didn't have the TCE and PCE exposures
7 relevant to Camp Lejeune and was, therefore, an
8 appropriate control group.
9 Q. Are you aware of what chemicals are
10 present at Camp Pendleton?
11 A. Not specifically.
12 Q. Would it make it difficult to compare the
13 populations of Camp Lejeune and Camp Pendleton if
14 they were exposed to different, but nonetheless some
15 toxic chemicals?
16 MR. TELAN: Object to the form of the
17 question.
18 THE WITNESS: No.
19 BY MR. GIBBONS:
20 Q. One of the limitations of this study is
21 that it has a lack of direct exposure data for both
22 Camp Lejeune and Camp Pendleton --
23 (Reporter clarification.)
24 BY MR. GIBBONS:
25 Q. One of the limitations of this study is

1 that -- the lack of direct exposure data for both
2 Camp Lejeune and Camp Pendleton cohorts, correct?
3 MR. TELAN: Object to the form of the
4 question.
5 THE WITNESS: Do you --
6 BY MR. GIBBONS:
7 Q. Limitations discussion begins on page 11.
8 A. Do you mean that it didn't include the
9 water contaminant concentration levels in the same
10 way that, for example, Dr. Reynolds modeled it for
11 Mrs. Rothschild?
12 Q. Correct.
13 It doesn't have individualized levels of
14 exposure for these study subjects, correct?
15 A. Correct.
16 Q. This study only assessed mortality,
17 correct?
18 A. Correct.
19 Q. Okay. And this study doesn't consider
20 other risk factors for health outcomes, correct?
21 A. Do you mind rephrasing that?
22 Q. Sure.
23 Dr. Bove, the author of this study, didn't
24 have data on other risks factors for health outcomes
25 in this report, correct?

1 I'll direct your attention to page 11.
2 The left column, third paragraph down (as read):
3 Study weakness was the lack of
4 data on risk factors, such as
5 smoking, alcohol consumption and
6 occupational history, before and
7 after active duty service or
8 employment at Camp Lejeune or
9 Camp Pendleton.
10 Did I read that correctly?
11 A. I see that. It's important to understand
12 that Bove is looking at -- I can't even count how
13 many different outcomes, including cancers, chronic
14 kidney disease, COPD, for example, and not just
15 Parkinson's.
16 So if we were to think about Parkinson's
17 and relevance to this case, smoking, for example, is
18 protective in Parkinson's. Alcohol consumption is
19 not considered a particular risk factor. And so
20 Bove is really talking about this entire breadth of
21 health outcomes that he measured. And -- and not
22 just Parkinson's.
23 Q. Okay. On page 11, table 5, at the
24 beginning shows the total number of Parkinson's
25 disease across two cohorts, correct?

1 A. Correct.
2 Q. Camp Lejeune is listed as 30,
3 Camp Pendleton is 31, correct?
4 A. Correct.
5 Q. Is that a statistically significant
6 difference between Camp Lejeune and Camp Pendleton?
7 MR. TELAN: Object to the form of the
8 question.
9 THE WITNESS: I'm not performing a
10 statistical analysis. I don' think the authors did
11 either, but they're at least similar numbers.
12 BY MR. GIBBONS:
13 Q. Okay. Given the presence of TCE and PCE
14 at Camp Lejeune, but not at Camp Pendleton, wouldn't
15 you expect Camp Lejeune's number of Parkinson's
16 disease to be higher?
17 MR. TELAN: Object to the form of the
18 question.
19 THE WITNESS: Not necessarily. One reason
20 for that could be the latency of time between the
21 exposure and the study. Of course, we know
22 Parkinson's disease can have a many-decade latency.
23 But really to answer the question of risk, we look
24 to studies that were designed in a way to study risk
25 and not mortality, as this study was.

1 BY MR. GIBBONS:
2 Q. Okay. If you'll turn to page 6, please.
3 The standardized mortality rates for
4 Camp Lejeune and Camp Pendleton were not
5 significantly -- statistically significantly
6 different from the general U.S. population, correct?
7 A. The risk ratio between Camp Lejeune
8 compared to Camp Pendleton is 2.0.
9 Q. Correct.
10 I should have specified, this is for Navy
11 and Marines rather than for civilians, correct?
12 A. Table 2 is for --
13 Q. Yes. It starts at the top.
14 A. -- Navy and Marines. Yes, I agree.
15 Q. And then moving over onto page 7, the
16 standardized mortality ratios, comparing civilian
17 employees for the underlying cause of death, it's
18 the table 3.
19 A. I see. And the risk ratio is 1.15 for
20 Camp Lejeune.
21 Q. Correct.
22 And again, that's not statistically
23 significant, correct?
24 MR. TELAN: Object to form of the
25 question.

1 THE WITNESS: The authors don't calculate
2 statistical significance.
3 BY MR. GIBBONS:
4 Q. Okay.
5 A. But as we discussed, a ratio above 1.1 is
6 meaningful.
7 Q. Okay. Bove published two other studies
8 regarding the mortality of personnel at
9 Camp Lejeune, correct?
10 2014, they were referred to 2014A and
11 2014B, correct?
12 A. Correct.
13 Q. Did you independently review this study?
14 A. 2014A and B?
15 Q. The 2023 study -- sorry, 2024 study that
16 we were just discussing.
17 A. Yes. And I cited to it on page 55 of my
18 report.
19 Q. Okay.
20 A. In general, as we've discussed before,
21 because we generally consider Parkinson's not to
22 cause death, we say people die with Parkinson's and
23 not because of Parkinson's, mortality studies may be
24 useful. We might identify certain subpopulations of
25 people with Parkinson's that have a shortened

1 lifespan. But it's not, again, accepted that people
2 generally die because of Parkinson's.
3 So I take this mortality data in that
4 context.
5 Q. Okay. So you reviewed it based on your
6 experience and expertise with Parkinson's rather
7 than as an epidemiologist then, correct?
8 A. Correct.
9 Q. Okay.
10 (Whereupon, Deposition Exhibit 9 was
11 marked for identification.)
12 BY MR. GIBBONS:
13 Q. Dr. Andruska, I handed you Exhibit 9,
14 which is the 2014A study, the mortality study of
15 Marines and Navy service members at Camp Lejeune.
16 You also reviewed this report for your --
17 this study for your report, correct?
18 A. Yes.
19 Q. This had the same civilian -- or same
20 military cohort population as the Bove 2024 study,
21 correct?
22 A. I'd have to double-check specifically.
23 Q. Okay. Just like the 2024 report, this
24 doesn't evaluate direct chemical exposure in the
25 individuals, correct?

1 A. Correct.
2 Q. Okay. And as you were just explaining,
3 because this study only assesses mortality, it may
4 not perfectly reflect the total number of
5 Parkinson's cases, correct?
6 A. Yes. Or the risk of Parkinson's.
7 Q. Okay. And these studies also didn't --
8 just like the 2024 study, this study doesn't account
9 for smoking, correct?
10 A. Again, I'll double-check.
11 Do you have a reference?
12 Q. Give me one second.
13 Page 13. (As read):
14 Another limitation was the
15 lack of information on smoking and
16 other risk factors. Such risk
17 factors, if associated with
18 exposure, could be confounders,
19 biasing hazard ratios in either
20 direction and distorting
21 exposure-response relationships.
22 Correct?
23 A. Yes, in either direction.
24 Q. Okay. To your knowledge, did this study
25 account for genetic factors?

1 A. No.
2 Q. Okay. Head injuries?
3 MR. TELAN: Object to the form.
4 THE WITNESS: I'm not sure.
5 BY MR. GIBBONS:
6 Q. Okay.
7 A. I don't think so.
8 I might add, if you don't mind, to your
9 prior question about measured levels, Bove 2014 does
10 discuss on page 3 about the author's exposure
11 assessment and using the ATSDR's historical
12 reconstruction of the contamination using
13 groundwater modeling.
14 Q. Correct.
15 A. Okay.
16 Q. But those are not individualized exposure
17 calculations, correct?
18 A. Correct.
19 Q. Okay.
20 (Whereupon, Deposition Exhibit 10 was
21 marked for identification.)
22 BY MR. GIBBONS:
23 Q. I've just handed you Exhibit 10, which is
24 Bove's 2014B, the mortality study of civilians in
25 Camp Lejeune, correct?

1 A. Correct.
2 Q. Did you independently review this study?
3 A. Yes.
4 Q. Okay. And your -- your review was based
5 on your expertise as an Parkinson's expert, correct,
6 not as an epidemiologist?
7 A. Correct.
8 Q. Okay. And just like Bove 2024 and Bove
9 2014A, this study didn't evaluate direct chemical
10 exposure to individuals, correct?
11 A. Yes.
12 Q. And it also only assessed mortality,
13 right?
14 A. Right.
15 Q. Okay. Just like 2014A, it didn't consider
16 or control for smoking? It's on page 12.
17 A. Agreed.
18 Q. Okay. To your knowledge, it didn't
19 account for genetic factors, correct?
20 A. Correct.
21 Q. It also didn't consider head injuries,
22 correct?
23 MR. TELAN: Object to the form.
24 THE WITNESS: Correct.
25 / /

1 BY MR. GIBBONS:
2 Q. Did you independently review this study as
3 well?
4 A. Bove 2014B?
5 Q. Correct.
6 A. Yes.
7 Q. Again, this was analyzed as a Parkinson's
8 expert, not as an epidemiologist, correct?
9 A. Correct.
10 Q. Okay. In 2014B, Dr. Bove acknowledged
11 that a limitation of the study -- correction -- the
12 study had errors and base assignment likely
13 occurred?
14 MR. TELAN: Where are you at?
15 MR. GIBBONS: On page 11. The third
16 paragraph from the bottom.
17 BY MR. GIBBONS:
18 Q. (As read):
19 Another serious limitation of
20 this study was exposure
21 misclassification bias. There were
22 several sources of exposure
23 misclassification, for example, due
24 to lack of information on workplace
25 locations. We assumed that all the

1 Camp Lejeune workers were located
2 or spent considerable time during
3 the workday at mainside area of the
4 base served by the Hadnot Point
5 treatment plant. Although this
6 assumption is true for most
7 workers, undoubtedly, some did not
8 work in the main site area.
9 Did I read that correctly?
10 A. You did.
11 Q. Okay. Are you aware of how many water
12 systems there were at Camp Lejeune between 1953 and
13 1987?
14 A. I'm aware generally the relevant ones in
15 this case are Hadnot Point and Tarawa Terrace.
16 Q. Are you also aware of the Holcomb
17 Boulevard Water Treatment System?
18 A. It's familiar, now that you say it.
19 Q. Are you aware of any other water systems
20 on Camp Lejeune?
21 A. I couldn't name them today.
22 Q. Okay. Are you aware of which water
23 systems serviced Ms. Rothschild's workplace?
24 A. Tarawa Terrace.
25 Q. Okay. And that is not the same as the

1 Hadnot Point Water Treatment System, correct?
2 A. Correct.
3 Q. This study didn't account for any
4 individuals working at Tarawa Terrace, correct?
5 A. This study included all persons who lived
6 on base. And I think it's an important distinction,
7 because it would have included a mix of persons,
8 including those at Tarawa Terrace. Bove and
9 colleagues, discusses that Hadnot Point is an
10 assumption, probably for several reasons. But he
11 did not exclude people for inclusion in this trial
12 that lived or worked in Tarawa Terrace.
13 Q. Okay. Wouldn't assuming that everyone in
14 this study was at Hadnot Point also misclassify
15 Ms. Rothschild?
16 MR. TELAN: Object to the form.
17 THE WITNESS: This study is a mortality
18 study, and it's looking at the likelihood that
19 people with Parkinson's and other diseases die.
20 Insofar as Bove models exposure levels, then Hadnot
21 Point is relevant or not relevant to Tarawa Terrace.
22 But I don't think it changes my overall opinions.
23 BY MR. GIBBONS:
24 Q. Okay. Are you aware of what the
25 predominant chemicals that are alleged to have

1 contaminated the Hadnot Point water system are?
2 A. I'm aware that there was TCE largely and
3 also PCE.
4 Q. Do you know which one of those was
5 predominant at Hadnot Point?
6 A. I believe it was TCE.
7 Q. Okay. Is that the same as Tarawa Terrace?
8 A. Hadnot Point?
9 Q. The TCE being the predominant contaminant?
10 A. No. In Tarawa Terrace, PCE was the
11 predominant contaminant.
12 Q. Okay. So would assuming that everyone's
13 exposure was based on Hadnot Point have the same
14 effect?
15 A. Can you say that differently?
16 MR. TELAN: Object to the form.
17 BY MR. GIBBONS:
18 Q. Since Hadnot Point was primarily
19 contaminated with TCE and Tarawa Terrace was
20 primarily contaminated with PCE, wouldn't it be a
21 large misclassification to extrapolate everyone
22 being exposed at Hadnot Point?
23 MR. TELAN: Object to the form.
24 THE WITNESS: Again, if we're only
25 applying what you've mentioned to exposure levels of

1 PCE and TCE specifically. However, this is a
2 mortality study. We're not -- I'm not opining on
3 Mrs. Rothschild's mortality, so hypothetically, if
4 we were talking about mortality, I would, again,
5 point out that this study included, you know, all
6 people on Camp Lejeune and did not exclude people on
7 Tarawa Terrace. And we're taking this study as one
8 piece of a large puzzle, and we're looking at all of
9 the data about TCE and PCE from 1939 and before to
10 present to -- to make these opinions.
11 BY MR. GIBBONS:
12 Q. Okay. You also relied on several studies
13 authored by Samuel Goldman, correct?
14 A. Correct.
15 MR. GIBBONS: Why don't we take a break
16 for lunch right now before we get into this.
17 THE VIDEOGRAPHER: Going off the record at
18 12:44 p.m.
19 (Whereupon, a lunch recess was taken
20 from 12:44 p.m. to 1:26 p.m.)
21
22
23
24
25

1 THE VIDEOGRAPHER: We are back on the
2 record at 1:26 p.m.
3 BY MR. GIBBONS:
4 Q. Dr. Andruska, before we go back into the
5 questions, is there anything from your previous
6 testimony that you wanted to correct?
7 A. Thank you for asking.
8 No.
9 Q. All right. We'll continue then.
10 Before we broke, I was beginning to ask
11 about other studies that you considered for your
12 report, including several studies by Samuel Goldman,
13 correct?
14 A. Correct.
15 MR. GIBBONS: I'm going to introduce
16 Exhibit 11.
17 (Whereupon, Deposition Exhibit 11 was
18 marked for identification.)
19 BY MR. GIBBONS:
20 Q. Dr. Andruska, this is the Goldman 2012
21 study, Solvent Exposures in Parkinson's Disease Risk
22 in Twins, correct?
23 A. Correct.
24 Q. This sample -- the sample for this study
25 was 198 pairs of twins, correct?

1 A. Correct.
2 Q. And the study subjects were World War II
3 veterans, correct?
4 A. Correct.
5 Q. This study relied on self-reporting from
6 the veterans or their family members, correct?
7 A. Correct.
8 Q. Or other proxies, correct?
9 A. Yes.
10 Q. Okay. Solvent exposure was inferred based
11 on occupational and hobby history, correct?
12 A. Correct.
13 Q. Okay. For deceased and cognitively
14 impaired subjects, proxy informant provided
15 occupational history and hobby history, correct?
16 A. Correct.
17 Q. Okay. The authors didn't have exposure
18 samples for the population, correct?
19 A. Correct.
20 Q. Inferring exposure based on job history
21 and hobby history collected through self- or
22 proxy-reported history could lead to exposure
23 misclassification, correct?
24 MR. TELAN: Object to form.
25 THE WITNESS: It's possible, yes.

1 BY MR. GIBBONS:
2 Q. Okay. Data on head injuries was also
3 provided through interviews, correct?
4 A. I don't recall. I'm happy to look.
5 Q. Okay. It's on page 777. It's the second
6 paragraph in the right column (as read):
7 Additional questionnaires
8 collected data on lifetime history
9 of smoking and head injury.
10 A. I see that, yes.
11 Q. Okay.
12 A. And they defined head injury as (as read):
13 Ever having a head injury that
14 resulted in a loss of consciousness
15 or amnesia.
16 Q. Correct.
17 For deceased and cognitively impaired
18 subjects, proxy informant provided information on
19 the head injuries as well, correct?
20 It's the bottom of that same paragraph on
21 777. It says (as read):
22 Suitable proxy informants were
23 identified for deceased and
24 cognitively impaired subjects
25 scoring above 27 on the education

1 adjusted modified telephone
2 interview for cognitive status.
3 Correct?
4 A. You read it correctly. Your question was
5 about whether proxy informants gave information
6 about head injuries.
7 Q. Okay. If it's not explicitly mentioned
8 there, it would follow that proxies were also used
9 to collect the head injury information based on the
10 questionnaires, correct?
11 A. It's possible. Of course, it would be to
12 the best of their knowledge, and as the authors
13 point out, these proxy and family member histories
14 are, if anything, biased towards the null because
15 they may not have all of the exposure information
16 that the informant themselves would.
17 Q. Okay. Would you agree that twins don't
18 necessarily have identical exposures, particularly
19 in adulthood?
20 A. They may not have exactly the same
21 exposures, of course, but as things go, they would
22 have likely similar exposure experiences.
23 Q. And twins might live in different
24 residential locations that may lead to different
25 environmental exposures, correct?

1 A. Yes.
2 Q. Goldman 2012 didn't talk about other
3 exposure information, correct, such as residential
4 history?
5 A. I don't think so.
6 Q. The authors didn't have residential
7 history for each twin, correct?
8 A. I think they focused on occupational and
9 hobby exposures.
10 Q. Yeah.
11 It's -- I'll represent to you that it does
12 not address residential histories in the study.
13 A. Okay.
14 Q. Okay. Would you agree that TCE, PCE and
15 carbon tetrachloride have been used extensively
16 worldwide?
17 MR. TELAN: Object to the form.
18 THE WITNESS: Extensively is a broad term,
19 but they are relatively common solvents.
20 BY MR. GIBBONS:
21 Q. Go to page 780.
22 A. Going back, relatively common with the
23 caveat that, as I'm sure you know, TCE was banned
24 for production in the U.S. in 2002, and PCE has been
25 banned in 2024. So I'm speaking historically.

1 Q. Okay.
2 A. Which page?
3 Q. 780.
4 A. Thank you.
5 Q. Second column says (as read):
6 TCE, PERC, that is PCE, and
7 carbon tetrachloride have been used
8 extensively worldwide for decades.
9 Did I read that correctly?
10 A. You did.
11 Q. Confidence intervals provide measured
12 precision, correct?
13 MR. TELAN: Object to the form.
14 THE WITNESS: Correct.
15 BY MR. GIBBONS:
16 Q. Narrower confidence interval indicates
17 more precision, correct?
18 MR. TELAN: Object to the form.
19 THE WITNESS: Correct.
20 BY MR. GIBBONS:
21 Q. A wider confidence interval indicates less
22 precision, correct?
23 MR. TELAN: Object to the form.
24 THE WITNESS: Correct.
25 / /

1 BY MR. GIBBONS:
2 Q. In interpreting confidence interval, one
3 should consider whether the underlying population is
4 biased, correct?
5 MR. TELAN: Object to the form.
6 THE WITNESS: I would say we would
7 consider bias and try to eliminate sources of bias
8 in a study like this, but I would differ to an
9 epidemiologist on a more detailed answer than that.
10 BY MR. GIBBONS:
11 Q. Okay. Would you agree that a
12 confidence -- confidence interval is less meaningful
13 where the sample of the population is not
14 representative of the underlying population?
15 MR. TELAN: Object to the form.
16 THE WITNESS: Yes.
17 BY MR. GIBBONS:
18 Q. Okay.
19 A. And that a confidence interval may be
20 skewed or wide when the sample population is small,
21 even if it is a relevant population.
22 Q. Okay. The Goldman 2012 study found a
23 relative risk ratio for TCE to be 6.1 with a
24 95 percent confidence ratio of 1.2 to 33, correct?
25 A. Correct.

1 Q. Okay. Is 1.2 to 33 a wide confidence
2 interval?
3 MR. TELAN: Object to the form.
4 THE WITNESS: I would, again, defer to a
5 statistician or an epidemiologist, but certainly the
6 magnitude of that ratio is --
7 BY MR. GIBBONS:
8 Q. Is high?
9 A. Is high.
10 Q. Okay. Goldman found a relative risk ratio
11 for PCE to be 10.5 with a 95 percent confidence
12 interval of 0.97 to 113, correct?
13 A. Just to make sure we're speaking the right
14 language, you said relative risk ratio?
15 Q. Sorry.
16 A. This is an odds ratio.
17 Q. Odds ratio.
18 A. Correct.
19 Q. Okay. And the Goldman 2012 study found
20 that there was statistical significance for the
21 association of TCE with the risk of Parkinson's
22 disease, correct?
23 A. Can you restate that, just repeat what you
24 said, please?
25 Q. Yeah.

1 The Goldman 2012 study found that TCE was
2 associated with the increased risk of Parkinson's
3 disease, correct?
4 A. Correct.
5 Q. Okay. However, it was only trending
6 towards significance for PCE being associated with
7 Parkinson's disease, correct?
8 MR. TELAN: Object to the form.
9 THE WITNESS: If we're only looking at the
10 P values and we're making the distinction that for
11 PCE, the odds ratio is 10.5 and the P value is 0.053
12 versus 0.050, I would say, again, that it's
13 important to consider -- consider the meaning of a P
14 value, as I explained it before, and not to consider
15 P values or other statistical tests as so binary
16 that we disregard data that doesn't meet a somewhat
17 arbitrary cutoff, but that we take it in full
18 context.
19 BY MR. GIBBONS:
20 Q. Fair enough.
21 But nonetheless, the Goldman study did
22 find that the association between PCE and
23 Parkinson's disease was only trending towards
24 significance, it was not statistically significant,
25 correct?

1 MR. TELAN: Object to the form.
2 THE WITNESS: I believe the authors use
3 that phrasing, trending towards significance, again,
4 to refer to this P value that was three
5 one-thousandths of a point off of 0.05. And so I
6 agree on the language the authors used. However, we
7 have to acknowledge that the odds ratio itself is
8 quite staggering at 10.5.
9 BY MR. GIBBONS:
10 Q. Okay. The confidence interval for PCE was
11 0.97 to 113.
12 That's significantly wider than the TCE
13 confidence interval of 1.2 to 33, correct?
14 MR. TELAN: Object to the form.
15 THE WITNESS: It's wider. Statistically
16 is a loaded term in science, and, again, I would
17 defer to an epidemiologist.
18 BY MR. GIBBONS:
19 Q. And that would indicate that there was
20 less precision with results for PCE, correct?
21 A. It could, yes. And as we've discussed,
22 this is a small sample size, which could account for
23 the wide intervals and has been addressed in the
24 article.
25 Q. Okay. Goldman attempted to consider only

1 PCE exposure with this data, correct?
2 The sample included ten cases exposed to
3 TCE and five to PCE, correct?
4 A. I'll just double-check those numbers.
5 What page are you on?
6 MR. TELAN: Are you looking at table 3?
7 MR. GIBBONS: I believe so. Hold on.
8 MR. TELAN: I just don't remember what
9 your question was. I thought you said five and
10 three, but maybe I misheard you.
11 BY MR. GIBBONS:
12 Q. Yeah. Sorry. Not -- sorry.
13 It's on table 3, where Goldman separates
14 out PERC, or PCE, from TCE, which is also separated
15 out from TCE or PCE, correct?
16 A. Correct.
17 Q. Okay. And there's different P values
18 associated with each one, correct?
19 A. Correct.
20 Q. As you already indicated, there was a
21 small sample size limitation in this study, correct?
22 A. Correct.
23 Q. Which I believe Dr. Goldman addressed on
24 page 781 at the bottom (as read):
25 The major limitations of the

1 study are its sample size, which
2 yielded imprecise risk estimates
3 and exposure inferences based on a
4 retrospective recall, a virtually
5 unavoidable limitation for disease,
6 such as Parkinson's disease, in
7 which relevant exposures may occur
8 decades before clinical disease is
9 apparent.
10 Did I read that correctly?
11 A. Yes, you did.
12 Q. Okay. Another weakness of the study was
13 that the proxy informants were a large proportion of
14 the subjects, much greater portion among case
15 subjects, correct?
16 MR. TELAN: Object to form.
17 BY MR. GIBBONS:
18 Q. It's further down in the same paragraph.
19 I'll read. It says (as read):
20 However -- sorry. One second. Okay.
21 (As read):
22 Another weakness is reliance
23 on proxy information for a large
24 proportion of the subjects with a
25 much greater proportion among case

1 subjects.
2 Did I read that correctly?
3 A. You did. And, of course, I lost my place.
4 But I just wanted to point out that the proxy
5 estimates would underreport or underrepresent the
6 actual exposures because of the inherent bias in
7 using proxy.
8 Q. Would you agree that a proxy might be
9 biased towards overreporting?
10 MR. TELAN: Object to the form.
11 THE WITNESS: One moment, please.
12 Just earlier in the same paragraph (as
13 read):
14 However, because raters were
15 unaware of case status exposure and
16 misclassification would be toward
17 the null, reducing the likelihood
18 of finding a significant
19 association and reliance on proxy
20 information was addressed by
21 relying on job tasks rather than
22 relying exclusively on the
23 self-reported chemical exposures.
24 So inherent strengths and weaknesses, some
25 biasing towards the null, some underestimating the

1 magnitude of risk.
2 BY MR. GIBBONS:
3 Q. Okay. There's difficultly isolating
4 specific effects of single agents because many of
5 the respondents worked in settings that involve
6 exposure to multiple agents, correct?
7 A. In some cases. And we can see in table 3
8 that in addition to PERC alone and TCE alone,
9 there's also some calculations for any of six
10 solvents and any of four solvents, excluding TCE and
11 PERC. All of these were considered.
12 Q. In the ATSDR's assessment of the evidence
13 for Camp Lejeune, the Goldman 2012 study was
14 included in the review, correct?
15 A. I believe so, but happy to look at the
16 report.
17 Is there another question coming that I
18 might want to reference the report for?
19 Q. I don't think you necessarily need to
20 reference it. I believe we covered it earlier. It
21 was one of the cited cases.
22 But after using the Goldman 2012 study,
23 ATSDR concluded that evidence for TCE and PCE did
24 not meet the sufficient standard, correct?
25 A. We're talking about --

1 MR. TELAN: Object to the form.
2 THE WITNESS: Are you referencing page 99
3 of the ATSDR 2017 report --
4 BY MR. GIBBONS:
5 Q. Correct.
6 A. -- that TCE met clinical equipoise, but at
7 the time, evidence was more limited for PCE?
8 Q. Correct.
9 So it found that TCE met the equipoise
10 standard, correct?
11 A. Correct.
12 Q. Okay. And after reviewing the Goldman
13 2012, ATSDR concluded that evidence for PCE was
14 below equipoise, correct?
15 A. At the --
16 MR. TELAN: Form objection.
17 THE WITNESS: At the time, yes. And
18 acknowledging that should a common metabolite be
19 found, that this opinion would be different.
20 BY MR. GIBBONS:
21 Q. Okay. Is that the -- is the discovery of
22 that metabolite the main reason that you chose to
23 rely on the Goldman 2012 even for conclusions about
24 PCE?
25 MR. TELAN: Object to the form.

1 THE WITNESS: I relied on Goldman 2012
2 because it was a well-designed twin study with
3 relevant exposures, and I considered all of the
4 literature, as we've discussed.
5 BY MR. GIBBONS:
6 Q. Okay. If you'll go to table 1 on
7 page 778.
8 Down below at the bottom of table 1, there
9 are statistically significant differences between
10 the PD-affected twin and the unaffected twin,
11 correct?
12 One of which is cigarette smoking, right?
13 A. I want to make sure I'm following you.
14 You're looking at the bottom window of the table?
15 Q. Correct.
16 A. And we're focused on the "ever regular
17 cigarette smoking"?
18 Q. Correct.
19 And it's lower for the PD-affected twin,
20 correct?
21 54 percent versus 62 percent for the PD
22 unaffected twin?
23 A. Yes. A lower percentage of twins in the
24 study with PD smoked compared to twins without
25 Parkinson's.

1 Q. Okay. And there's a significantly higher
2 number of PD-affected twins that have head injuries
3 than the unaffected twins, correct?
4 A. First, do you mind if we go back to your
5 question about smoking?
6 Q. Sure.
7 A. Was it also a question about a
8 significantly lowered number of PD-affected twins
9 that smoked compared to PD-unaffected twins that
10 smoked?
11 Q. Correct.
12 A. My answer acknowledged the differences in
13 the absolute percentage. I don't think we discussed
14 that the P value is 0.3. So I wouldn't necessarily
15 use the word "significant" to describe it.
16 Q. Okay. Regardless of significance, there
17 is a lower percentage of smokers among the
18 Parkinson's affected twin versus the unaffected
19 twin, correct?
20 A. Apparently, yes.
21 Q. Okay. Moving back to head injury, under
22 "ever have" -- "ever head injury," the PD-affected
23 twins had a head injury 24 percent of the time,
24 whereas, the PD-unaffected twins only reported ever
25 having a head injury 9 percent of the time; is that

1 correct?
2 A. Correct.
3 Q. Traumatic head injury is a common risk
4 factor for Parkinson's disease, correct?
5 A. That's a nuanced topic of discussion. And
6 there's mixed data, as I'm sure you know. When we
7 talk about head injury and Parkinson's, of course,
8 we're not talking about all comers of head injury.
9 There's better data for head injury with loss or
10 consciousness and ICU admissions, there is better
11 data for persons with chronic traumatic
12 encephalopathy and Parkinsonism.
13 Now, these authors did clarify, as I
14 pointed out earlier, that they defined head injury
15 as a head injury that resulted in a loss of
16 consciousness or amnesia. But I just want to
17 clarify that all head injury is not a risk factor.
18 Q. Okay. Severe head injuries that result in
19 loss of consciousness are acknowledged to be a risk
20 factor for Parkinson's disease, correct?
21 A. Again, sometimes this has been studied or
22 used as inclusion for head injury with ICU admission
23 or hospital admission. And repeated head injuries
24 are more of a risk factor.
25 So it's -- it's a complex milieu and

1 spectrum of head injury, some of which are not
2 associated with Parkinson's and some of which may be
3 in more recent emerging data.
4 Q. So you -- strike that.
5 Do you think there is sufficient evidence
6 to show a link between certain types of head
7 injuries and Parkinson's disease?
8 A. Yes.
9 Q. Okay. You also relied on a -- sorry.
10 You reviewed this study as an expert on
11 Parkinson's disease, correct?
12 A. Correct.
13 Q. Not as an epidemiologist?
14 A. Correct.
15 Q. Okay. You also relied on Samuel Goldman's
16 2023 study, correct, "The Risk of Parkinson's
17 Disease Among Service Members at Marine Corps Base
18 Camp Lejeune"?
19 A. Yes.
20 (Whereupon, Deposition Exhibit 12 was
21 marked for identification.)
22 BY MR. GIBBONS:
23 Q. Dr. Andruska, I've just handed you what's
24 going to be Exhibit 12.
25 This is the Goldman 2023 study I just

1 described, correct?
2 A. Correct.
3 Q. Okay. The sample population for the
4 Goldman 2023 study was the same as the population
5 for the Bove 2014 studies, correct?
6 A. Correct.
7 Q. Okay. And the study of cohorts were
8 previously assembled by the U.S. Agency for Toxic
9 Substances in a disease registry, which is known as
10 the ATSDR, as recorded by Bove, et al.?
11 A. Correct.
12 Q. This is also the same population that was
13 used in Bove's 2024 mortality study, correct?
14 A. Correct.
15 Q. Within this cohort, Goldman only
16 identified the analytic cohort that had used the
17 Veterans Health Administration or Medicare health
18 services, correct?
19 A. Correct.
20 Q. Okay. This excludes individuals that had
21 never received VA care or Medicare services,
22 correct?
23 A. Correct.
24 Q. That limits the study population, correct?
25 A. Actually, the author's state the cohort

1 included individuals who used the Veteran's Health
2 Administration or Medicare. That -- that wouldn't
3 exclude people who also used commercial insurance
4 providers.
5 Q. Reading the whole sentence on page 674, it
6 says (as read):
7 The cohorts included 172,120
8 individuals who served at
9 Camp Lejeune and 168,361 who served
10 at Camp Pendleton.
11 Correct?
12 A. Correct.
13 Q. And that's the population that was -- the
14 cohort that was studied by Bove, correct?
15 And it says (as read):
16 Within these, we identified an
17 analytical cohort that included all
18 individuals whoever used the
19 Veterans Health Administration or
20 Medicare Health Service --
21 Healthcare Services.
22 Correct?
23 A. That's correct.
24 Q. That would indicate that it is a further
25 narrowing of the cohort, correct?

1 A. That is a correct statement.
2 Q. Okay. Assuming that it was, in fact, a
3 further narrowing of the cohort, would that be a
4 limitation of the study?
5 MR. TELAN: Object to the form.
6 THE WITNESS: No. Because the reason that
7 scientists would restrict a cohort to subjects who
8 use certain services or had certain database
9 information would be to make their analysis more
10 accurate.
11 BY MR. GIBBONS:
12 Q. Is that your testimony as an expert in
13 epidemiology?
14 MR. TELAN: Object to the form.
15 THE WITNESS: It's my assessment as a
16 scientist.
17 BY MR. GIBBONS:
18 Q. Okay. Either way, the exposures in the
19 2023 study are based solely on whether an individual
20 stationed at Camp Lejeune or Camp Pendleton,
21 correct?
22 A. Correct.
23 Q. Okay. And as we discussed in the Bove
24 cohort studies, there was no data on direct
25 exposures for those individuals, correct?

1 A. Not on an individual basis, correct.
2 Q. Okay. On page 769, in the second column
3 in the second paragraph, Dr. Goldman stated (as
4 read):
5 We cannot be certain that
6 everyone who resided at
7 Camp Lejeune between 1975 and 1985
8 was, in fact, exposed to biologic
9 meaningful levels of contaminants
10 and were unable to account for
11 other environmental exposures that
12 individuals from either camp may
13 have sustained before, during or
14 after military service.
15 Did I read that correctly?
16 A. You read it correctly.
17 Q. Okay.
18 A. Of course.
19 Q. (As read):
20 However, inclusion of
21 unexposed individuals in the
22 Camp Lejeune cohort would tend to
23 bias results toward null.
24 Did I read that correctly?
25 A. You read it correctly, and I was just

1 going to add that, of course, we do have exposure
2 data specific to Mrs. Rothschild as an individual,
3 but this is a -- is a cohort study.
4 Q. Would you agree that the lack of exposure
5 data is a limitation of the Goldman study?
6 MR. TELAN: Object to the form.
7 THE WITNESS: No. Goldman, in fact,
8 comments on -- on median monthly exposures and their
9 magnitude above MCL levels. So I think it's not a
10 limitation.
11 BY MR. GIBBONS:
12 Q. Is it your opinion that exceeding an MCL
13 is evidence of causation?
14 MR. TELAN: Object to the form.
15 THE WITNESS: Not by itself, no.
16 BY MR. GIBBONS:
17 Q. Okay.
18 A. Again, we're taking the totality of animal
19 studies and mechanistic data and population studies,
20 and these are all important pieces of the puzzle.
21 Q. Okay. And Goldman studies the association
22 between TCE and Parkinson's Disease, correct, in
23 this study?
24 A. Correct.
25 Q. Okay. Goldman noted that the water was

1 contaminated with other VOCs, such as PCE, correct?
2 A. That's correct.
3 Q. But the conclusions in this report are
4 limited to the connection between TCE and PD,
5 correct?
6 A. Goldman references TCE, meaning he calls
7 out TCE as an individual contaminant in some places,
8 but it's important to remember that the study is
9 looking at TCE, PCE, and other VOCs together,
10 meaning he's not making an opinion or an assessment
11 only about TCE.
12 Q. On page 679, the second-to-last paragraph
13 just above "conclusions," Goldman stated (as read):
14 Finally, although TCE was the
15 VOC present in Camp Lejeune water
16 supply at highest concentration,
17 the water also contained high
18 levels of PCE, vinyl chloride and
19 Benzene. These other compounds, or
20 mixtures of compounds, could have
21 contributed to the associations we
22 observed.
23 Correct?
24 A. Correct.
25 Q. Okay. Is that what you were referring to?

1 A. Yes. That he didn't separate TCE, that he
2 looked at the water, and the measured levels of
3 these compounds in the water, but made risk
4 assessments.
5 Q. Okay. You also cited Goldman's 2024
6 study, correct?
7 A. Correct.
8 (Whereupon, Deposition Exhibit 13 was
9 marked for identification.)
10 BY MR. GIBBONS:
11 Q. Dr. Andruska, I just handed you
12 Exhibit 13, which is Goldman's 2024 study.
13 "Parkinson's Disease Progression and Exposure to
14 Contaminated Water at Camp Lejeune," correct?
15 A. Correct.
16 Q. This study covered the same population
17 analyzed in the 2023 study, correct?
18 A. Correct.
19 Q. Which was itself also based on the same
20 population studied in the Bove 2024 and 2014
21 studies, correct?
22 A. Correct.
23 Q. Okay. And just like the 2023 study, this
24 one was also limited only to ones that had received
25 healthcare through the VHA or Medicare, correct?

1 A. I would think so. It's about 73,000 and
2 80-some thousand for each population.
3 Q. Okay.
4 A. I get those numbers from Goldman 2023.
5 Q. Okay. And like the other studies we've
6 discussed, Bove and Goldman's, this study also
7 didn't consider individualized exposure estimates,
8 correct?
9 A. Correct.
10 Q. And like the other study, Goldman inferred
11 exposure based on residential location and time,
12 correct?
13 A. Do you mind repeating that?
14 Q. Sure.
15 Goldman inferred exposure based on
16 residential time and location, correct?
17 A. On residential time and Camp Lejeune
18 versus Camp Pendleton location, yes.
19 Q. Okay. It's only for Camp Lejeune,
20 correct?
21 It's under the exposure determination,
22 estimated exposure to VOCs at Camp Lejeune was
23 determined by the ATSDR as previously reported,
24 correct?
25 A. Yes.

1 Q. And Goldman didn't have any other data on
2 other lifetime exposures for the study, correct?
3 A. Of chlorinated solvents?
4 Q. Correct.
5 A. Correct.
6 Q. Okay. This study doesn't consider
7 Camp Pendleton at all, does it?
8 A. Correct. I was mistaken. This is a study
9 about rates of progression and time to complications
10 of Parkinson's.
11 Q. Okay. But this 2024 study didn't observe
12 any earlier age Parkinson's diagnosis in exposed
13 individuals, correct?
14 A. Again, the study was looking at rates of
15 progression and time to complication. Are you
16 referencing a table --
17 Q. On page 1734, which is, I think, the third
18 page under "results."
19 A. Age at PD diagnosis averaged 55.3 years
20 and was similar in exposed and unexposed
21 individuals.
22 Yes, that's correct. And as I've
23 mentioned before, this is a relatively young cohort
24 considering Parkinson's disease and latency, as we
25 discussed earlier, of many decades from exposure to

1 disease comes into play, so we may not expect to see
2 a difference in Parkinson's incidence with this
3 relatively young cohort.
4 Q. Would you agree that chemical cause of
5 Parkinson's disease would decrease the latency
6 period?
7 MR. TELAN: Object to the form.
8 THE WITNESS: No.
9 BY MR. GIBBONS:
10 Q. No?
11 You believe it would increase the risk of
12 developing Parkinson's disease?
13 A. Correct.
14 Q. So it would have no impact on the age of
15 diagnosis?
16 MR. TELAN: Object to the form.
17 THE WITNESS: I'm not aware of evidence to
18 support that. We do have good evidence that the
19 latency period is still quite long for Parkinson's,
20 and this includes people who have toxic exposures.
21 BY MR. GIBBONS:
22 Q. Okay. What is the average age of onset of
23 motor -- motor dysfunction for Parkinson's disease?
24 A. There's a very wide range. But if we look
25 at the literature, the average for so-called regular

1 Parkinson's disease can be around 55 to 60 years
2 old.
3 Q. And how old was Ms. Rothschild when she
4 first began exhibiting symptoms of Parkinson's
5 disease?
6 A. I believe, based on her medical records,
7 and Dr. Perlmutter's documentation, that she was
8 about 51.
9 Q. Okay. That would be earlier than typical,
10 correct?
11 MR. TELAN: Object to the form.
12 THE WITNESS: Earlier than the stated
13 average, the 55 to 60, but not unusual given the
14 wide spectrum. And not considered young onset or
15 early onset Parkinson disease.
16 BY MR. GIBBONS:
17 Q. Okay. You also cited to a study by Liu,
18 et al. titled "Trichloroethylene Ethylene and
19 Parkinson's Disease, a Risk Assessment," correct?
20 A. Correct.
21 Q. Okay.
22 (Whereupon, Deposition Exhibit 14 was
23 marked for identification.)
24 BY MR. GIBBONS:
25 Q. Just to clarify, you evaluated the Goldman

1 2024 study also as an expert in Parkinson's disease,
2 not as an epidemiologist, correct?
3 A. That's correct.
4 Q. Okay. You've been handed what's
5 Exhibit 14, the Liu 2018 study I just mentioned.
6 A. Yes.
7 Q. Sorry, 2017.
8 A. 2017.
9 Q. This study exposed animals via oral
10 gavage, correct?
11 Did I pronounce that correctly?
12 A. Yes.
13 Q. It's on page 6202 under "animals and
14 treatment."
15 (As read):
16 Mice at three months of age
17 received either 400 milligrams per
18 kilogram per day TCE in vehicle,
19 which is olive oil, or an equal
20 volume of vehicle by oral gavage
21 five days a week for eight months.
22 Did I read that correctly?
23 A. Yes, you did.
24 Q. Turning to figure 1 on page 6205, we see a
25 bar graph where the Y axis is measuring thousands of

1 TH plus -- or TH cells, correct?
2 A. The Y axis is measuring total number of
3 cells. We've got TH and Nissl stain represented.
4 Q. All right. For the TH, the cell count
5 appears to be about 11,000 for vehicle,
6 approximately 8,000 for TCE with three months and
7 about 50,000 for TCE with eight months, correct?
8 A. That's correct.
9 Q. That means that the end of eight months of
10 daily exposure to TCE, there was still approximately
11 50 percent the number of TH cells as the vehicle,
12 correct?
13 A. That's correct.
14 Q. Okay. On 6204, you stated (as read):
15 Statistical analysis of the
16 cell accounting indicated a
17 significant loss of TH positive
18 neurons by 32.7 percent at three
19 months and 52.3 percent after
20 eight-month treatment with TCE
21 compared to vehicle.
22 Correct?
23 A. I just want to reference my report.
24 Do you have a page number?
25 MR. TELAN: I think it's 6204.

1 MR. GIBBONS: 6204 of this exhibit.
2 MR. TELAN: Midway down the results
3 column, it looks like.
4 THE WITNESS: I'm with you.
5 BY MR. GIBBONS:
6 Q. Okay.
7 A. Can we take a moment just to define "TH
8 amino reactivity"?
9 Q. Sure.
10 What would that mean, in your opinion?
11 A. I'd need to refer to the article.
12 Q. I believe it's defined on the first page
13 of the exhibit, TH's tyrosine hydroxylase.
14 A. Understood.
15 I just want to make sure we're on the same
16 page about how to interpret TH positivity, and then
17 what it means in this context.
18 We can say at least that tyrosine
19 hydroxylase is an enzyme that the authors may be
20 looking at enzyme activity.
21 I'm happy to go on if you have more
22 questions, and I can come back to this if it's
23 relevant.
24 Q. Okay. And -- sorry, TH positive neuron
25 losses were documented in mice that were receiving

1 400 milligrams per kilogram of TCE five days per
2 week for eight months, correct?
3 A. We might more specifically say these are
4 neurons with TH activity. I just want to be careful
5 about overstepping to say these bar graphs represent
6 cell death or neuron loss.
7 Q. Okay. Well, let's talk about dopamine
8 levels then, also in figure 1 on page 6205. You
9 would agree that there is approximately a 50 percent
10 loss in dopamine from eight months of TCE exposure,
11 correct?
12 A. Correct.
13 Q. And the loss of dopamine is one of the
14 primary driving factors in the development of
15 Parkinson's disease, correct?
16 A. Correct.
17 Q. Do you know how the doses that these mice
18 were given compares to the levels that were detected
19 at Camp Lejeune?
20 A. I'm not sure.
21 Q. Okay.
22 A. It's hard to approximate animal and human
23 levels.
24 Q. Do you recall the total cumulative dose
25 that Dr. Reynolds calculated for Ms. Rothschild?

1 A. I've included it in my report, and I can
2 refer to it. But I believe it was around 10,000
3 micrograms of PCE. And let me just double-check.
4 We would be on page 27. And cumulative
5 consumption, according to Dr. Reynolds' exposure
6 estimates, is between 7,580 and 32,153 for PCP,
7 depending on which consumption rate is used to
8 calculate it.
9 Q. Okay. And just for quick math, 32,000
10 micrograms would be the equivalent of 32 milligrams,
11 correct?
12 A. Yes.
13 Q. Okay.
14 A. 32 grams.
15 Q. 32 grams. Sorry.
16 And these mice were given 400 milligrams
17 per kilogram, correct?
18 A. Yes.
19 Q. Every day for -- sorry, five days a week
20 for eight months, correct?
21 A. Correct.
22 Q. The 32 grams were Ms. Rothschild's total
23 cumulative exposure, correct?
24 A. Correct.
25 Q. Okay.

1 A. Of course, only considering her ingestion
2 and not inhalation or dermal routes.
3 Q. Okay. This study doesn't deal with
4 inhalation or dermal exposure, correct?
5 A. The Liu study?
6 Q. Correct.
7 A. Correct.
8 Q. Okay.
9 A. And just to be clear and add to what I
10 said, in animal studies, we may use higher doses to
11 your earlier point because we're also looking at
12 shorter durations of exposure because we're trying
13 to recapitulate disease in the 12-month lifespan of
14 a rodent, whereas, in humans, as we know, the
15 latency of disease could be decades and we don't
16 have that kind of time in animals to wait.
17 Q. Okay. And on the first page of the Liu
18 study, the -- in the abstract, the last sentence
19 reads (as read):
20 TCE exposure may stimulate the
21 endogenous -- endogenous formation
22 of T-A-C-L-O, TaClo, which is
23 responsible for dopaminergic
24 neurodegeneration. However, even
25 prolonged administration of TCE was

1 insufficient for producing a
2 greater than 50 percent loss of
3 nigral dopamine neurons, indicating
4 that additional comorbid factors
5 would be needed for mimicking the
6 profound loss of dopamine neurons
7 seen in Parkinson's disease.
8 Did I read that correctly?
9 A. You read it correctly.
10 Q. Okay. With respect to the animal studies
11 that you relied on in your report, did any of the
12 studies, including this one, report on the
13 permanence of the effect following cessation of
14 exposure?
15 A. Meaning after cessation of exposure, do
16 the dopaminergic neurons regrow --
17 Q. Correct.
18 A. -- and the disease reverse?
19 Q. Correct.
20 A. I'm not aware of that.
21 Q. Okay. And, again, for every study, you
22 reviewed this as a Parkinson's expert, not as an
23 epidemiologist, correct?
24 A. Correct. And I think what I could also
25 say throughout today as a Parkinson's expert and a

1 scientist.
2 Q. Did you search for any literature on
3 chemical similarities between the differences
4 between TCE and PCE?
5 A. I've certainly read literature about it.
6 It came up in the -- the works that I've included in
7 my reliance list.
8 Q. Okay. Did you search for any literature
9 on the toxicological similarities and differences
10 between TCE and PCE?
11 A. When you say "toxicological similarities,"
12 meaning how they behave, how they cause disease?
13 Q. Do they produce similar toxicological
14 effects to each other?
15 A. Again, that analogy came up in much of the
16 literature that I've read and referenced.
17 Q. Okay. Turning back to your report, on
18 page 49, I believe it is, you wrote that (as read):
19 PCE is very closely related to
20 TCE, having only one additional
21 chlorine atom in place of a
22 hydrogen atom. PCE can readily
23 transform into TCE, and their
24 structural analogy suggests similar
25 toxicologies.

1 Did I read that correctly?
2 A. Yes.
3 Q. So you're contending that the structural
4 similarity of TCE and PCE supports the
5 generalization of risk from TCE to PCE?
6 MR. TELAN: Object to the form.
7 THE WITNESS: Supports that they're
8 similar molecules, that it makes sense that they
9 have similarly increased -- that they similarly both
10 increase risk for Parkinson's, that PCE as having an
11 additional chlorine atom as compared to TCE is even
12 more lipophilic than TCE. Meaning, it will more
13 readily go to and store and accumulate in brain
14 tissue over time than TCE, that they, again, have
15 the same downstream metabolites, yes.
16 BY MR. GIBBONS:
17 Q. Okay. This sort of chemically structural
18 analogy is known as a structure activity
19 relationship, correct?
20 A. I'm not --
21 MR. TELAN: Object to the form.
22 THE WITNESS: That's not a term that I
23 commonly use.
24 BY MR. GIBBONS:
25 Q. Are you familiar with the term?

1 A. I may have heard it before, or read it
2 before.
3 Q. Okay. Do you have any training in
4 structural similarities and their relationship to
5 toxicity?
6 A. I mean, again, I have a Ph.D. in
7 biochemistry, so I'm well familiar. And for some
8 nuances, I might refer -- might defer to a
9 toxicologist.
10 Q. Okay. Have you ever published any studies
11 in the field of structure activity relationships or
12 chemical similarities?
13 A. No.
14 Q. Okay. Do you have any sources that
15 support the notion that a structurally similar
16 molecule produce similar toxicological effects?
17 MR. TELAN: Form objection.
18 THE WITNESS: That's a very broad
19 question.
20 There are references that I cite to even
21 in this report about PCE and TCE that discuss their
22 analogy and similarity and common metabolic pathways
23 and their common downstream effects of inflammation
24 and reactive oxygen specious formulation and
25 mitochondrial damage. So yes.

1 BY MR. GIBBONS:
2 Q. Okay. I just ask because I noticed in
3 your report, your statement that the similarities
4 between TCE and PCE suggesting similar toxicologies
5 didn't have any sort of footnote or citation, but
6 the majority of the rest of your paragraph did.
7 A. Are you still on page 49?
8 Q. Correct.
9 A. The references are 94 and 95 at the end of
10 the paragraph.
11 Q. Okay. And those are both publications by
12 the ATSDR, correct?
13 A. Correct.
14 Q. Okay. Would you agree that hydrogen
15 peroxide molecule has only one additional atom of
16 oxygen compared to a water molecule?
17 A. That's true, but I would not compare them
18 in the same way that we can discuss TCE and PCE.
19 Q. Would you agree that hydrogen peroxide and
20 water have different toxicological characteristics?
21 A. I'm not aware that water is toxic.
22 Q. Okay. So they would have different
23 characteristics, then.
24 So water would be toxic -- water is not
25 toxic, but hydrogen peroxide could be toxic in

1 specific dosages, correct?
2 A. Perhaps.
3 Q. When you said that you wouldn't make that
4 comparison, what did you mean?
5 A. I mean that it's too big of a stretch to
6 take any other two molecules that each vary by one
7 atom and make the same kind of analogy that -- that
8 we know exists for TCE and PCE because each molecule
9 and each class of molecules is different. And here
10 we're talking about molecules that cross the blood
11 brain barrier, go to substantia nigra or known to
12 cause Parkinson's. So it's too broad of a
13 generalization to pick two other molecules and draw
14 the exact same conclusions.
15 Q. Okay. When you were doing your literature
16 review for your report, did you come across a study
17 called "Comparative Analysis of Metabolism of
18 Trichloroethylene and Tetrachloroethylene Among
19 Mouse Tissues and Strains"?
20 A. I don't recall.
21 Q. If you did, it is not listed in your
22 reliance list, correct?
23 A. I could take your word for it or we could
24 look through it.
25 Q. Give me one moment.

1 MR. TELAN: What was the title of that
2 again, Hanley?
3 THE WITNESS: Compar- --
4 MR. GIBBONS: "Comparative Analysis of
5 Metabolism of Tetrachloroethylene and
6 Tetrachloroethylene Among Mouse Tissues and
7 Strains."
8 MR. TELAN: Is it on her materials
9 considered list?
10 MR. GIBBONS: I do not believe so.
11 (Whereupon, Deposition Exhibit 15 was
12 marked for identification.)
13 BY MR. GIBBONS:
14 Q. Dr. Andruska, just handed you what is now
15 Exhibit 15.
16 Have you seen this document before?
17 A. Not that I recall.
18 Q. Okay.
19 MR. TELAN: If you're going to ask her
20 questions about this, it is a 21-page article, or
21 22-page article, which she has not looked at. Or 23
22 pages.
23 MR. GIBBONS: That's fine. We can go off
24 the record and give her time to review it if you'd
25 like.

1 MR. TELAN: No. Don't go off the record
2 on these. If you want to ask her questions about a
3 document she hasn't seen before, she's entitled to
4 review it on the record, and then you can ask
5 questions as you like.
6 BY MR. GIBBONS:
7 Q. Okay. If you would turn to page 11,
8 please. It's the conclusions page.
9 Conclusion reads (as read):
10 In summary, this study
11 provides a comparative analysis for
12 the metabolisms between TCE and
13 PCE. We show that one atom
14 replacement of chlorine can
15 substantially affect the metabolism
16 via both oxidated and glutathione
17 conjugation pathways, the
18 qualitative and quantitative
19 differences between TCE and PCE
20 metabolites, as well as the
21 tissue-specific distribution of
22 metabolites can shed light on
23 differences between be TCE and
24 PCE-induced toxicities.
25 Did I read that correctly?

1 A. You read it correctly.
2 Q. Okay. That would suggest that one atom
3 replacement of chlorine can substantially affect the
4 toxicity between TCE and PCE, correct?
5 MR. TELAN: Object to the form. Lacks
6 predicate and foundation.
7 MR. GIBBONS: You can just limit it to
8 form.
9 MR. TELAN: I'm sorry?
10 MR. GIBBONS: You can limit it to form. I
11 understand your meaning.
12 MR. TELAN: Well, I didn't because you're
13 handing her a document and asking her to interpret
14 it, and it's an unfair question because she hasn't
15 read the question.
16 MR. GIBBONS: That would be a foundation
17 issue.
18 MR. TELAN: That's what I said. I said
19 lack of foundation, lack of predicate.
20 THE WITNESS: Thank you for the question.
21 I just need to review this.
22 MR. GIBBONS: What are we at?
23 THE VIDEOGRAPHER: 4 hours 29 minutes.
24 BY MR. GIBBONS:
25 Q. Okay.

1 A. I'm not done reviewing it yet, but I would
2 just pause to comment based on skimming the
3 introduction alone.
4 This is an article that focuses on toxic
5 differences of TCE and PCE, especially in liver and
6 kidney. They speak specifically about how the
7 metabolites can contribute to the nephrotoxicity or
8 kidney disease of TCE and PCE and also of cancers.
9 The article talks about differences in
10 metabolite pathways that are different than the
11 metabolites at hand in this case, where they're
12 talking about trichloroacetic acid as a metabolite
13 and trichloroethanol and chloral hydrate. Whereas,
14 we're talking about TaClo as it is specifically
15 relevant to brain, substantia nigra and Parkinson's.
16 And I'll continue reading. That was just the
17 introduction.
18 Q. Okay. Well, that was my only real
19 question about this study. But this does suggest
20 that the single atom of difference of chlorine can
21 have a different effect on toxicity, correct?
22 MR. TELAN: Object to the form. And I
23 think you -- she's not finished with her answer to
24 your prior question, which I think you just asked
25 again. But I think she should be allowed the time

1 to fully respond to the question as it was prompted.
2 THE WITNESS: This is also an article
3 about the interstrain variability as it relates to
4 different mouse models of non-Parkinson disease.
5 And, again, I'm still in the introduction, so ...
6 I'm yet to see anything that would change
7 my opinions.
8 BY MR. GIBBONS:
9 Q. Dr. Andruska, in the interest of time, I'm
10 going to limit my question.
11 So --
12 MR. TELAN: You're withdrawing your last
13 question?
14 MR. GIBBONS: Yes. I will modify my
15 question.
16 BY MR. GIBBONS:
17 Q. The study that I've just shown you, one of
18 the conclusions is that a single atom replacement of
19 chlorine can substantially affect the toxicity
20 between TCE and PCE, correct?
21 MR. TELAN: Object to the form of the
22 question. This question is essentially the same as
23 you had asked it before.
24 MR. GIBBONS: I'm asking specifically
25 about the conclusion of the study.

1 MR. TELAN: If you want her to read -- I
2 don't mind you asking her to read what the
3 conclusions says, but if you're asking her to
4 validate or verify the actual conclusion, then she
5 needs to be able to read the study.
6 MR. GIBBONS: Okay. Let me do this
7 instead.
8 BY MR. GIBBONS:
9 Q. Dr. Andruska, you specified that there was
10 a difference between neurotoxicity and
11 nephrotoxicity, which is primarily what this study
12 discusses, correct?
13 A. I mentioned that the study looks at a
14 variety of organs and, in part, focuses on
15 nephrotoxicity, yes.
16 Q. Based on the conclusions and whatever
17 you've read so far about the study, would you agree
18 that it is possible that a single atom replacement
19 of chlorine can affect the toxicity between TCE and
20 PCE in general toxicity, not neuro- or
21 nephro-specific toxicity?
22 MR. TELAN: Same objection and lack of
23 foundation, if we're not going to allow her to read
24 the article in its entirety.
25 THE WITNESS: I think it's important to

1 note that this is not an article about toxicity.
2 Rather, it's an article about the metabolism of
3 these compounds amongst three different strains of
4 mice.
5 And so I could not generalize these
6 conclusions to toxicity.
7 BY MR. GIBBONS:
8 Q. Okay. Based on what you've read so far,
9 you still see no reason to change your opinion that
10 the structural similarity between TCE and PCE would
11 suggest similar toxicities?
12 MR. TELAN: Object to the form.
13 THE WITNESS: Correct.
14 BY MR. GIBBONS:
15 Q. Okay.
16 MR. GIBBONS: Let's go ahead and take a
17 break.
18 THE VIDEOGRAPHER: We are going off the
19 record at 2:53 p.m.
20 (Whereupon, a recess was taken from
21 2:53 p.m. to 3:07 p.m.)
22 THE VIDEOGRAPHER: We are back on the
23 record at 3:07 p.m.
24 BY MR. GIBBONS:
25 Q. Dr. Andruska, is there anything from your

1 prior testimony that you'd like to correct before we
2 move on?
3 A. No, thank you.
4 Q. Okay. Back to your literature search, did
5 you come across a 2016 review called "Target Organ
6 Metabolism Toxicity In Mechanisms of
7 Trichloroethylene and Perchloroethylene, Key
8 Similarities, Differences and Data Gaps," by -- bear
9 with me for this name -- Cichocki?
10 A. I don't recall. I can tell you from just
11 reading part of the article you gave me that's an
12 article that was referenced in the metabolism
13 article and may have been referenced in other
14 articles that I cited to as well.
15 Q. If you had reviewed it and considered it,
16 would it have been in your materials considered
17 list?
18 A. Yes.
19 Q. Okay. Are you aware the National Research
20 Council wrote a report on Camp Lejeune in 2009?
21 A. I couldn't have told you the specific
22 year, but I am generally aware of the NRC.
23 Q. Okay. Did you review the NRC's 2009 study
24 when you were writing your report?
25 A. I don't recall.

1 Q. Okay.
2 A. I would think that it's not in my reliance
3 list, so I probably did not review it in -- for this
4 context, but happy to check the reliance list again,
5 noting that counsel provided an updated list last
6 night that we're not referencing today.
7 Q. Okay. I will represent to you, at least
8 in the first two material considered lists that we
9 reviewed, it was not in your material considered
10 list.
11 If you had reviewed the material, it would
12 be in one of those lists, correct?
13 A. One of those three lists, yes.
14 Q. Are you familiar with the Bradford Hill
15 considerations?
16 A. I am.
17 Q. Your Bradford Hill considerations are ones
18 that guide evaluation of evidence of causation,
19 correct?
20 MR. TELAN: Object to the form.
21 THE WITNESS: They are viewpoints as
22 originally described, that can be considered when
23 looking at causation and, of course, they are now
24 60 years old and developed in a time when we didn't
25 have mechanistic genetic good toxicologic data as we

1 do now.
2 But yes, I am aware of them.
3 BY MR. GIBBONS:
4 Q. Have you ever used the Bradford Hill
5 factors in your work?
6 A. Yes, I have.
7 Q. Okay. Have you used it in your clinical
8 practice?
9 A. As you know, they are -- viewpoints are
10 considerations most specifically for causation. The
11 general principles of strength of associations,
12 specificity, temporality, biological plausibility,
13 dose, coherence experiment analogy, et cetera, are,
14 I think, often generally relevant when making
15 decisions, for example, a differential diagnosis.
16 But we wouldn't formally use the Bradford Hill
17 considerations in a purely clinic setting.
18 Q. Okay. You did use the Bradford Hill
19 considerations in your report, though, correct?
20 A. I note that I am aware of them, I'm
21 familiar with their application, and I understand
22 them and have performed my own analysis. This is
23 page 63.
24 Q. Okay. The full quote was that you (as
25 read):

1 I have also reviewed the
2 reports of Dr. Boehme and Cannon,
3 and I reviewed their applications
4 of the Bradford Hill
5 considerations. I understand the
6 application of the Bradford Hill
7 consideration and have performed my
8 own analysis.
9 Correct?
10 A. That's correct.
11 Q. Can you explain the analysis that you
12 performed?
13 A. The analysis is specifically, most
14 specifically section 7, which includes the molecular
15 pathology of Parkinson's disease, the studies that
16 summarize the toxicity of TCE and PCE in animals and
17 humans, the mechanistic analysis, the studies that
18 I've included that would support many aspects of the
19 Bradford Hill considerations. But I defer to
20 Drs. Bayman, Cannon to do a formal Bradford Hill
21 analysis.
22 Q. So you didn't explicitly perform a
23 Bradford Hill analysis within your report, then,
24 correct?
25 A. Correct.

1 Q. Okay. You also considered the report of
2 Dr. Gary Miller, correct?
3 A. Correct.
4 Q. It was listed within your reliance
5 materials?
6 A. I have read it, yes.
7 Q. Did you conduct a Bradford Hill
8 consideration review of Dr. Miller's report?
9 MR. TELAN: Object to the form.
10 THE WITNESS: I'm not sure I understand
11 your question.
12 BY MR. GIBBONS:
13 Q. Did you analyze Dr. Miller's application
14 of the Bradford Hill criteria?
15 A. I read Dr. Miller's report. I don't
16 recall, specifically sitting here, the details of
17 what he included in the report.
18 Q. Okay. Would you have used the same
19 analysis for Dr. Miller's report that you used for
20 Dr. Boehme and Dr. Cannon's reports?
21 A. I would have considered it equally, and if
22 I had specifically disagreed with something in his
23 report, I would have mentioned it.
24 Q. Okay. And you also considered the report
25 of Dr. Lucio Costa, correct?

1 A. It sounds familiar.
2 Q. Okay. Would it be in Exhibit 5? That
3 page, I believe.
4 MR. TELAN: What page number, Hanley? I'm
5 sorry.
6 MR. GIBBONS: Page 11 of Exhibit 5.
7 MR. TELAN: Okay. So page 76?
8 MR. GIBBONS: I believe so. Hold on one
9 second.
10 It might be Exhibit 6.
11 MR. TELAN: Mind if I show it to her just
12 so that --
13 MR. GIBBONS: Yeah.
14 MR. TELAN: Here you go.
15 THE WITNESS: Yes, I see it's in my
16 reliance list.
17 BY MR. GIBBONS:
18 Q. Okay. So you did review that, then?
19 A. Yes.
20 Q. Okay. And you would have used the same
21 analysis of their application of the Bradford Hill
22 considerations that you did for Drs. Boehme and
23 Dr. Cannon?
24 A. Yes.
25 Q. Okay. Bradford Hill is relevant for

1 general causation, correct?
2 A. Correct.
3 Q. To your knowledge, were any of the
4 materials that you relied on for your report
5 considered non-statistically significant for the
6 relationship between Parkinson's disease and TCE?
7 MR. TELAN: Object to the form.
8 THE WITNESS: We have discussed today a
9 few examples of studies where the P value was not
10 exactly less than 0.05.
11 I don't have other examples on the top of
12 mind.
13 BY MR. GIBBONS:
14 Q. Same answer for studies that evaluated the
15 relationship between Parkinson's disease and PCE?
16 A. Yes.
17 Q. Okay. Same question about studies
18 evaluating the relationship between vinyl chloride
19 and Parkinson's disease?
20 MR. TELAN: Object to the form.
21 MR. GIBBONS: Okay. I'll rephrase.
22 BY MR. GIBBONS:
23 Q. Did you consider any non-statistically
24 significant studies evaluating the relationship
25 between Parkinson's disease and vinyl chloride?

1 A. Vinyl chloride specifically was not within
2 the scope of my report, but, of course, as we've
3 discussed today, there are some studies that have
4 looked at chlorinated hydrocarbons as a class, and
5 same answer.
6 Q. Okay. Did you consider any
7 non-statistically significant studies evaluating the
8 relationship between Parkinson's disease and
9 Benzene?
10 A. Same answer.
11 Q. Did you consider any non-statistically
12 significant studies evaluating the relationship
13 between Parkinson's disease and a mixture of all
14 four VOCs that I previously mentioned?
15 A. Same answer. And if they were, of course,
16 we would put them in that clinical context that
17 we've already discussed.
18 Q. Okay. For your report, you relied on the
19 exposure calculations of Dr. Kelly Reynolds,
20 correct?
21 A. That's correct.
22 (Whereupon, Deposition Exhibit 16 was
23 marked for identification.)
24 BY MR. GIBBONS:
25 Q. You were just handed Exhibit 16.

1 Is this the report by Dr. Kelly Reynolds
2 that I'm referring to?
3 A. This is Dr. Kelly Reynolds' report.
4 Sitting here today, I don't recall if what I
5 reviewed was specifically her exposure calculations
6 for Mrs. Reynolds and/or -- and this report.
7 Q. Okay. I just want to clarify, when you
8 said the calculations for Mrs. Reynolds, did you
9 mean the calculations for Ms. Rothschild?
10 A. Yes, thank you. Hopefully, Dr. Reynolds
11 did not have TCE or PCE exposure.
12 (Whereupon, Deposition Exhibit 17 was
13 marked for identification.)
14 MR. GIBBONS: Handing you Exhibit 17.
15 BY MR. GIBBONS:
16 Q. This is Appendix 23 to Dr. Reynolds'
17 report. Title of the appendix is Diane Rothschild's
18 Parkinson's disease, correct?
19 A. Correct.
20 Q. Did you review these tables in writing
21 your report?
22 A. I did, yes.
23 Q. Okay. Does this show the model exposure
24 calculations that Dr. Reynolds provided for you?
25 A. Yes, it does.

1 Q. In page 62 of your report, you wrote (as
2 read):
3 Dr. Reynolds' charts support
4 my opinions that Ms. Rothschild had
5 substantial exposure to toxic
6 chemicals at Camp Lejeune. The
7 charts detail reasonable estimated
8 consumption dose for
9 Ms. Rothschild. Exposure to these
10 levels of PCE alone represent a
11 substantial exposure.
12 Did I read that correctly?
13 A. Yes, you did.
14 Q. Okay. Did you define substantial exposure
15 anywhere in your report?
16 A. No.
17 Q. Did you quantify substantial exposure
18 anywhere in your report?
19 A. I did not define or quantify the word
20 "substantial."
21 Q. Okay. Did you identify a specific
22 threshold amount whereby an individual exposed to
23 TCE is guaranteed to develop Parkinson's disease?
24 MR. TELAN: Object to the form.
25 THE WITNESS: As we've discussed earlier

1 today, I don't doubt that such a threshold exists,
2 but it's not known based on the inherent
3 limitations.
4 BY MR. GIBBONS:
5 Q. Okay. Is the same statement true for PCE
6 as well?
7 A. That's correct.
8 Q. And the same statement true for Benzene as
9 well?
10 A. Yes.
11 MR. TELAN: Object to the form.
12 BY MR. GIBBONS:
13 Q. Same statement true for vinyl chloride?
14 MR. TELAN: Object to the form.
15 THE WITNESS: Yes.
16 BY MR. GIBBONS:
17 Q. Is the same true for a mixture of all the
18 aforementioned VOCs?
19 MR. TELAN: Object to the form.
20 THE WITNESS: Yes.
21 BY MR. GIBBONS:
22 Q. Okay. Did you independently calculate the
23 amount of TCE that Ms. Rothschild was exposed to
24 during her time at Camp Lejeune?
25 A. No.

1 Q. Did you independently calculate the amount
2 of PCE that Ms. Rothschild was exposed to during her
3 time at Camp Lejeune?
4 A. No.
5 Q. Did you independently calculate the amount
6 of Benzene that Ms. Rothschild was exposed to at her
7 time at Camp Lejeune?
8 A. No.
9 Q. Did you independently calculate the amount
10 of vinyl chloride that Ms. Rothschild was exposed to
11 while she was at Camp Lejeune?
12 A. No. I relied on Dr. Reynolds.
13 Q. Okay. You didn't do any independent
14 verification of Ms. Rothschild's Camp Lejeune
15 residence or work histories, correct?
16 A. When I spoke to Mrs. Rothschild during my
17 own exam, I briefly covered her mili- -- or her
18 exposure -- her time on Camp Lejeune as part of my
19 social history. And I spoke to Dr. Reynolds as well
20 to make sure I understood her calculations, but I
21 didn't independently recalculate the numbers.
22 Q. Okay. Did you review any maps of
23 Camp Lejeune to understand where Ms. Rothschild
24 lived on the base?
25 A. Yes.

1 Q. Did you examine any maps of Camp Lejeune
2 that showed the location of water treatment plants
3 on Camp Lejeune?
4 A. It's quite possible. I can't remember
5 exactly which maps I've seen in this case.
6 Q. Okay. Would there be any maps that you
7 reviewed that were not included in the studies that
8 you referenced in your materials considered list?
9 A. No.
10 Q. Okay. Did you review any maps that
11 purported to show where Ms. Rothschild worked on the
12 base?
13 A. Are you asking more specifically within
14 Tarawa Terrace?
15 Q. Just within Camp Lejeune in general.
16 A. I understand that she worked in
17 Tarawa Terrace. The maps delineate that section of
18 Camp Lejeune, so yes.
19 Q. Okay. So follow-up question now, which
20 you have already answered, or indicated.
21 Did you review any maps that specifically
22 showed where in Tarawa Terrace Ms. Rothschild
23 worked?
24 A. I'm not sure.
25 Q. Okay. Did you review any maps that showed

1 where her day-to-day activities other than work
2 occurred on the base?
3 A. I'm not sure.
4 Q. Are you aware of whether Dr. Reynolds' use
5 of the total mass of ingested chemicals is a
6 standard exposure metric in risk assessment?
7 A. I would defer to Dr. Reynolds on that.
8 Q. Okay. Are you aware of whether total mass
9 ingested is generally accepted in the field of
10 toxicology?
11 A. Same answer.
12 Q. Are you aware of any -- whether any
13 epidemiological studies apply the same exposure
14 metric that Dr. Reynolds used in her report?
15 A. I've certainly seen micrograms per liter
16 and parts per billion as units in other
17 epidemiologic studies. Again, beyond that, I would
18 refer to Dr. Reynolds.
19 Q. So just to clarify, you're not sure
20 whether you've reviewed epidemiological studies that
21 use the total cumulative micrograms per liter for
22 calculating exposure?
23 A. To be clear, micrograms per liter is a
24 concentration. A total cumulative amount would not
25 specify a per-volume rate. So I'm not sure I

1 understand your question.
2 Q. Okay. Same question. We'll disregard the
3 micrograms per liter. It's concentration, as you
4 pointed out.
5 Are you aware of whether any
6 epidemiological studies use the same metric, which
7 is total volume of exposure that Dr. Reynolds used?
8 A. I'm not sure.
9 Q. Okay. Would you agree that most reliable
10 epidemiological studies provide cumulative exposure
11 in terms of concentration times number and months
12 supposed?
13 MR. TELAN: Object to the form.
14 THE WITNESS: They could provide that.
15 I've also mentioned epidemiologic studies today that
16 might report hours per week and years of exposure.
17 So I'm not too pigeonholed on the specific units of
18 rate. But I agree, generally, that rate or total
19 exposure can be helpful.
20 BY MR. GIBBONS:
21 Q. Okay. You used Dr. Reynolds' total
22 ingestion metric to conclude the potential health
23 affects for Diane Rothschild, correct?
24 A. Correct. In conjunction with the
25 remaining totality of the evidence that -- that I've

1 mentioned a few times already.
2 Q. Did you use that ingestion calculation in
3 any other ways in your report?
4 A. I don't believe so. If I misunderstand
5 the question, please clarify.
6 Q. Do you know whether the use of total
7 ingestion is an acceptable methodology for
8 causation?
9 MR. TELAN: Object to the form.
10 THE WITNESS: Again, I would defer to
11 other experts.
12 BY MR. GIBBONS:
13 Q. Okay. Have you ever used that metric in
14 any of your peer-reviewed literature?
15 A. That I've personally authored?
16 Q. Correct.
17 A. No.
18 Q. Okay. Have you ever used this methodology
19 in your clinical practice to ascertain patient's
20 health outcomes?
21 MR. TELAN: Object to the form.
22 THE WITNESS: No. And the answer both for
23 this question and the previous question is, again,
24 my research is not germane to micrograms or total
25 exposure, and my clinical practice is focused on

1 diagnosis and treatment.
2 BY MR. GIBBONS:
3 Q. Okay. In your report on page 27, you show
4 that Ms. Rothschild was exposed to a cumulative dose
5 of between 245 micrograms per liter and 1,265
6 micrograms per liter, correct?
7 MR. TELAN: TCE you're talking about?
8 MR. GIBBONS: Correct. For TCE.
9 THE WITNESS: Charts 1 through 5 do have
10 that range, and on page 26, I also reference central
11 tendency exposures commensurate with the ATSDR
12 estimate of civilian workers' central tendency
13 exposures starting at 1.227. That's chart 3. And
14 then discuss how ATSDR has higher rates of
15 consumption for relative maximum exposure and how
16 Mrs. Rothschild is also drinking more water -- was
17 drinking more water depending on whether she was
18 working inside or outside.
19 So my point here is we're -- we're
20 generally between chart 3 and chart 5, although, I
21 also point out that chart 2 has the higher rate of
22 consumption. So it's a bit, I would say, between
23 chart 2 and chart 5.
24 BY MR. GIBBONS:
25 Q. Okay.

1 A. They're out of order.
2 Q. Understood.
3 Would you agree that the total model
4 exposure for Ms. Rothschild to TCE was between
5 245 micrograms and 1,265 micrograms?
6 A. Yes.
7 Q. And those calculations were performed by
8 Dr. Reynolds, correct?
9 A. Correct.
10 Q. Ms. Rothschild's total model exposure for
11 PCE ranged from 7,580 micrograms to a high of
12 32,153 micrograms, correct?
13 A. Yes. Although, I'll give you, if we're
14 including 245 micrograms for TCE, we should include
15 6,177 micrograms for PCE.
16 Q. Correct. Sorry. My mistake.
17 So a lower model limit of
18 6,177 micrograms?
19 A. Yes. If we're using chart 1, which has
20 its caveats as previously mentioned.
21 Q. Okay. In your report going back to
22 page 62, the last paragraph, you wrote (as read):
23 At PCE concentrations of 40.1
24 to 43.76 parts per billion or
25 micrograms per liter is estimated

1 specifically for Ms. Rothschild.
2 During her time working at
3 Camp Lejeune, her PCE exposure is
4 far above the EPA's maximum
5 contaminant levels in drinking
6 water of 5 parts per billion.
7 Did I read that correctly?
8 A. You did.
9 Q. You go on to say that (as read):
10 Exposure to median PCE levels
11 of 15.4 parts per billion is
12 substantial and known to cause
13 Parkinson's disease.
14 Did I read that correctly?
15 A. You did.
16 Q. Do you have a source for the second
17 statement that exposure to median PCE levels of 15.4
18 parts per billion is substantial and known to cause
19 Parkinson's disease?
20 A. This is Goldman 2023, which I reference in
21 the next sentence.
22 Q. Okay. And Goldman's 2023 study
23 specifically said that PCE was known to cause
24 Parkinson's disease?
25 A. Well, that's a -- maybe a bit of a

1 mischaracterization. Goldman showed that at median
2 PCE levels of 15.4, there was a -- you know, four
3 people who lived on base -- worked on base for at
4 least three months. There was an odds ratio of 1.7
5 for the risk of Parkinson disease in these exposed
6 people.
7 Q. So would it be fair to characterize it as
8 likely to cause Parkinson disease rather than known
9 to cause Parkinson's disease?
10 A. Or demonstrated to cause Parkinson's
11 disease.
12 Q. Okay. And we've already talked about the
13 way in which the EPA establishes MCLs, correct?
14 A. Correct.
15 Q. Okay. And that MCLs are designed to be
16 used for in drinking water concentrations over a
17 lifetime of exposure of approximately 70 years,
18 correct?
19 MR. TELAN: Object to the form. Asked and
20 answered.
21 THE WITNESS: Yes.
22 BY MR. GIBBONS:
23 Q. Are you aware that health protective
24 assumptions go into determining the MCL?
25 A. Am I aware of --

1 MR. TELAN: Form objection.
2 BY MR. GIBBONS:
3 Q. I said are you aware of the health
4 protective assumptions that go into determining an
5 MCL?
6 A. I'm not exactly sure.
7 Q. Okay. Are you aware that the EPA uses
8 cumulative dose average over a lifetime to evaluate
9 the disease risk?
10 MR. TELAN: Object to the form.
11 THE WITNESS: I'm not specifically aware.
12 BY MR. GIBBONS:
13 Q. Okay. Is an exposure to drinking water
14 concentration above an MCL guaranteed to constitute
15 a health risk?
16 A. Again --
17 MR. TELAN: Object to the form.
18 THE WITNESS: Guaranteed is a strong word.
19 I don't think anything in science or medicine is
20 guaranteed.
21 BY MR. GIBBONS:
22 Q. Okay. Let me rephrase.
23 An exposure to water -- to drinking water
24 concentrations in excess of an MCL doesn't
25 necessarily constitute a health risk, correct?

1 MR. TELAN: Object to the form.
2 THE WITNESS: I would disagree with that.
3 Since the definition or what we do know about MCL is
4 that levels below the MCL are reasonably considered
5 not to cause disease. You're asking about the
6 inverse, could levels higher than the MCL cause
7 disease?
8 BY MR. GIBBONS:
9 Q. Could levels higher than the MCL still not
10 be considered a health risk?
11 MR. TELAN: Object to the form.
12 THE WITNESS: I would say no.
13 BY MR. GIBBONS:
14 Q. Okay. So any time that there's exposure
15 to a level above an MCL, that would be considered a
16 health risk?
17 MR. TELAN: Object to the form.
18 THE WITNESS: It could be.
19 BY MR. GIBBONS:
20 Q. Okay. Could be or would be?
21 MR. TELAN: Form.
22 THE WITNESS: Would be until proven
23 otherwise.
24 BY MR. GIBBONS:
25 Q. Okay. You were hired as an expert in

1 Parkinson's disease, correct, for this case?
2 A. I am an expert in Parkinson's disease, and
3 part of my scope was to perform a differential
4 etiology and a differential diagnosis. So yes, and
5 considering a broader implication.
6 Q. Okay. Have you ever been qualified by a
7 court as an expert in genetics?
8 A. Not specifically, no.
9 Q. Okay. What do you mean, not specifically?
10 A. I'm referencing that you asked a similar
11 question earlier, and I explained that I have
12 expertise as a scientist in genetics, but if we're
13 making a distinction between being a geneticist,
14 then the answer to your question is no.
15 Q. Have you ever been retained as an expert
16 witness in the field of genetics?
17 A. There are certainly -- there have been
18 certainly other Parkinson's cases in the legal arena
19 where genetics of Parkinson's disease and
20 Parkinsonisms has played a very large role. And
21 that has entered into my scope.
22 Q. Okay. Would you agree that medicine is
23 not an exact science?
24 A. Sometimes, yes. But science is an exact
25 science.

1 Q. Fair enough. We're talking tautologies
2 now.
3 Would you agree that the cause of
4 Parkinson's disease is multifactorial?
5 MR. TELAN: Object to the form.
6 THE WITNESS: Parkinson's disease is
7 generally established to have more than one
8 potential cause, or at least a potential mix of
9 factors, but for any given individual, that answer
10 could vary.
11 BY MR. GIBBONS:
12 Q. Okay. Would you agree that no single
13 factor categorically guarantees a person will
14 develop Parkinson's -- Parkinson's disease in their
15 lifetime?
16 A. If we're distinguishing or speaking about
17 risk factors, yes.
18 Q. Okay. Meaning someone could have a risk
19 factor and it still not be the cause of their
20 Parkinson's disease, correct?
21 A. Someone could have a risk factor and it
22 not be the only cause of their Parkinson's disease.
23 Q. But if they have a risk factor and
24 Parkinson's disease, then the risk factor would be a
25 cause of their Parkinson's disease, correct?

1 A. It can be a more nuanced distinction
2 between risk factors and causes. And so I might
3 just clarify that a risk factor, like age or sex, is
4 not a determinant of Parkinson disease.
5 Q. Okay. Are there any risk factors that you
6 believe are determinant of Parkinson's disease?
7 MR. TELAN: Object to the form.
8 THE WITNESS: Not if we define risk
9 factors as influential variables that are not
10 explicitly causative in and of themselves, no.
11 BY MR. GIBBONS:
12 Q. Okay. Are environmental factors a risk
13 factor in Parkinson's disease?
14 MR. TELAN: I'll just object to the form.
15 It's overbroad.
16 THE WITNESS: Again, a nuanced discussion.
17 And it may not be relevant for our purpose today.
18 But we are more apt to say that environmental
19 exposures can be a cause of Parkinson disease or
20 increased someone's risk for Parkinson's disease.
21 And I'm more comfortable with that language.
22 BY MR. GIBBONS:
23 Q. Okay. So environmental exposure could be
24 a risk factor in Parkinson's disease?
25 MR. TELAN: Object to the form.

1 THE WITNESS: Could increase one's risk or
2 cause.
3 BY MR. GIBBONS:
4 Q. Okay. And what are some other risk
5 factors that would or could increase someone's risk
6 of developing Parkinson's disease?
7 A. I've mentioned sex. There's more men than
8 women diagnosed with Parkinson disease. While we
9 used to think that was largely driven by diagnostic
10 bias or medical care seeking behaviors, there's
11 better evidence now to suggest that there may be a
12 hormonal or true sex difference driving male sex as
13 a risk factor. We know that smoking is an anti-risk
14 factor or protective in Parkinson's.
15 We've discussed certain types of head
16 trauma today that could be a risk factor as well.
17 Q. Okay. Would old age be considered a risk
18 factor of developing Parkinson's disease?
19 MR. TELAN: Object to the form.
20 THE WITNESS: Age is a risk factor.
21 BY MR. GIBBONS:
22 Q. Is there an increase in frequency in
23 developing Parkinson's disease after age 55?
24 A. There is an increased incidence of
25 Parkinson's with -- with age in general. The way

1 it's been studied, the way it's quantified in the
2 literature is to break age into decades or blocks of
3 decades, and we could quote, then, the incidents of
4 Parkinson's by age bracket.
5 Q. Okay.
6 A. I used 55 to 60 earlier as an average age
7 of onset in the spectrum of Parkinson's disease. So
8 two slightly different things, but, of course, both
9 age.
10 Q. Okay. I understand.
11 Given that, would you say that it is rare
12 for someone to develop Parkinson's disease in their
13 30s?
14 A. It is less common.
15 Q. What about in their 40s?
16 MR. TELAN: Object to the form.
17 THE WITNESS: Broadly speaking, more
18 common. And if we were to name each subsequent
19 decade, again, more common.
20 BY MR. GIBBONS:
21 Q. Okay. So the risk of developing
22 Parkinson's disease increases with age?
23 A. Correct.
24 Q. Okay. Is there ever a point that you're
25 aware of at which point the risk of Parkinson's

1 disease decreases with age?
2 A. No.
3 Q. Okay.
4 A. Not with advancing age, to be clear.
5 Q. Advancing age, sorry.
6 Increasing in age leads to a naturally
7 dopaminergic neuron loss, correct?
8 A. That's generally thought to be true. It's
9 hard to measure without autopsy, so certain
10 limitations there.
11 Q. Okay. Is lack of exercise a risk factor
12 for developing Parkinson's disease?
13 A. No, I wouldn't call it a risk factor. I
14 think it's more accurate to say that exercise can be
15 protective or neuro protective in slowing down
16 disease progression.
17 Q. Are there dietary factors that are risk
18 factors for Parkinson's?
19 A. No.
20 Q. Is increased alcohol use considered a risk
21 factor for Parkinson's?
22 A. No.
23 Q. Is there any association between dairy
24 intake and increased risk for Parkinson's?
25 A. It's the reason I paused when you asked

1 about diet. Dairy is one class of foods that has
2 been queried or that for some people may impact
3 their experience with Parkinson's, meaning they
4 may -- there may be groups of foods, for example,
5 gluten or dairy, that for certain people who have
6 intolerances or just because of the natural
7 variation, these subgroups of people may choose to
8 try eliminating, for example, dairy, but this is not
9 a risk factor or a universal treatment, if you will,
10 for Parkinson's.
11 Q. Is positive family history associated with
12 increased risk of Parkinson's disease?
13 A. In Parkinson's, we think about genetic
14 mutations. When a person with Parkinson's has more
15 than one first degree relative also with
16 Parkinson's, this is an extension of what we've just
17 spoken about. The prevalence of Parkinson's is
18 common enough that typically having one first degree
19 relative with Parkinson's could be explained by
20 chance. But if we see patterns that are consistent
21 with autosomal dominant or autosomal recessive gene
22 mutations, such as multiple persons in each
23 generation or every other generation with
24 Parkinson's, then we're often thinking more strongly
25 about an inherited genetic mutation.

1 Q. Can genetic mutations occur spontaneously?
2 A. They can.
3 Q. Okay. Going back to environmental
4 factors, I just want to be a little bit more
5 specific.
6 Is exposure to air pollution considered a
7 risk factor for Parkinson's disease?
8 A. This has been looked at a little bit, and
9 maybe. But there's just not a lot of evidence on
10 that topic. And it's hard to study.
11 Q. Okay. What about exposure to micro
12 plastics or nano plastics?
13 A. Good question. It's an interesting modern
14 topic in neurology and medicine. I'm not aware of
15 whether that's been looked at in Parkinson's
16 specifically.
17 Q. Okay. Are exposures to specific chemicals
18 or toxins known to be a risk factor for Parkinson's
19 disease?
20 A. Yes. Or likely to cause Parkinson's, as I
21 have specified earlier.
22 Q. Is MPTP one of those chemicals?
23 A. Yes.
24 Q. Okay. Is exposure to pesticide considered
25 a risk factor for Parkinson's?

1 A. Certain chemicals -- certain pesticides
2 are thought to cause Parkinson's.
3 Q. Are there any occupational-related
4 exposures that are thought to be connected to
5 Parkinson's disease?
6 A. Occupational exposures is a broad
7 question, of course, and more important is the root
8 cause or the proximate exposure. When we talk about
9 occupational exposures in addition to the chemicals
10 that are at hand today, we might also consider heavy
11 metals that a person is exposed to through their
12 occupation.
13 Q. Okay. Between heavy metals, organic
14 solvents that we've already discussed and
15 pesticides, would growing up on a farm also be
16 considered a risk factor for Parkinson's disease?
17 MR. TELAN: Object to the form.
18 THE WITNESS: Some of the epidemiologic
19 literature in the pesticide realm has looked at
20 farm-related associations, and data there is more
21 mixed than if we look specifically at people who
22 apply pesticides and have more granularly documented
23 exposures to pesticides.
24 BY MR. GIBBONS:
25 Q. Okay.

1 A. I think the reason some of the studies are
2 positive is in addition to the dermal inhalation and
3 ingestion routes that we've talked about today,
4 chemicals, including pesticides and solvents, can
5 also seep into the ground and affect the water and
6 people can be exposed, for example, on farms, if
7 they are on a water supply where there are near
8 field contaminants of chemicals.
9 Q. Okay. Are there certain chemical -- or --
10 sorry.
11 Are there certain medical comorbidities
12 that are associated with increased risk of
13 Parkinson's disease?
14 For example, are there any certain
15 infectious diseases that are associated with
16 increased risk of Parkinson's?
17 A. It's a good question. It's a clearer
18 answer if we use the term "Parkinsonism" instead of
19 Parkinson disease. But even so, we might point to
20 infections like the Spanish flu of 1918 as causing
21 Parkinson's. But we're on safer ground if we step
22 back and think about the Parkinsonisms more broadly.
23 Q. Just to clarify Parkinsonisms is a broader
24 category that includes other diseases besides
25 Parkinson's disease, correct?

1 A. It can, but as I define Parkinsonisms in
2 my report, it's really an umbrella term to reference
3 symptoms that can be seen in Parkinson's and can
4 also be seen in isolation or in the milieu of other
5 diseases that are not specifically Parkinson
6 disease.
7 Q. Okay. Is upper gastrointestinal mucosal
8 damage considered a risk factor for Parkinson's
9 disease?
10 MR. TELAN: Object to the form.
11 THE WITNESS: That's a very specific
12 question. And if you have a study, I'd be happy to
13 look at it. I -- I'm certainly aware of a gut-brain
14 link in Parkinson's and a postulated route for some
15 types of Parkinson's to spread from the gut to the
16 brain, but this is not something that we screen for
17 clinically or use in standard clinical practice.
18 BY MR. GIBBONS:
19 Q. Okay. But you are aware of literature
20 that links gastrointestinal mucosal damage to
21 Parkinson's to some degree? You don't need specific
22 studies.
23 Is there literature that considered some
24 link between the two?
25 A. Of mucosal damage to Parkinson's?

1 Q. Correct.
2 A. I couldn't quote any literature today.
3 Q. More generally, is there any literature
4 that links the gut micro biome with Parkinson's
5 disease?
6 A. Yes.
7 Q. Okay. Is having multiple strokes a risk
8 factor for Parkinson's disease?
9 A. No.
10 Let me clarify. If one had a stroke in
11 the basal ganglia or, in other words, in anatomic
12 regions of the brain that are responsible for
13 Parkinson disease, one could have Parkinsonism that
14 could mimic Parkinson disease, but the pure answer
15 to your question is no, strokes don't cause
16 Parkinson's disease's disease.
17 Q. So a stroke in, for instance, the
18 substantia nigra pars compacta that depletes the
19 dopaminergic neurons, that could cause Parkinsonism,
20 but not necessarily Parkinson's disease, correct?
21 A. Correct.
22 Q. Okay. And that would only be verifiable
23 during the autopsy, correct?
24 A. No. You could see a stroke on a
25 structural brain MRI, and also part of our standard

1 clinical practice is to obtain such an MRI at the
2 time of diagnosis to rule out mimics such as this.
3 Q. Okay. Are the exposure or use of
4 medications that block or deplete dopamine in the
5 brain considered a risk factor for Parkinson's?
6 A. Again, it's important to distinguish
7 between medication-induced Parkinsonism, and if
8 we're using that more accurate terminology, then
9 yes. And, of course, as I mentioned earlier,
10 dopaminergic -- anti-dopaminergic medications are
11 something we would ask about in a -- in a good
12 Parkinson's history to make sure that we're not
13 missing a Parkinsonism mimic. But it -- there are
14 ways to differentiate between medication-induced
15 Parkinsonism and Parkinson disease.
16 MR. GIBBONS: Okay. What are we at right
17 now?
18 THE VIDEOGRAPHER: 5 hours 37 minutes.
19 MR. GIBBONS: Let's take a five-minute
20 break real quick.
21 THE VIDEOGRAPHER: Going off the record at
22 4:03 p.m.
23 (Whereupon, a recess was taken from
24 4:03 p.m. to 4:17 p.m.)
25 THE VIDEOGRAPHER: We are back on the

1 record at 4:17 p.m.
2 BY MR. GIBBONS:
3 Q. Dr. Andruska, before we took a break,
4 would you like -- or before we come back into the
5 questions, is there anything from your previous
6 testimony you'd like to correct?
7 A. Not to correct, but I might add on the
8 topic of genetics, you asked me about spontaneous
9 mutations and we were talking about genetic factors.
10 And we -- and I didn't mention at the time the idea
11 of penetrants, which is quite relevant in
12 Parkinson's. I discuss it in my report. I define
13 it. Briefly it's the idea that just because someone
14 has a genetic mutation doesn't mean that they will
15 develop disease, and that likelihood is based on the
16 percent penetrants of the mutation.
17 Q. Okay. Shifting slightly, would you agree
18 that every patient is different with their own
19 idiosyncratic risk factors?
20 A. Yes.
21 Q. Would you agree that the precise cause of
22 Parkinson's disease is generally unknown?
23 MR. TELAN: Object to the form.
24 THE WITNESS: For most patients in typical
25 clinical practice, yes.

1 BY MR. GIBBONS:
2 Q. Okay. And what about in this case in
3 particular?
4 A. This is different. Of course, we're here
5 in a legal context and we're looking at causes, and
6 we have very granular data specific to
7 Mrs. Rothschild. It's wonderful, but it's also very
8 unique and distinct from a typical clinical
9 environment.
10 Q. Okay. Are you familiar with the term
11 "idiopathic"?
12 A. Yes.
13 Q. Okay. Would you agree that some
14 Parkinson's disease has an idiopathic etiology?
15 A. On pages, I think, 29 to 31 of my report,
16 I discuss the nuances of idiopathic. It's an
17 important point. And -- and we can talk through the
18 evolution of it. But the bottom line is, idiopathic
19 is an outdated term that we now, as movement
20 disorder specialists, use sometimes. And when we
21 do, we imply that we now understand that Parkinson
22 disease has usually a combination of genetic and/or
23 environmental factors.
24 Q. Okay. Dr. Perlmutter diagnosed
25 Ms. Rothschild with idiopathic Parkinson's disease,

1 correct?
2 A. He did. And he defined idiopathic in his
3 deposition as -- I'm paraphrasing, but I did quote
4 in my report along the lines of the standard
5 presentation and progression of Parkinson disease
6 similar to what I've just defined.
7 Q. Would you agree that generally idiopathic
8 Parkinson's disease is defined as having an unknown
9 cause?
10 MR. TELAN: Object to the form.
11 THE WITNESS: No. For the reasons I -- I
12 just said.
13 BY MR. GIBBONS:
14 Q. Okay. Would you agree that between 70 and
15 80 percent of Parkinson's disease cases do not have
16 a specifically known cause?
17 MR. TELAN: Object to the form.
18 THE WITNESS: If you -- if you have a
19 reference to that, I'd be happy to look at it. The
20 percentages that we might quote are in 5 to
21 10 percent of cases. We may have an identified
22 genetic mutation, penetrants notwithstanding, and --
23 and we've already discussed today that while there
24 are many causes or modifiers or risk factors for
25 Parkinson disease, each individual is different and

1 we're often not privileged to really dive into their
2 causality as we are in a legal case.
3 BY MR. GIBBONS:
4 Q. Okay. Putting aside specific percentages,
5 would you agree that in most cases, Parkinson's
6 disease does not have an attributable cause?
7 MR. TELAN: Form objection.
8 BY MR. GIBBONS:
9 Q. Or identifiable cause, rather.
10 A. Yes. Because in the clinic, it doesn't
11 serve our purpose to treat the Parkinson disease
12 today to know a priori what the cause is since our
13 treatments are not cause-specific.
14 Q. Okay. In your clinical practice, how
15 often do you attempt to determine the cause of a
16 patient's Parkinson's disease?
17 A. I don't have a number today, but as I
18 mentioned before, patients will often ask me about
19 causes of Parkinson disease in general and their
20 specific causes. And we will often talk about that.
21 And -- and I consider causes in my social history
22 and elsewhere in addition to a differential
23 diagnosis.
24 Q. Okay. How often do you employ a
25 differential diagnosis in your practice?

1 A. I would say with every patient.
2 Q. Okay. What causes do you consider when
3 you're doing your differential?
4 MR. TELAN: Object to the form.
5 THE WITNESS: A differential diagnosis for
6 Parkinson disease typically includes the framework
7 that I laid out for Mrs. Rothschild starting on
8 page 57. I might reference it now for clarity.
9 I would typically consider structural
10 changes, vascular damage, stroke, tumor,
11 inflammatory process. Again, these are mimics or
12 Parkinsonism mimics, vascular Parkinsonism,
13 anti-dopaminergic medications, certain occupational
14 exposures, Wilson disease, infectious and
15 inflammatory processes, certain types of head
16 trauma, genetic factors, atypical Parkinsonian
17 syndromes, and then on a case-by-case basis, other
18 diagnoses that may be relevant.
19 BY MR. GIBBONS:
20 Q. After ruling in all reasonably potential
21 causes, would you agree that a differential
22 diagnosis requires ruling out potential causes until
23 you can't rule any more out?
24 MR. TELAN: Object to the form.
25 THE WITNESS: You said after ruling in all

1 potential causes, would I rule out as many diagnoses
2 as I could?
3 BY MR. GIBBONS:
4 Q. Correct.
5 A. The process of a differential diagnosis, I
6 think, is better described as considering all
7 potential and relevant causes and either making a
8 hierarchical list, as we said earlier, and/or ruling
9 out the ones that don't fit, and ultimately applying
10 well-established diagnostic criteria to a patient's
11 symptoms and presentations in order to rule it in
12 with as much certainty as we can.
13 Q. You ultimately concluded that the cause of
14 Ms. Rothschild's Parkinson's disease was her
15 exposure to Camp Lejeune water, correct?
16 A. That a cause of her Parkinson's at least
17 as likely as not is her exposure to the water at
18 Camp Lejeune, yes.
19 Q. Okay. So the cause of her disease was not
20 unknown, then, correct?
21 A. Correct.
22 Q. Okay. How did you overcome the
23 possibility that her cause was unknown?
24 MR. TELAN: Object to the form.
25 THE WITNESS: In the presence of a

1 reasonably established cause, then by definition, I
2 can say that the cause is not unknown.
3 BY MR. GIBBONS:
4 Q. Okay. You indicated that the Camp Lejeune
5 water was a cause of her Parkinson's, but not
6 necessarily the only cause, correct?
7 A. I'm acknowledging that the standard in
8 this case allows for multiple etiologies and that
9 I'm here today to comment on the TCE and PCE.
10 Q. Okay.
11 A. And I considered other etiologies, and
12 Mrs. Rothschild doesn't have other, you know, known
13 exposures or causes of Parkinson's.
14 Q. Okay. And you're confident that
15 Ms. Rothschild's Parkinson's is not genetic in
16 nature?
17 A. Reasonably so, yes.
18 Q. Has Ms. Rothschild undergone any genetic
19 testing?
20 A. Not to my knowledge. Again, it wouldn't
21 be indicated in a person who is either the only
22 person in the family with Parkinson's or even if
23 someone had a single relative. This would not be a
24 situation where we would likely perform genetic
25 testing.

1 Q. Okay. If Ms. Rothschild had genetic
2 testing and it showed she had one of the genes
3 that's associated with increased risk of Parkinson's
4 disease, would you consider that to be more likely
5 than not the cause of her Parkinson's?
6 MR. TELAN: Object to the form of the
7 question.
8 THE WITNESS: It depends on the gene. And
9 this is where penetrants is relevant.
10 For the two most common genes that are
11 genetic factors for Parkinson's, the penetrants is
12 between approximately 19 and 30 percent. So less
13 than 50. Again, it's not a binary factor where, if
14 you have the gene, you get the disease.
15 BY MR. GIBBONS:
16 Q. And just for clarity, what were those two
17 genes that you were just referencing?
18 A. LRRK2, L-R-R-K-2, and G-B-A, and I discuss
19 those in my report in much more detail along with
20 their mechanisms and penetrants.
21 Q. Okay. Would you agree that occupational
22 exposure levels would be magnitudes higher than the
23 chemical levels present in the Camp Lejeune water
24 for TCE and PCE?
25 MR. TELAN: Object to the form.

1 THE WITNESS: Not necessarily.
2 BY MR. GIBBONS:
3 Q. Okay. For instance, in a dry cleaning
4 environment, would individuals that work in a dry
5 cleaner that used PCE-based solvents have a higher
6 occupational exposure than the levels of PCE in the
7 Camp Lejeune water?
8 MR. TELAN: Form objection.
9 THE WITNESS: I -- I would need more
10 details to answer that.
11 BY MR. GIBBONS:
12 Q. Okay. Your opinion is that the
13 Camp Lejeune water is the single risk factor that is
14 at least as likely as not the cause of
15 Ms. Rothschild's Parkinson's disease, correct?
16 A. I think we're saying the same thing, but I
17 would just clarify, I looked at TCE and/or PCE as
18 causes of Ms. Rothschild's Parkinsonism. And I
19 acknowledge that she does not have any other
20 significant exposures or risk factors that could
21 also be a cause of her Parkinson's.
22 Q. Okay.
23 A. And that if she did, it doesn't negate the
24 causality of her TCE and PCE exposure. The two
25 causes can exist.

1 Q. Okay. But you did not identify any other
2 causes, correct?
3 A. Correct.
4 Q. Okay. Are you confident that if
5 Ms. Rothschild had not been exposed to the water at
6 Camp Lejeune, she would not have developed
7 Parkinson's?
8 A. If she was not exposed to the water at
9 Camp Lejeune, she would be back in the bucket of the
10 general population where she would have a less than
11 1 percent -- where she would be in a group with less
12 than 1 percent Parkinson's incidence at her age of
13 onset. That's a very different situation.
14 Q. Okay. There are other people at
15 Camp Lejeune that also developed Parkinson's
16 disease, correct?
17 A. Yes.
18 Q. Even if they weren't exposed to the water?
19 MR. TELAN: Object to the form of the
20 question.
21 THE WITNESS: I don't know that.
22 BY MR. GIBBONS:
23 Q. Okay. The presence of risk factors in a
24 patient doesn't necessarily preclude the possibility
25 that the precise cause of the patient's Parkinson's

1 disease is unknown, right?
2 A. Do you mind repeating that?
3 Q. Sure.
4 The presence of a potential risk factor in
5 a patient doesn't preclude the possibility that the
6 patient's Parkinson's disease is still unknown,
7 correct?
8 MR. TELAN: Object to the form.
9 THE WITNESS: To emphasize what I said
10 earlier about our archaic use of the term
11 "idiopathic," we might still generally know that
12 Parkinson's for most people is a mix of genetic
13 and/or environmental factors, along with other
14 baseline risk factors, like age and gender, even if
15 we can't specifically identify a cause for an
16 individual person.
17 BY MR. GIBBONS:
18 Q. Okay. Have you conducted independent
19 medical evaluations in prior cases?
20 A. Yes.
21 Q. How many?
22 A. Probably 30 to 40.
23 Q. Were any of those of individuals with
24 Parkinson's disease?
25 A. Most of them were, yes.

1 Q. Do you typically conduct these exams
2 virtually or in person?
3 A. It's a mix.
4 Q. Okay. Can you give me an estimated
5 breakdown between the two?
6 A. If the patients happen to be in
7 California, which is where I live, I have always
8 examined them in person. If patients live across
9 the country, it could be -- and I'm purely
10 speculating -- a 50/50 mix, depending on the
11 specifics of the case where an uncertain diagnosis,
12 a recent diagnosis, less thorough documentation,
13 treatment by a nonspecialist, might push me more
14 towards an in-person exam where I can see them
15 face-to-face. And the converse is also true. If
16 the diagnosis is well-established by a reputable
17 movement disorder specialist, it's been a lengthy
18 diagnosis, there's a solid reputable medical record,
19 then the in-person exam doesn't add to my diagnostic
20 accuracy.
21 Q. Do you believe that there are limitations
22 in conducting an exam remotely, then?
23 A. There, of course, are a couple things that
24 we omit in a -- in an exam over video, but they
25 don't necessarily limit or -- limit the exam.

1 Q. Okay. What kinds of things would you not
2 be able to conduct via a remote examination?
3 A. And this is true for my clinical research
4 as well. And this is kind of well established
5 practice now.
6 But we omit muscle tone because that must
7 be felt by an examiner, and we omit a backwards pull
8 test. But the -- the vast majority of the exam is
9 still adequately performed by observation.
10 Q. Can you walk me through your typical
11 process for an independent medical examination of a
12 Parkinson's disease patient?
13 A. Sure.
14 Just to make sure I'm respecting your time
15 in answering accurately, could you be just a bit
16 more specific?
17 Q. Sure.
18 What are the specific tests and
19 evaluations that you perform during a medical
20 evaluation?
21 A. I generally start with a general physical
22 exam, if I can. Again, it doesn't impact my
23 Parkinson's diagnosis, but I might observe general
24 medical systems.
25 Then -- and I'm going from broad to

1 specific, and you asked about a Parkinson's exam,
2 but, of course, I'll always be more thorough when
3 possible. I might add a general neurologic exam, of
4 which there's some overlap with Parkinson's, but
5 would include examining the cranial nerves,
6 strength, sensation, and so forth, gait.
7 But a good Parkinson's exam is guided by
8 the unified Parkinson's disease rating scale, and
9 specifically part 3, which is the motor sub score.
10 I've done this for Mrs. Rothschild. I've included
11 it in my report. Briefly, it addresses the main
12 pillars of Parkinson's motor function, including
13 tone, tremor, bradykinesia, gait and -- well,
14 there's a bit of overlap here. Body bradykinesia,
15 dyskinesia and a Hoehn and Yahr staging score.
16 Q. Okay. Is the Montreal cognitive
17 assessment also part of that?
18 A. A Montreal cognitive assessment is a
19 memory assessment, and as part of a general
20 neurologic exam, I will assess mental status, which
21 gets at the same thing, either with questions that
22 test mental status or -- and/or a formal MoCA, as
23 you said.
24 Q. Okay. Are there any other steps that
25 you'd normally perform in a Parkinson's IME?

1 A. Let me just take a peek at my report so I
2 don't miss anything.
3 Page 19. I clarified that you were asking
4 about exam maneuvers, but, of course, I interview
5 the patient and take a thorough history. That, of
6 course, includes a chronology, a focus Parkinson's
7 motor and non-motor review, review of their imaging,
8 their labs, review of systems, social history,
9 family history. And I think I've touched on the --
10 the main points of the Parkinson's exam.
11 Q. Okay. How long do your typical IMEs last?
12 A. It depends on, not surprisingly, the --
13 sometimes, the mental status of the patient and
14 the -- the speed and clarity in which they or their
15 informant is able to kind of complete the history
16 with me. With Mrs. Rothschild, I spent over an
17 hour. And that's fairly typical.
18 Q. Okay. Do you have a standard set of
19 questions that you ask in your IME for the social
20 history?
21 A. They're not written out, but as I've
22 walked through them earlier, I -- yes, typically ask
23 about -- I'm happy to repeat them, but ask about all
24 the factors I listed earlier.
25 Q. Okay. Do you typically permit another

1 person to be in the room when you're performing an
2 exam?
3 A. If the patient agrees, yes.
4 Q. Do you ever find that it causes
5 distractions?
6 A. I have not run into that, no.
7 Q. Do you typically perform a differential
8 diagnosis when you're conducting an IME?
9 A. I do, yes.
10 Q. As you've stated previously and in your
11 report, you did conduct an IME of Ms. Rothschild,
12 correct?
13 A. Correct.
14 Q. Have you ever performed an IME of
15 Mr. Micalane?
16 A. No.
17 Q. Mr. Peterson?
18 A. No.
19 Q. Mr. Sparks?
20 A. No.
21 Q. Mr. Welch?
22 A. No.
23 I'm assuming these are other plaintiffs in
24 this litigation?
25 Q. You are correct, they are.

1 A. Okay. Then definitely no.
2 Q. Okay. Your examination of Ms. Rothschild
3 was conducted virtually, correct?
4 A. Correct.
5 Q. Was anyone else present with
6 Ms. Rothschild when you examined her?
7 A. Yes. Her long-time friend,
8 Mr. Gary Smith, was present.
9 Q. Did he answer any questions that you
10 asked?
11 A. He did help provide collateral
12 information. As I note in my report, and I'm
13 quoting now (as read):
14 As his knowledge of her
15 symptoms and history dates back
16 decades.
17 Q. Do you think his presence had any impact
18 on Ms. Rothschild's answers?
19 A. No. Other than adding clarity where --
20 where her answers were -- were helped by additional
21 information.
22 Q. Okay. I believe you said Ms. Rothschild's
23 IME was over an hour long, correct?
24 A. Correct.
25 Q. Okay. Did you take any notes during your

1 IME of Ms. Rothschild?
2 A. I directly typed Mrs. Rothschild's
3 responses and my observations into the draft of my
4 report. So there's nothing separate.
5 Q. Okay. So they're contained within the
6 report predominantly within this section of your
7 evaluation?
8 A. Correct. They're contained in the report
9 starting on page 19.
10 Q. Okay. Did you remove any notes when you
11 were revising drafts?
12 A. No.
13 Q. Okay. Would you agree that
14 Ms. Rothschild's Parkinson's disease treatment up
15 until now was reasonable and medically necessary?
16 A. Yes.
17 Q. Would you agree that Dr. Perlmutter has
18 treated Dr. Rothschild's Parkinson's disease
19 appropriately?
20 A. Yes.
21 Q. On page 36 in your report, let's see --
22 sorry.
23 Correction, page 35 of your report, the
24 first paragraph in a progression and prognosis of
25 Ms. Rothschild motor and non-motor symptoms, you

1 wrote (as read):
2 Ms. Rothschild has experienced
3 a progressive degeneration with
4 more rapid deterioration in recent
5 months.
6 Did I read that correctly?
7 A. Yes.
8 Q. Okay. What did you mean by "more rapid
9 deterioration"?
10 A. Meaning that as she described to me and as
11 I read from Dr. Perlmutter's notes, but mostly based
12 on what she reported to me, her symptoms in the last
13 year, in the last months, have been accumulating
14 faster than they have over the past 20-plus years.
15 Q. Okay. And Ms. Rothschild has had
16 Parkinson's disease symptoms for approximately
17 25 years; is that correct?
18 A. That's correct.
19 Q. Okay.
20 A. And that's not unusual in Parkinson's to
21 see an uptick in symptom accumulation in later
22 stages.
23 Q. Okay. Would you agree with
24 Dr. Perlmutter's assessment that Ms. Rothschild's
25 Parkinson's disease was extremely slow progressing?

1 A. It was slow progressing in the beginning,
2 slower than now. And -- and that's something that
3 he or I would say to describe patients, and we would
4 still be describing patients in a -- in a typical
5 spectrum of Parkinson's disease, some peoples'
6 disease progresses slower and some progress faster.
7 Q. Okay. Did Dr. Rothschild display any
8 signs of dementia during your IME?
9 A. She did display some cognitive impairment.
10 The terminology we can -- we can discuss whether
11 it's mild cognitive impairment or dementia, but yes,
12 she did have cognitive impairment.
13 Q. How would you differentiate between
14 cognitive impairment and dementia?
15 A. There's a -- there's a couple ways, and --
16 and some people -- some institutions, including
17 where I trained and where Dr. Perlmutter still
18 practices, doesn't make the distinction. They would
19 say any abnormal level is dementia. But for others
20 who make the distinction, they may use cutoffs --
21 score cutoffs in a Montreal cognitive assessment or
22 Mini-Mental Status Exam of, say, 24 out of 30, for
23 example, to define mild cognitive impairment or
24 dementia.
25 Q. Okay.

1 A. There's other ways to assess, as I alluded
2 to, and that may be a more functional assessment.
3 Again, originating from Wash U and their dementia
4 research center, we might perform a CDR, or clinical
5 dementia rating, scale, which is not based on the
6 same tasks or questions as a MoCA, but is really
7 based on functional cognitive abilities in one's
8 daily life.
9 Q. Okay. Would you agree that genetic-caused
10 Parkinson's usually has a lower prevalence of
11 dementia?
12 A. It depends on which genetic mutations. I
13 think our best data is that -- is a study out of
14 Mayo Clinic that after 20 years of Parkinson's, most
15 people, greater than 90 percent, will have some
16 dementia. This is Parkinson's disease dementia
17 specifically, and that prevalence increases with
18 time.
19 Q. Okay. Is that specific to Parkinson's
20 caused by genetics or is that --
21 A. All matters.
22 Q. Okay. Do -- does genetically-caused
23 Parkinson's often have a later presentation of
24 dementia?
25 A. No.

1 Q. Okay.
2 MR. TELAN: You don't have to fill that
3 out today. We can do that later.
4 BY MR. GIBBONS:
5 Q. You reviewed the life care plan that was
6 prepared for Ms. Rothschild?
7 A. Yes, I have.
8 Q. You concluded that the treatment and care
9 set out in the life care plan was reasonably
10 medically necessary, correct?
11 A. Correct.
12 Q. Were you instructed to opine on future
13 care and treatment in this matter?
14 A. Yes, I was.
15 Q. What methodology did you use to determine
16 Ms. Rothschild's treatment and care plans were
17 reasonably necessary?
18 A. My -- my experience and professional
19 judgment as a movement disorder specialist.
20 Q. Okay. Do you have any expertise in
21 medical billing practices?
22 A. I code all of my patient encounters for
23 billing purposes.
24 Q. Okay. Did you have an opinion on the
25 amounts that were offered for the life care plan and

1 costs?
2 A. No, I do not.
3 Q. Okay. Do you believe Ms. Rothschild's a
4 candidate for deep brain stimulation?
5 A. She could be.
6 Q. Are there any risk factors due to her age?
7 A. Age alone, no. Really, risk factors are
8 determined based on other personal factors, but
9 there isn't a numerical age cutoff for DBS.
10 Q. Okay. Did you review any of
11 Ms. Rothschild's medical bills?
12 A. I can't remember if this was the case for
13 Mrs. Rothschild, but in general, when I receive a
14 batch of medical records to review, there may be
15 some coding statements mixed in. I don't know if
16 that was the case for Mrs. Rothschild and I don't
17 remember any specifics.
18 Q. When you see coding language within
19 medical records that you're reviewing, do you pay
20 any particular attention to them or are they just
21 incidentally in the records?
22 A. I look at them, but they don't impact my
23 medical opinion of the case.
24 Q. In your clinical practice, are you
25 ordinarily responsible for evaluating medical bills

1 of your patients?
2 A. No.
3 Q. Okay. Do your patients discuss medical
4 billing with you?
5 A. Not usually, no.
6 Q. Does your office usually have a separate
7 department that manages medical billing?
8 A. Yes.
9 Q. Do you have any oversight of that office?
10 A. I review their monthly statements, and I
11 employ them, yes.
12 Q. Okay. How frequently would you say you're
13 involved in that department's daily business
14 activities?
15 A. Monthly.
16 Q. In prior cases where you've served as an
17 expert witness, have you ever opined on whether a
18 party's medical bills were reasonably and medically
19 necessary?
20 A. Bills, no.
21 Q. Courses of treatment?
22 A. Yes.
23 Q. Have you ever testified in a trial before?
24 A. Yes, I have.
25 Q. How many?

1 A. Two.
2 Q. Who are the parties in that case?
3 A. The Santa Clara County District Attorney's
4 Office versus Li, L-I, and in the Nastasia Freeman
5 versus United States of America.
6 Q. The Nastasia Freeman case was part of the
7 Red Hill litigation, correct?
8 A. Yes. And I think you referred to it under
9 the Feindt.
10 Q. Correct. The Feindt versus United States?
11 A. This is the same case, yes.
12 Q. Were either of those cases involving
13 Parkinson's disease?
14 A. Yes.
15 Q. Which ones?
16 A. The People versus Li.
17 Q. When was People versus Li?
18 Sorry, let me clarify.
19 When did you testify in People versus Li?
20 A. I think 2019.
21 Q. Okay. And in the Feindt case?
22 A. 2024.
23 Q. Have you ever been involved in litigation
24 in your personal capacity?
25 A. No.

1 Q. Have you communicated with any of the
2 other experts in this case besides Dr. Reynolds?
3 A. No.
4 Q. Are there any answers to my questions that
5 you wish to change before we finish up today?
6 A. No. Thank you.
7 Q. Okay. Is there anything else that you'd
8 like to add?
9 A. No.
10 MR. TELAN: Object to the form.
11 MR. GIBBONS: Okay. All right. That
12 finishes my questions. At this time, I'll turn it
13 over to opposing counsel for redirect.
14 MR. TELAN: Okay. Let's take a quick
15 break. How much time on record do we have?
16 THE VIDEOGRAPHER: 6 hours 20 minutes.
17 MR. TELAN: All right.
18 THE VIDEOGRAPHER: And we're going off the
19 record at 5:01 p.m.
20 (Whereupon, a recess was taken from
21 5:01 p.m. to 5:09 p.m.)
22 THE VIDEOGRAPHER: We are back on the
23 record at 5:09 p.m.
24 / /
25 / /

1 EXAMINATION BY MR. TELAN
2 BY MR. TELAN:
3 Q. Dr. Andruska, just a few questions for
4 you.
5 In your review of Dr. Perlmutter's
6 records, did you see that he had documented, perhaps
7 somewhere around 2016, that Ms. Rothschild began to
8 fall more frequently?
9 MR. GIBBONS: Objection. Form.
10 THE WITNESS: If you don't mind, I'll just
11 double-check.
12 BY MR. TELAN:
13 Q. Sure.
14 A. He's certainly a good documentarian.
15 Around 2015, at least, she has problems
16 with walking and balance. Of course, walking was
17 one of her early presenting symptoms. She has --
18 BY MR. TELAN:
19 Q. Let me -- go ahead.
20 A. Pardon me. She has near falls in 2017.
21 She has problematic falls in 2018.
22 Q. Okay. Does that history or information
23 allow you to conclude that at least from a postural
24 instability standpoint, her Parkinson's disease is
25 advancing or progressing?

1 A. Yes.
2 Q. Dr. Perlmutter noted in September of 2018
3 that Ms. Rothschild had fallen about ten months ago
4 walking out of a grocery store and apparently broke
5 her pelvis, pubic bone and tailbone.
6 Do you see that note?
7 A. Yes, I did.
8 Q. Is it your opinion that the fall that
9 caused her to fracture her pelvis and tailbone was
10 as a natural consequence of her Parkinson's disease?
11 MR. GIBBONS: Objection. Form.
12 THE WITNESS: Likely, yes. In the sense
13 that Parkinson's typically causes loss of postural
14 reflexes, the ability to kind of recover from an
15 imbalance episode, and it's very typical for people
16 with Parkinson's to fall, especially in later
17 stages.
18 BY MR. TELAN:
19 Q. And is that an opinion you -- an opinion
20 that you hold to a reasonable degree of medical
21 probability?
22 A. Yes.
23 Q. You had mentioned generally that her falls
24 continued in that 2018 time frame?
25 MR. GIBBONS: Objection. Form.

1 THE WITNESS: Yes.
2 BY MR. TELAN:
3 Q. Okay. As she advances in age, does her
4 risk of falling increase, particularly as her
5 Parkinson's disease progresses?
6 A. Yes.
7 MR. GIBBONS: Objection to form.
8 BY MR. TELAN:
9 Q. You also noted, I think, in your report
10 Dr. Perlmutter's notes regarding Mrs. -- or
11 Ms. Rothschild's problems with dysphasia --
12 dysphasia; is that correct?
13 A. Yes.
14 Q. Is that -- also, is dysphasia a recognized
15 complication of Parkinson's disease?
16 A. Yes.
17 Q. And in her case, did you note that her
18 dysphasia was also getting worse over time?
19 A. Yes.
20 Q. And what is the risk of dysphasia
21 advancing as you saw it in Ms. Rothschild's records?
22 MR. TELAN: Objection. Form.
23 THE WITNESS: The risks are many. They
24 can include weight loss and failure to thrive
25 through decreased caloric intake. Decreased caloric

1 intake and weight loss predisposes people to poor
2 recovery from infections. There's the risk of
3 choking, of frank choking. There's also the risk of
4 aspiration and subsequent aspiration pneumonia.
5 BY MR. TELAN:
6 Q. Does the risk of choking increase as the
7 Parkinson's disease progresses?
8 A. Yes. Because the severity of the
9 dysphasia progresses also.
10 Q. In terms of your opinion regarding the
11 dysphasia that we just talked about, is that an
12 opinion that you hold to a reasonable degree of
13 medical certainty as well?
14 A. Yes.
15 Q. There are records in Dr. Perlmutter's
16 notes referencing her urinary difficulties.
17 Do you recall those?
18 A. Yes.
19 MR. GIBBONS: Objection. Form.
20 BY MR. TELAN:
21 Q. Is -- are the urinary issues that she is
22 suffering from, in your opinion, related to her
23 advancement of her Parkinson's disease?
24 A. It's likely, yes.
25 Q. Is that also an opinion you hold to a

1 reasonable degree of medical certainty?
2 A. Yes.
3 MR. GIBBONS: Objection. Form.
4 BY MR. TELAN:
5 Q. In terms of your experience clinically
6 with patients who suffer from Parkinson's disease,
7 has it been your experience that as patients age and
8 their Parkinson's disease progresses, that they do
9 suffer from bouts of depression and anxiety over the
10 advancement of their disease?
11 MR. GIBBONS: Objection. Form.
12 THE WITNESS: They can, yes. You're
13 alluding to a situational depression where patients
14 are distraught or depressed about the limitations of
15 their disease. And there's also a recognized
16 prevalence of anxiety and depression as an inherent
17 non-motor symptom of Parkinson disease.
18 BY MR. TELAN:
19 Q. As to the opinions that you've offered
20 here today relative to Ms. Rothschild specifically
21 that her Parkinson's disease is at least as likely
22 as not a result of her exposure to the water at
23 Camp Lejeune, when you use the term "water" in that
24 context, are you referencing her exposure
25 specifically to PCE primarily and TCE secondarily?

1 A. Yes.
2 MR. GIBBONS: Objection. Form.
3 MR. TELAN: Thanks for your time. Those
4 are all the questions I've got.
5 And we'll read.
6 THE VIDEOGRAPHER: Anything else?
7 MS. PLATT: Yes.
8 MR. GIBBONS: I'm just going to reiterate
9 that I'd like to leave the deposition open because
10 of the late production we received last night on the
11 reliance materials. Any questions that I have would
12 be relatively short and brief, but I'm going to
13 leave the option open.
14 MR. TELAN: I'd object to it, just as
15 previously stated.
16 MR. GIBBONS: Okay. No further questions.
17 Thank you for your time, Dr. Andruska.
18 THE VIDEOGRAPHER: And keep your mics in
19 place for just a moment.
20 We have reached the end of today's
21 testimony, and we are going off the record today,
22 July 8th, 2025. And the time is 5:18 p.m. Thank
23 you.
24 (Whereupon, the deposition adjourned at
25 5:18 p.m.)

1 I declare under penalty of perjury the
2 foregoing is true and correct. Subscribed at
3 ______________________, ________________________,
CITY STATE
4
this ___ day of ____________, 2025.
5
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6 WITNESS SIGNATURE
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1 CERTIFICATE OF REPORTER
2 I, Kathleen A. Maltbie, Certified
3 Shorthand Reporter licensed in the State of
4 California, License No. 10068, the State of Nevada,
5 CCR 995, and the State of Texas, CSR 12212, hereby
6 certify that deponent was by me first duly sworn,
7 and the foregoing testimony was reported by me and
8 was thereafter transcribed with computer-aided
9 transcription; that the foregoing is a full,
10 complete, and true record of proceedings.
11 I further certify that I am not of counsel
12 or attorney for either or any of the parties in the
13 foregoing proceeding and caption named or in any way
14 interested in the outcome of the cause in said
15 caption.
16 The dismantling, unsealing, or unbinding
17 of the original transcript will render the
18 reporter's certificates null and void.
19 In witness whereof, I have hereunto set my
20 hand this day:
21 _______ Reading and Signing was requested.
_______ Reading and Signing was waived.
22 ___x___ Re[illegible]ested.
23 Kathleen Maltbie
____
KATHLEEN A. MALTBIE
24 RPR-RMR-CRR-CCRR-CLR-CRC-RDR
California CSR 10068, Nevada CCR 995
25 Texas CSR 12212

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0.05 70:13,24
0.05. 141:5 187:10
0.050 140:12
0.053 140:11
0.3. 148:14
0.97 139:12 141:11
00858 5:18
00897 1:6

1

1 5:8 13:23 14:3 87:8 147:6,8 162:24 165:8 197:9 198:19 225:11 225:12
1,265 197:5 198:5
1.0 87:4
1.1 87:18 112:3 112:9 122:5
1.10 111:22
1.15 121:19
1.2 87:19,21 88:1,2 138:24 139:1 141:13
1.227. 197:13
1.7 200:4
10 5:4 6:20 27:1 125:20,23 218:21
10,000 26:10 166:2
10.5 139:11 140:11
10.5. 141:8
100 43:1 56:6 85:25
10068 1:24 9:19 249:4,24
10:04 46:19,21
10:15 46:21,23
11 7:3 118:7 119:1,23 127:15 132:16 132:17 175:7 186:6
11,000 163:5
1100 3:19
113 139:12 141:11
115 6:11
11:24 89:7,9
11:36 89:9,11
12 7:5 126:16 150:20,24 167:13
12212 1:25 249:5,25
123 6:15
125 6:20
12:44 131:18 131:20
13 5:8 6:9 7:8 124:13 157:8 157:12
132 7:3
1333 8:12
14 7:11 161:22 162:5
15 7:14 27:1 40:3 68:16 72:10 174:11 174:15
15.4 199:11,17 200:2
150 7:5
157 7:8
16 7:18 188:22 188:25
161 7:11
168,361 152:9
17 7:20 189:12 189:14
172,120 152:7
1734 159:17
174 7:14
188 7:18
189 7:20
19 223:12 230:3 233:9
1918 212:20
1939 131:9
1953 128:12
1972 116:20
1973 75:12
1975 75:12 154:7
198 132:25
1985 116:20 154:7
1987 128:13
1:26 131:20 132:2

2

2 5:12 15:2 22:14,17 44:21 45:18 112:18 113:19 121:12 197:21,23 223:18
2.0 88:4
2.0. 121:8
20 27:1 234:14 236:14 241:16
20005 3:19
2002 136:24
20044 4:11
2009 181:20,23
2012 68:24 69:19 79:25 80:7,8,24 81:7 81:24 98:12 132:20 136:2 138:22 139:19 140:1 145:13 145:22 146:13 146:23 147:1
2014 122:10 125:9 151:5 157:20

2014a 122:10 122:14 123:14 126:9,15
2014b 98:12 122:11 125:24 127:4,10
2015 78:17 79:4 242:15
2016 181:5 242:7
2017 6:10 57:21 83:12 97:10 99:7,13 105:4,7 106:1 106:14 114:18 146:3 162:7,8 242:20
2018 162:5 242:21 243:2 243:24
2019 240:20
202 3:21 4:7,12
2021 68:25
2023 67:23 68:14 72:8 122:15 150:16 150:25 151:4 153:19 157:17 157:23 158:4 199:20,22
2024 19:21 21:23 22:6 28:23 115:12 122:15 123:20 123:23 124:8 126:8 136:25 151:13 157:5 157:12,20 159:11 162:1 240:22
2025 1:14 2:4 5:18 8:1,9 29:5 247:22 248:4
20530-0001 4:5
21 174:20
219 3:3
22 5:12 174:21
23 5:15 7:21 174:21 189:16
24 148:23 235:22
242 5:5
245 197:5 198:5,14
246 3:5
2495 10:13
25 234:17
26 197:10
260 3:7
27 134:25 166:4 197:3
27603 3:12
27607 3:8
278-5852 3:5
28 5:19
29 176:23 217:15
29440 3:4
2:53 180:19,21

3

3 5:15 23:18,22 28:12 95:13,14 113:19 121:18 125:10 142:6 142:13 145:7 197:13,20 229:9
3.0 88:11
30 79:8 120:2 223:12 226:22 235:22
30s 207:13
31 120:3 217:15
32 166:10,14,15 166:22
32,000 166:9
32,153 166:6 198:12
32.7 163:18
33 138:24 139:1 141:13
340 4:10
35 233:23
36 233:21
366 67:24
37 215:18
3801 3:7
3:07 180:21,23

4

4 5:19 28:8,13 28:15 95:11,15 176:23
40 72:12 226:22
40.1 198:23
400 162:17 165:1 166:16
404 3:13
40s 207:15
43.76 198:24
45 115:1
47 115:1
49 169:18 172:7
4:03 215:22,24
4:17 215:24 216:1

5

5 5:22 26:15,15 93:7,13,17 94:11 116:13 119:23 186:2,6 197:9,20,23 199:6 215:18 218:20
50 41:25 163:11 165:9 168:2 223:13
50,000 163:7
50/50 227:10

51 161:8
512 3:16
514-2000 4:7
52 80:25
52.3 163:19
529-3351 3:9
54 147:21
55 110:12 111:5 122:17 161:1,13 206:23 207:6
55.3 159:19
57 220:8
5:01 241:19,21
5:09 241:21,23
5:18 247:22,25

6

6 5:18 6:3 93:8 93:13,18 94:11 95:19 96:2 112:13,17 113:21 121:2 186:10 241:16
6,177 198:15,18
6.1 138:23
60 161:1,13 182:24 207:6
62 147:21 190:1 198:22
6202 162:13
6204 163:14,25 164:1
6205 162:24 165:8
63 183:23
64 24:6 58:3 75:4
6434 249:23
660 10:13
674 152:5
679 156:12
690-0990 3:16

7

7 6:6 97:6,10 113:18 121:15 184:14
7,580 166:6 198:11
70 63:21 64:3,4 200:17 218:14
73,000 158:1
76 186:7
769 154:2
777 134:5,21
778 147:7
780 136:21 137:3
781 142:24
7:23 1:6 5:18

8

8 1:14 2:3 5:4 6:11 8:1 107:2 107:5,7,19 115:18,22
8,000 163:6
80 158:2 218:15
843 3:5
850 25:11
856-8254 3:21
880-0389 4:12
8th 8:9 247:22

9

9 6:15 111:20 112:1 123:10 123:13 148:25
90 236:15
90,000 25:15
900 3:11
915-8886 3:13
919 3:9
93 5:22 6:3
94 172:9
94.5 71:2
94039 10:14
95 70:25 108:11,21 138:24 139:11 172:9
950 4:5 25:11
97 6:6
97,000 25:16 26:23
99 97:23 98:7 99:15 109:16 112:12 114:10 146:2
995 1:24 249:5 249:24
9:08 8:1,10
9:48 2:4

a

a.m. 2:4 8:1,10 46:19,21,21,23 89:7,9,9,11
abbreviations 13:16
abilities 236:7
ability 11:13 13:3 20:21 41:2 49:12 56:8,20 104:24 243:14
able 13:6 83:21 179:5 228:2 230:15
abnormal 235:19
above 2:10 49:1 98:25 108:8 111:22 122:5 134:25 155:9 156:13 199:4 201:14 202:15
abreast 31:23 32:17
absence 60:6
absolute 148:13

absolutely 21:2
abstract 167:18
academic 57:6
acceptable 63:7 63:15 109:9 196:7
accepted 123:1 194:9
account 77:11 77:21 104:16 124:8,25 126:19 129:3 141:22 154:10
accounted 77:25 78:3 104:23
accounting 163:16
accumulate 170:13
accumulating 234:13
accumulation 234:21
accuracy 227:20
accurate 13:7 94:13 153:10 208:14 215:8
accurately 102:22 228:15
achieve 107:21
acid 177:12
acknowledge 141:7 224:19
acknowledged 106:20 127:10 148:12 149:19
acknowledging 146:18 222:7
act 50:2,8 52:8 53:16,21,25 54:3,10,13,21 55:15
action 2:12 46:10,14 106:8
active 119:7
activities 194:1 239:14
activity 164:20 165:4 170:18 171:11
actual 69:24,25 90:1 144:6 179:4
actually 151:25
add 14:22 19:2 50:21 106:2,9 114:14 125:8 155:1 167:9 216:7 227:19 229:3 241:8
adding 232:19
addition 145:8 211:9 212:2 219:22
additional 14:22 27:17 74:13 134:7 168:4 169:20 170:11 172:15 232:20
additive 74:14 81:22 82:15
address 10:12 31:19 64:17 136:12
addressed 52:12 141:23 142:23 144:20
addresses 229:11
adds 84:22
adequately 228:9
adjourned 247:24
adjust 25:2
adjusted 135:1
administering 90:11
administration 151:17 152:2 152:19 167:25
admission 149:22,23
admissions 149:10
admitted 100:24
adulthood 135:19
advancement 245:23 246:10
advances 244:3
advancing 208:4,5 242:25 244:21
affairs 47:16 105:8,21
affect 12:25 13:3 175:15 176:3 178:19 179:19 212:5
affected 147:10 147:19 148:2,8 148:18,22
affects 195:23
affirmative 11:23
aforemention... 191:18
age 159:12,19 160:14,22 162:16 205:3 206:17,20,23 206:25 207:2,4 207:6,9,22 208:1,4,5,6 225:12 226:14 238:6,7,9 244:3 246:7
agencies 78:10

agency 13:18 151:8
agent 37:11,14 114:23
agents 145:4,6
ago 11:2 23:9 40:18 243:3
agree 52:6 61:7 61:10 64:2 80:11 81:8 82:23 84:17 85:7 86:1,7 88:14 89:2,13 90:6 92:9,18 94:25 98:6 101:8,18 103:3 103:15,24 104:10 121:14 135:17 136:14 138:11 141:6 144:8 155:4 160:4 165:9 172:14,19 179:17 195:9 195:18 198:3 203:22 204:3 204:12 216:17 216:21 217:13 218:7,14 219:5 220:21 223:21 233:13,17 234:23 236:9
agreed 126:17
agreement 5:12 22:11,18 23:1 26:4,7,8
agrees 231:3
ah 67:14
ahead 180:16 242:19
aided 249:8
air 210:6
airplane 15:20
al 45:19 68:24 98:12,12 111:13 151:10 161:18
alcohol 38:22 119:5,18 208:20
alleged 20:10 20:18 71:14 72:4 129:25
allow 62:10 179:23 242:23
allowed 177:25
allows 26:18 222:8
alluded 236:1
alluding 246:13
amended 6:3 93:18
america 8:14 240:5
american 5:14
amino 164:8
amnesia 134:15 149:16
amount 26:16 26:19 57:1 66:7,24 70:13 190:22 191:23 192:1,5,9 194:24
amounts 237:25
analogous 82:10
analogy 115:7 169:15,24 170:18 171:22 173:7 183:13
analyses 78:22 80:20 89:21
analysis 7:14 33:1 83:15 89:15 112:23 116:16 120:10 153:9 163:15 173:17 174:4 175:11 183:22 184:8,11,13,17 184:21,23 185:19 186:21
analytic 151:16
analytical 152:17
analyze 86:7,18 88:14 185:13
analyzed 111:17 127:7 157:17
analyzing 92:10
anatomic 214:11
andruska 1:13 2:2,9 5:9,13,16 5:20 8:17 9:22 10:3,7 14:2,3 22:17 23:21 28:11 46:25 89:13 93:12 94:10 97:9 115:21 123:13 132:4,20 150:23 157:11 174:14 178:9 179:9 180:25 216:3 242:3 247:17
andruska's 6:4
animal 80:20 83:15 91:25 92:6 155:18 165:22 167:10 168:10
animals 64:14 64:16 162:9,13 167:16 184:16
anna 4:11
anna.e.ellison 4:12

announce 9:14
annual 26:12
answer 11:13 11:20 12:5,10 12:16 13:11,12 32:3,16 38:12 44:23 54:5 55:9 56:4 60:19,21 62:10 62:14 66:14 67:2 73:14,15 76:16 80:25 83:25 91:22 113:15 120:23 138:9 148:12 177:23 187:14 188:5,10,15 194:11 196:22 203:14 204:9 212:18 214:14 224:10 232:9
answered 56:3 73:6 193:20 200:20
answering 34:1 228:15
answers 13:7 14:20 68:13 232:18,20 241:4
anti 38:25 206:13 215:10 220:13
anxiety 246:9 246:16
anybody 19:13
apart 50:15 69:22 74:2
apparent 143:9
apparently 148:20 243:4
appearances 3:1 4:1
appeared 2:9
appears 163:5
appendix 5:22 7:21 93:17,18 94:10 189:16 189:17
applicable 114:6
application 86:15 183:21 184:6 185:13 186:21
applications 184:3
applied 47:5
apply 53:17 57:13,16 61:4 194:13 211:22
applying 130:25 221:9
appropriate 14:17 53:17 86:15 117:8
appropriately 233:19
approximate 41:3 165:22
approximately 25:15 26:15 27:8 41:25 63:20 68:15 163:6,10 165:9 200:17 223:12 234:16
apt 205:18
arbitrary 140:17
archaic 226:10
area 128:3,8
arena 203:18
argumentative 65:13
army 37:12
arrive 61:5
article 32:8 141:24 164:11 174:20,21 177:4,9 178:2 179:24 180:1,2 181:11,12,13
articles 32:2 33:25 71:10 181:14
ascertain 36:17 196:19
aside 219:4
asked 12:15 33:7 56:3 73:5 100:19 177:24 178:23 200:19 203:10 208:25 216:8 229:1 232:10
asking 28:4 37:8 45:2,24 49:7 67:12 78:21 83:8 101:3 102:4 132:7 176:13 178:24 179:2,3 193:13 202:5 230:3
asks 54:5
aspects 91:5 184:18
aspiration 245:4,4
assembled 151:8
assertion 110:3
assess 102:23 106:15 107:13 229:20 236:1
assessed 118:16 126:12
assesses 124:3
assessment 6:7 7:13 35:4,11 35:15 53:18 77:21 80:9

86:19 88:25 97:10 105:4,7 106:1,14 107:3 107:8 109:21 111:21 112:14 125:11 145:12 153:15 156:10 161:19 194:6 229:17,18,19 234:24 235:21 236:2
assessments 35:8 99:9 157:4
assessor 34:23 34:25 35:1
assignment 116:8,9 127:12
assist 24:1
associated 45:17 101:10 101:20 102:1 102:17 104:1 104:12 124:17 140:2,6 142:18 150:2 209:11 212:12,15 223:3
association 82:23 83:3,20 86:22,25 87:5 87:12 107:23 139:21 140:22 144:19 155:21 208:23
associations 64:7 156:21 183:11 211:20
assume 12:6
assumed 116:7 127:25
assuming 53:10 129:13 130:12 153:2 231:23
assumption 128:6 129:10
assumptions 200:24 201:4
atom 169:21,22 170:11 172:15 173:7 175:13 176:2 177:20 178:18 179:18
atsdr 6:6 13:19 48:17 50:15 57:21 78:20 83:12 87:17,23 97:3,10 98:24 99:10 100:1,18 104:15,21,22 105:4,7,20 106:14 107:2 107:24 111:3 111:20 112:13 112:14 113:3 113:10 114:17 145:23 146:3 146:13 151:10 158:23 172:12 197:11,14
atsdr's 125:11 145:12
attached 26:7
attachment 14:11 15:2
attempt 219:15
attempted 141:25
attending 18:13
attention 114:25 119:1 238:20
attorney 10:17 13:10 16:18 30:5 54:8 249:12
attorney's 240:3
attorneys 17:10
attributable 219:6
atypical 220:16
author 100:16 100:19 118:23
author's 32:10 125:10 151:25
authored 17:2 115:10 131:13 196:15
authority 110:2
authors 33:11 85:12 111:10 111:12 120:10 122:1 133:17 135:12 136:6 141:2,6 149:13 164:19
autopsy 208:9 214:23
autosomal 209:21,21
available 49:13 56:8 99:18 102:11,13,15
avenue 4:5
average 160:22 160:25 161:13 201:8 207:6
averaged 159:19
aware 19:2,5 19:17 20:9,17 30:21 40:12 57:12,14 61:13 63:3,5,6,10,24 67:15 75:19 81:21 97:17 99:10 100:9,15 100:17,22 101:4 104:14 104:19 105:2,5 105:6,17,19,25 106:7 109:4,8 117:3,5,9

128:11,14,16 128:19,22 129:24 130:2 160:17 168:20 172:21 181:19 181:22 183:2 183:20 194:4,8 194:12 195:5 200:23,25 201:3,7,11 207:25 210:14 213:13,19
axis 162:25 163:2

b

b 4:6 122:14 223:18
back 17:5 18:25 19:9 39:11 46:22 72:16 76:7 80:6,23 83:11 86:5 89:10 109:19 111:19 112:12 114:9 132:1,4 136:22 148:4,21 164:22 169:17 180:22 181:4 198:21 210:3 212:22 215:25 216:4 225:9 232:15 241:22
background 20:4 28:25 29:17,21 36:14 36:25 54:16
backwards 228:7
balance 242:16
banned 66:13 136:23,25
bar 162:25 165:5
barrier 173:11
basal 214:11
base 6:14,18,22 7:7 65:17 72:9 72:13 75:6,12 75:22 79:6 104:17 115:15 127:12 128:4 129:6 150:17 192:24 193:12 194:2 200:3,3
based 49:13 62:9 72:17 76:21 81:6 82:12 85:9 110:21 116:7 123:5 126:4 130:13 133:10 133:20 135:9 143:3 153:19 157:19 158:11 158:15 161:6 177:2 179:16 180:8 191:2 216:15 224:5 234:11 236:5,7 238:8
baseline 226:14
bases 36:19
bash 3:15
basing 56:25
basis 29:16 154:1 220:17
batch 238:14
bayman 184:20
bayshore 8:12
bear 181:8
began 161:4 242:7
beginning 12:10 119:24 132:10 235:1
begins 118:7
behalf 8:22 9:1 27:21
behave 169:12
behaviors 206:10
believe 22:9 25:15 44:21 45:8 50:6,19 68:25 69:13,16 74:5 75:12 79:4 95:11 96:16 97:19 101:24 114:9 116:4 130:6 141:2 142:7,23 145:15,20 160:11 161:6 164:12 166:2 169:18 174:10 186:3,8 196:4 205:6 227:21 232:22 238:3
bell 3:3
belllegalgrou... 3:5
ben 4:10
benzene 36:5 80:13 156:19 188:9 191:8 192:6
best 11:13 20:21 22:5 41:2 43:4 49:11,12 56:8 56:20 57:23 87:23 94:15 104:24 135:12 236:13
better 32:3 36:25 91:7 149:9,10 206:11 221:6
beyond 194:17
bias 127:21 138:7,7 144:6 154:23 206:10
biased 135:14 138:4 144:9

biasing 124:19 144:25
big 173:5
billed 25:13
billing 237:21 237:23 239:4,7
billion 62:18 194:16 198:24 199:6,11,18
bills 238:11,25 239:18,20
binary 140:15 223:13
biochemistry 30:2 171:7
biologic 115:4 154:8
biological 183:12
biome 214:4
bit 18:10 19:16 36:25 38:1 74:4 82:20 197:22 199:25 210:4,8 228:15 229:14
block 215:4
blocks 207:2
blood 173:10
board 5:14
body 46:11 62:19 79:16,18 229:14
boehme 184:2 185:20 186:22
bone 243:5
boone 3:7
bottom 111:25 112:17 116:14 127:16 134:20 142:24 147:8 147:14 217:18
boulevard 128:17
bouts 246:9
bove 98:12 100:15 105:25 110:18 111:12 115:10 118:23 119:12,20 122:7 123:20 125:9 126:8,8 127:4,10 129:8 129:20 151:5 151:10 152:14 153:23 157:20 158:6
bove's 115:12 125:24 151:13
box 4:10
boyer 4:15 9:11
bracket 207:4
bradford 182:14,17 183:4,16,18 184:4,6,19,20 184:23 185:7 185:14 186:21 186:25
bradykinesia 229:13,14
brain 82:6 170:13 173:11 177:15 213:13 213:16 214:12 214:25 215:5 238:4
branch 4:4 36:18,22 37:6
breadth 119:20
break 12:14,16 46:17 89:4 131:15 180:17 207:2 215:20 216:3 241:15
breakdown 227:5
breaks 12:13
brief 20:3,5 247:12
briefly 192:17 216:13 229:11
broad 32:25 73:9 136:18 171:18 173:12 211:6 228:25
broader 78:23 89:22 203:5 212:23
broadly 58:5 59:8,9 207:17 212:22
broke 132:10 243:4
brought 53:21 78:25
bryson 3:10
bucket 225:9
build 105:20
burden 52:13 52:14 54:22
burlingame 8:12
business 10:11 239:13

c

c 8:2 167:22
cable 82:20
calculate 65:21 122:1 166:8 191:22 192:1,5 192:9
calculated 74:14 165:25
calculating 194:22
calculation 196:2
calculations 125:17 145:9 188:19 189:5,8 189:9,24 192:20 198:7

calibrated 95:1
california 1:24 2:8 8:13 9:20 10:14 16:10 227:7 249:4,24
call 41:14 51:5 114:25 208:13
called 19:14 68:1 160:25 173:17 181:5
calling 99:6
calls 156:6
caloric 244:25 244:25
camp 1:6 6:8 6:14,18,22 7:7 7:10 10:19 17:24 19:17 20:10,15,18,22 21:19 23:7 36:9 37:18 50:2,8 52:8 53:16 54:3,9 54:13,21 55:15 58:7 59:1,25 64:3,21 65:17 65:19 74:6,17 74:23,24 75:10 75:25 76:12 77:18 78:18 79:8 89:25 104:16,20 105:10,14 115:16,23,24 116:1,5,10,22 116:24 117:3,6 117:7,10,13,13 117:22,22 118:2,2 119:8 119:9 120:2,3 120:6,6,14,14 120:15 121:4,4 121:7,8,20 122:9 123:15 125:25 128:1 128:12,20 131:6 145:13 150:18 152:9 152:10 153:20 153:20 154:7 154:12,22 156:15 157:14 158:17,18,19 158:22 159:7 165:19 181:20 190:6 191:24 192:3,7,11,14 192:18,23 193:1,3,15,18 199:3 221:15 221:18 222:4 223:23 224:7 224:13 225:6,9 225:15 246:23
cancers 6:9 119:13 177:8
candidate 238:4
cannon 184:2 184:20 186:23
cannon's 185:20
capacity 240:24
caption 249:13 249:15
carbon 136:15 137:7
care 19:3,4 40:20 41:10 151:21 206:10 237:5,8,9,13,16 237:25
career 39:18
careful 165:4
carolina 1:2 3:4 3:8,12 8:16 10:20
carry 92:11,14 92:24
case 8:14 15:4 19:21 20:4,5,7 20:16 22:25 23:4,11 24:12 24:19 25:1,4,7 25:14,19 26:4 26:24 27:5 42:16 43:6,7 43:17,22 44:13 44:24 45:16,20 45:22 46:6 52:23,25 53:21 57:2,2 59:10 60:21 61:4 69:12,19,22 71:14 72:5 78:25 85:20 87:7 88:22 89:24 90:17,20 90:20,23 91:7 91:17,24 92:11 92:14 114:6 119:17 128:15 143:14,25 144:15 177:11 193:5 203:1 217:2 219:2 220:17,17 222:8 227:11 238:12,16,23 240:2,6,11,21 241:2 244:17
cases 1:8 27:3,4 27:7,12,24 28:1 43:13 44:2,3,4,15 45:10 52:19 71:20 74:2 91:15 124:5 142:2 145:7,21 203:18 218:15 218:21 219:5 226:19 239:16 240:12
catalyzes 115:5

categorically 204:13
categorized 116:22
category 212:24
causal 55:3,4 55:17 96:19 107:23
causality 106:16 107:13 219:2 224:24
causation 17:2 54:3,17 59:7 59:11,13,16,18 61:1 64:12 81:10 82:24 83:1,8 84:4 92:10 98:14 99:1 100:2 155:13 182:18 182:23 183:10 187:1 196:8
causative 64:13 205:10
cause 2:10 37:21 42:2,7 55:20,21 57:20 58:1,8,10,13,20 58:22 59:2 60:1,7,14,16,22 61:2 62:6 65:1 65:12,20 66:8 66:18,24 67:6 71:13 74:7 76:2,14,19,23 78:13 80:5 81:17 83:22 85:25 121:17 122:22 160:4 169:12 173:12 199:12,18,23 200:8,9,10 202:5,6 204:3 204:8,19,22,25 205:19 206:2 210:20 211:2,8 214:15,19 216:21 218:9 218:16 219:6,9 219:12,13,15 221:13,16,19 221:23 222:1,2 222:5,6 223:5 224:14,21 225:25 226:15 249:14
caused 37:25 38:3,12 72:3 85:21,25 236:9 236:20,22 243:9
causes 42:22 51:6,7,21,25 52:1,2 57:18 82:10 205:2 217:5 218:24 219:19,20,21 220:2,21,22 221:1,7 222:13 224:18,25 225:2 231:4 243:13
causing 23:8 82:20 97:18 114:23 115:6 212:20
caution 116:17
cautionary 20:25 21:10
caveat 136:23
caveats 198:20
ccr 1:24 249:5 249:24
ccrr 1:25 2:6 249:24
cdr 236:4
cell 163:4,16 165:6
cells 163:1,3,11
cellular 64:17 92:6
censure 46:10
center 236:4
central 197:10 197:12
certain 38:25 56:6 64:18 70:13 102:23 122:24 150:6 153:8,8 154:5 206:15 208:9 209:5 211:1,1 212:9,11,14 220:13,15
certainly 32:1 36:10 37:24 41:16 139:5 169:5 194:15 203:17,18 213:13 242:14
certainty 43:1 49:2,8,11 56:1 56:9,12 57:6 86:6 221:12 245:13 246:1
certificate 249:1
certificates 249:18
certifications 30:12 34:9 35:3
certified 2:6 9:18 249:2
certify 249:6,11
cessation 168:13,15
cetera 80:20 183:13
chance 70:25 71:1,3 85:3,9 93:24 209:20
change 29:4 114:21 178:6 180:9 241:5

changes 129:22 220:10
characteristics 172:20,23
characterize 200:7
characterized 80:17
charge 25:9
chart 197:13,20 197:20,21,23 197:23 198:19
charts 190:3,7 197:9
chat 94:5
check 123:22 124:10 142:4 166:3 182:4 242:11
chemical 61:19 63:16 66:12 90:14 101:9,19 101:25 103:25 104:11 123:24 126:9 144:23 160:4 169:3 171:12 212:9 223:23
chemically 170:17
chemicals 23:6 70:5 73:11,17 117:9,15 129:25 190:6 194:5 210:17 210:22 211:1,9 212:4,8
chief 29:2
chloral 177:13
chloride 36:1 39:9,10 80:13 156:18 187:18 187:25 188:1 191:13 192:10
chlorinated 68:18 73:25 80:17 96:14 159:3 188:4
chlorine 169:21 170:11 175:14 176:3 177:20 178:19 179:19
choice 90:18
choking 245:3 245:3,6
choose 53:11 209:7
chose 146:22
chronic 119:13 149:11
chronology 230:6
cichocki 181:9
cigarette 147:12,17
cindy 4:6 9:9
cindy.m.hurt 4:7
cir 109:21 110:14 111:8
circumstances 61:3
cirs 110:23
citation 110:4 172:5
cite 69:20 171:20
cited 17:9 64:6 71:8 79:5,10 79:16 87:24 110:2 111:12 122:17 145:21 157:5 161:17 181:14
city 248:3
civil 4:4
civilian 6:12,21 115:14 121:16 123:19 197:12
civilians 121:11 125:24
claims 11:10 47:19 48:5 53:21,25
clara 240:3
clarification 79:13 117:23
clarified 230:3
clarify 12:4 44:1 58:6 74:21 149:13 149:17 161:25 189:7 194:19 196:5 205:3 212:23 214:10 224:17 240:18
clarifying 112:3
clarity 12:9 24:16 220:8 223:16 230:14 232:19
class 68:18 73:25 173:9 188:4 209:1
cleaner 224:5
cleaners 39:21
cleaning 39:12 40:13 224:3
clear 12:19 82:14 167:9 194:23 208:4
clearer 212:17
clearly 11:20
clinic 183:17 219:10 236:14
clinical 16:14 31:13,25 34:5 36:7 38:10 39:4,17 42:5 42:20 48:18 49:5,15 50:13 50:16 51:13,16 55:20 57:8,21 57:23 83:13 109:4 143:8

146:6 183:7 188:16 196:19 196:25 213:17 215:1 216:25 217:8 219:14 228:3 236:4 238:24
clinically 41:5 213:17 246:5
closely 113:15 169:19
closeness 40:10
clr 1:25 2:6 249:24
code 237:22
coding 238:15 238:18
cognitive 135:2 229:16,18 235:9,11,12,14 235:21,23 236:7
cognitively 133:13 134:17 134:24
coherence 183:13
cohort 6:14,19 6:23 62:19 69:12,13,23 80:19 90:8,15 90:16,19,23 91:4,22 92:10 92:13,24 115:16,24,24 123:20 151:15 151:16,25 152:14,17,25 153:3,7,24 154:22 155:3 159:23 160:3
cohorts 64:5 92:4 118:2 119:25 151:7 152:7
coleman 3:10
collaborate 16:13
collateral 232:11
colleagues 41:12 110:19 129:9
collect 135:9
collected 133:21 134:8
college 35:13
column 119:2 134:6 137:5 154:2 164:3
combination 80:2,4 217:22
combinations 75:6
come 17:5 18:25 24:22 26:3 31:24 32:2,5 37:23 39:13 40:23 41:13 49:24 72:16 80:6 164:22 173:16 181:5 216:4
comers 149:8
comes 25:1 37:24 160:1
comfortable 30:3 76:22 205:21
coming 50:21 145:17
commencing 2:4
commensurate 197:11
comment 39:11 177:2 222:9
commented 45:2 87:25
comments 155:8
commercial 152:3
common 13:16 41:15 48:21 82:9 114:22 115:3 136:19 136:22 146:18 149:3 171:22 171:23 207:14 207:18,19 209:18 223:10
commonly 170:23
communicated 241:1
communicati... 15:5,24 16:17 17:6,23 18:1
community 106:7
comorbid 168:4
comorbidities 212:11
compacta 214:18
compar 174:3
comparable 92:4
comparative 7:14 173:17 174:4 175:11
compare 65:23 117:12 172:17
compared 91:25 115:23 121:8 147:24 148:9 163:21 170:11 172:16
compares 165:18
comparing 121:16
comparison 91:6 173:4

compensate 105:9
compensation 25:6
complete 23:10 28:24 94:12 230:15 249:10
completed 18:21 22:19
completely 52:4 66:13
complex 149:25
complication 159:15 244:15
complications 159:9
compound 60:18
compounds 156:19,20 157:3 180:3
comprehensive 38:21
computer 249:8
concentration 63:16 66:2 67:24 68:15 75:17 78:7 104:5,8 118:9 156:16 194:24 195:3,11 201:14
concentrations 62:20 63:8,11 64:20 70:5 71:19 72:10 75:8 77:16,22 198:23 200:16 201:24
concerned 38:16
concerns 40:23
conclude 55:2,4 55:16 83:21 195:22 242:23
concluded 145:23 146:13 221:13 237:8
concludes 98:24 100:1
conclusion 84:4 98:7 102:12 175:9 178:25 179:4
conclusions 59:14 61:5 75:10 146:23 156:3,13 173:14 175:8 178:18 179:3 179:16 180:6
conditions 13:2
conduct 31:15 33:1 54:12 65:7 185:7 227:1 228:2 231:11
conducted 31:11 33:20 67:17 68:8 100:13 104:15 226:18 232:3
conducting 42:13 227:22 231:8
conference 15:21 16:7
conferences 15:16,25
confidence 70:13,19 88:15 88:20,24 107:9 107:25 108:1,4 108:11,22 109:1,10 110:6 111:11 137:11 137:16,21 138:2,12,12,19 138:24 139:1 139:11 141:10 141:13
confident 222:14 225:4
conflicts 33:11 33:17
confound 91:8
confounders 91:9 124:18
confounding 69:5
conjugation 175:17
conjunction 20:22 44:8 195:24
connected 211:4
connection 22:24 25:7 27:15 33:7 35:18,25 36:4 37:11 78:19 79:7 105:13 156:4
cons 91:11
consciousness 134:14 149:10 149:16,19
consequence 243:10
consider 20:6,7 42:15,24 75:16 91:11 92:6 114:7 118:19 122:21 126:15 126:21 138:3,7 140:13,13,14 141:25 158:7 159:6 187:23 188:6,11 211:10 219:21 220:2,9 223:4
considerable 128:2

considerably 116:19
consideration 61:11 88:18 104:8 184:7 185:8
considerations 182:15,17 183:10,17,19 184:5,19 186:22
considered 6:4 27:24 69:9 73:19,22 75:20 78:3,17 79:12 79:17 89:16 93:19,23 108:18 111:23 111:24 112:4 112:14,16,24 113:11 114:3 119:19 132:11 145:11 147:3 161:14 174:9 181:15,16 182:8,9,22 185:1,21,24 187:5 193:8 202:4,10,15 206:17 208:20 210:6,24 211:16 213:8 213:23 215:5 222:11
considering 50:22,25 51:11 51:17 78:20 159:24 167:1 203:5 221:6
consistent 26:19 209:20
constant 26:17
constitute 26:25 201:14 201:25
consultation 40:17 41:14
consultations 41:4,9
consume 75:22
consumption 119:5,18 166:5 166:7 190:8 197:15,22
contact 20:2
contacted 19:19
contain 24:11 24:17 80:8
contained 156:17 233:5,8
contains 54:22
contaminant 62:23,25 118:9 130:9,11 156:7 199:5
contaminants 6:8 154:9 212:8
contaminated 6:13,17,21 7:10 58:7 74:24 104:16 115:15 130:1 130:19,20 156:1 157:14
contamination 116:18 125:12
contending 170:3
content 22:22
context 15:13 16:16 20:12 40:15 42:20 50:6 57:10 70:23 78:23 88:18 91:12 123:4 140:18 164:17 182:4 188:16 217:5 246:24
continue 132:9 177:16
continued 4:1 6:1 7:1 243:24
contrary 96:17
contribute 177:7
contributed 19:3 38:4 156:21
contributing 52:16 60:16
control 69:12 69:19,22 90:17 90:20,23 91:7 91:9,17,24 92:11,14 117:8 126:16
controlled 79:25 89:16 90:7
conversation 40:1
conversations 15:12 16:1 39:13 40:7,10
converse 227:15
conversion 115:5
copd 119:14
copy 22:21,22 52:8 54:9
corps 7:7 150:17
correct 12:20 14:9 17:3,4 18:23 19:7,8 22:2 23:13 24:7 26:8,9,10 26:11 28:20 29:8,13 30:5,6 30:7,8,10,11,14 30:17,25 31:1

31:4 34:7,8,11
34:16,24 35:19
35:20,22,23
36:1,2,5,6,9
39:8 44:6,9,10
44:12,14 45:4
46:1 47:1,6,7
47:14,23 48:12
51:22 52:2,3
53:2,3,5,6,8,22
53:23 54:23
55:5 58:1,4,20
61:5,6,8,14,15
61:20 62:24
64:15 67:22
70:2,3 72:18
72:21,22,24,25
73:18 74:8,9
74:18 76:2,3
76:15,16 77:18
77:19 78:8
83:4 84:19
85:3,15 86:8
86:13,19,22
87:5 88:13,25
92:17 95:17,18
99:3,4,12
100:4,21 102:8
102:17 103:17
106:16 108:1
109:23 110:1,3
110:7,8,11,17
110:18,23,24
111:6,25 112:2
112:16 113:1,2
113:4 114:13
115:10,11,16
115:17,24,25
116:2,4,10
118:2,12,14,15
118:17,18,20
118:25 119:25
120:1,3,4
121:6,9,11,21
121:23 122:9
122:11,12
123:7,8,17,21
123:25 124:1,5
124:9,22
125:14,17,18
125:25 126:1,5
126:7,10,19,20
126:22,24
127:5,8,9
129:1,2,4
131:13,14
132:6,13,14,22
132:23,25
133:1,3,4,6,7,8
133:11,12,15
133:16,18,19
133:23 134:3
134:16,19
135:3,10,25
136:3,7 137:12
137:14,17,19
137:22,24
138:4,24,25
139:12,18,22
140:3,4,7,25
141:13,20
142:1,3,15,16
142:18,19,21
142:22 143:15
145:6,14,24
146:5,8,10,11
146:14 147:11
147:15,18,20
148:3,11,19
149:1,2,4,20
150:11,12,14
150:16 151:1,2
151:5,6,11,13
151:14,18,19
151:22,23,24
152:11,12,14
152:22,23,25
153:1,21,22,25
154:1 155:22
155:24 156:1,2
156:5,23,24
157:6,7,14,15
157:17,18,21
157:22,25
158:8,9,12,16
158:20,24
159:2,4,5,8,13
159:22 160:13
161:10,19,20
162:2,3,10
163:1,7,8,12,13
163:22 165:2
165:11,12,15
165:16 166:11
166:17,20,21
166:23,24
167:4,6,7
168:17,19,23
168:24 170:19
172:8,12,13
173:1,22 176:4
177:21 178:20
179:12 180:13
181:1 182:12
182:19 183:19
184:9,10,24,25
185:2,3,25
187:1,2 188:20
188:21 189:18
189:19 191:7
192:15 195:23
195:24 196:16
197:6,8 198:8
198:9,12,16
200:13,14,18
201:25 203:1
204:20,25
207:23 208:7
212:25 214:1
214:20,21,23
216:6,7 218:1
221:4,15,20,21
222:6 224:15
225:2,3,16
226:7 231:12
231:13,25

232:3,4,23,24 233:8 234:17 234:18 237:10 237:11 240:7 240:10 244:12 248:2
correction 28:14 98:16 127:11 233:23
correctly 31:18 98:18 99:21,22 100:7 107:14 108:24 117:1 119:10 128:9 135:4 137:9 143:10 144:2 154:15,16,24 154:25 162:11 162:22 168:8,9 170:1 175:25 176:1 190:12 199:7,14 234:6
corroborate 70:5
corroborated 70:4
cory 4:15 9:11
costa 185:25
costs 25:22,24 238:1
council 181:20
counsel 3:1 4:1 8:18 9:2,16 22:8 50:7 182:5 241:13 249:11
count 119:12 163:4
country 227:9
county 240:3
couple 227:23 235:15
course 21:17,20 24:25 31:25 33:24 38:13 40:14 43:24 50:22,23 52:24 56:25 64:22 69:4,19 70:15 72:13 74:10 77:4 78:20,24 92:5 99:6 107:15 115:4 120:21 135:11 135:21 144:3 149:7 154:18 155:1 167:1 182:23 188:2 188:15 207:8 211:7 215:9 217:4 227:23 229:2 230:4,6 242:16
courses 34:21 35:10,14 239:21
court 1:1 2:10 8:15 9:14,19 11:6,16 46:14 203:7
cover 47:22
covered 145:20 157:16 192:17
covering 41:12
cranial 229:5
crc 1:25 2:6 249:24
credit 19:24
criteria 95:25 96:16 185:14 221:10
cross 91:18 173:10
crr 1:25 2:6 249:24
csr 1:24,24 9:15,19 249:5 249:24,25
cumulative 7:18 81:3 101:8,18,24 102:5,10,19 165:24 166:4 166:23 194:21 194:24 195:10 197:4 201:8
curious 73:10
current 10:11 32:18 38:23 39:24 41:8
currently 12:24 27:2 29:22 40:20 41:23
curriculum 5:20
cutoff 87:19,21 140:17 238:9
cutoffs 235:20 235:21
cv 1:6 5:18 28:17 29:10

d

d 5:18 8:2
d.c. 4:11
daily 163:10 236:8 239:13
dairy 208:23 209:1,5,8
damage 114:23 171:25 213:8 213:20,25 220:10
data 49:13 57:1 57:1,1,22 60:20 65:18 69:24 74:3 83:14,15 90:3 92:6,6,7 102:5 102:9,11 111:18 116:23 117:21 118:1 118:24 119:4 123:3 131:9 134:2,8 140:16 142:1 149:6,9

149:11 150:3 153:24 155:2,5 155:19 159:1 181:8 182:25 211:20 217:6 236:13
database 153:8
databases 95:23 96:1
date 8:9 25:13 26:3
dates 232:15
david 3:12 8:24
day 51:3,3 162:18 166:19 194:1,1 248:4 249:20
days 79:8 162:21 165:1 166:19
dbs 238:9
dc 3:19 4:5
deal 69:23 167:3
dealt 28:2
death 121:17 122:22 165:6
deaths 106:22
decade 120:22 207:19
decades 137:8 143:8 159:25 167:15 207:2,3 232:16
deceased 133:13 134:17 134:23
decided 94:19
decisions 183:15
declare 248:1
decrease 160:5
decreased 244:25,25
decreases 208:1
deep 238:4
deeper 32:10 40:25
defendants 3:17 4:2 27:22
defenses 11:11
defer 78:6 109:12 139:4 141:17 171:8 184:19 194:7 196:10
deferring 111:14
define 49:5 70:11 81:12,23 87:15 113:25 164:7 190:14 190:19 205:8 213:1 216:12 235:23
defined 113:3 134:12 149:14 164:12 218:2,6 218:8
defining 87:17
definitely 232:1
definition 56:12 87:23 113:23 202:3 222:1
degeneration 234:3
degree 49:2,7 49:10,15 55:25 56:7 57:5 84:19 86:5 209:15,18 213:21 243:20 245:12 246:1
delineate 193:17
deltas 87:15
dementia 235:8 235:11,14,19 235:24 236:3,5 236:11,16,16 236:24
demonstrate 64:19 81:22
demonstrated 200:10
department 3:18 4:3,10 9:9 10:18 18:13 47:16 105:7,21 239:7
department's 239:13
depend 25:18 56:16 60:19 74:19 84:8,13 91:14 102:25
dependent 85:10
depending 60:22 89:21 92:22 166:7 197:17 227:10
depends 223:8 230:12 236:12
deplete 215:4
depletes 214:18
deployment 104:17
deponent 8:16 249:6
deposed 10:21 27:11 44:3,5 44:11
deposition 1:12 2:1 5:9 8:11 11:16 13:9,23 14:7 16:19,20 16:25 17:7,11 22:14 23:18 25:11 28:3,6,8 43:21,24 93:7 93:25 97:6 100:25 115:18 123:10 125:20

132:17 150:20 157:8 161:22 174:11 188:22 189:12 218:3 247:9,24
depositions 43:18
depressed 246:14
depression 246:9,13,16
dermal 74:11 167:2,4 212:2
describe 18:9 23:3 80:25 148:15 235:3
described 69:15 95:5 112:15 151:1 182:22 221:6 234:10
describes 57:15 57:21
describing 81:1 235:4
description 5:7 6:2 7:2
design 91:4,8
designation 78:19
designed 63:7 63:14 80:19 83:15 91:5 106:2 120:24 147:2 200:15
detail 19:1 39:6 190:7 223:19
detailed 138:9
details 185:16 224:10
detected 165:18
deterioration 234:4,9
determinant 205:4,6
determination 158:21
determine 33:14 42:18 57:19 64:11 107:10 111:9 219:15 237:15
determined 42:2,7 110:15 158:23 238:8
determining 104:1,11 200:24 201:4
develop 68:11 72:21,24 105:8 190:23 204:14 207:12 216:15
developed 79:8 182:24 225:6 225:15
developing 61:18 160:12 206:6,18,23 207:21 208:12
development 79:21 165:14
diagnosed 206:8 217:24
diagnoses 42:15,18 50:23 51:1,13,17 220:18 221:1
diagnosis 42:11 42:14 50:22 51:12 159:12 159:19 160:15 183:15 197:1 203:4 215:2 219:23,25 220:5,22 221:5 227:11,12,16 227:18 228:23 231:8
diagnostic 206:9 221:10 227:19
diane 6:5 7:21 8:13 23:11,24 189:17 195:23
die 106:24 122:22 123:2 129:19
diet 209:1
dietary 208:17
differ 138:8
difference 44:2 120:6 160:2 177:20 179:10 206:12
differences 77:11,14 147:9 148:12 169:3,9 175:19,23 177:5,9 181:8
different 34:3 38:13 40:15 51:6 75:5,6,8 103:4 117:14 119:13 121:6 135:23,24 142:17 146:19 172:20,22 173:9 177:10 177:21 178:4 180:3 207:8 216:18 217:4 218:25 225:13
differential 42:11,14 43:3 45:3 50:22 51:12,12 183:15 203:3,4 219:22,25 220:3,5,21 221:5 231:7
differentiate 215:14 235:13
differently 33:4 130:15

difficult 117:12
difficulties 245:16
difficultly 145:3
dig 32:10
diplomate 5:13
direct 117:21 118:1 119:1 123:24 126:9 153:24
direction 124:20,23
directly 75:20 114:6 116:2 233:2
disability 47:17 47:19 48:5
disagree 85:5 92:19 111:7 202:2
disagreed 185:22
disagreement 90:22
disciplinary 46:10,14
disclosed 44:16 94:2
discover 11:10
discovery 146:21
discuss 19:21 24:18,22 39:5 39:15 48:2 58:12 73:11 77:2 94:7 115:1 125:10 171:21 172:18 197:14 216:12 217:16 223:18 235:10 239:3
discussed 15:17 15:22 25:3 45:10 50:24 52:20 53:15 73:7 81:6 122:5,20 141:21 147:4 148:13 153:23 158:6 159:25 187:8 188:3,17 190:25 206:15 211:14 218:23
discusses 129:9 179:12
discussing 122:16
discussion 20:6 38:5 118:7 149:5 205:16
discussions 17:20 54:6
disease 7:4,6,9 7:12,22 13:19 13:22 16:3,15 23:8 27:19 29:13 30:16 32:12,23 35:19 36:5 37:22 39:18 42:3,8 44:8 45:1 48:2 48:3 55:22 57:18 58:1,8 58:10,13,20,23 59:2 60:1,7,15 60:17,23 61:3 61:19 62:6 64:13 65:2,12 66:9,18,25 67:6,18 68:10 68:11 71:13 72:4,21 73:2 74:8 76:2,15 76:19,24 78:14 79:9,22 81:17 82:11 83:4,20 83:22 85:21 87:18 98:15,17 99:2,12 100:3 103:11 106:22 106:25 108:10 119:14,25 120:16,22 132:21 139:22 140:3,7,23 143:5,6,8 149:4,20 150:7 150:11,17 151:9 155:22 157:13 159:24 160:1,5,12,23 161:1,5,15,19 162:1 165:15 167:13,15 168:7,18 169:12 177:8 178:4 184:15 187:6,15,19,25 188:8,13 189:18 190:23 199:13,19,24 200:5,8,9,11 201:9 202:5,7 203:1,2,19 204:4,6,14,20 204:22,24,25 205:4,6,13,19 205:20,24 206:6,8,18,23 207:7,12,22 208:1,12,16 209:12 210:7 210:19 211:5 211:16 212:13 212:19,25 213:6,9 214:5 214:8,13,14,16 214:20 215:15 216:15,22 217:14,22,25 218:5,8,15,25 219:6,11,16,19 220:6,14 221:14,19 223:4,14

224:15 225:16 226:1,6,24 228:12 229:8 233:14,18 234:16,25 235:5,6 236:16 240:13 242:24 243:10 244:5 244:15 245:7 245:23 246:6,8 246:10,15,17 246:21
disease's 214:16
diseases 6:9 47:23 106:2,9 106:10 129:19 212:15,24 213:5
dismantling 249:16
disorder 16:14 29:18 217:20 227:17 237:19
disorders 16:6 18:14 30:2,20 31:22 38:22
display 235:7,9
disregard 140:16 195:2
distinct 217:8
distinction 41:21 49:14,18 70:11 129:6 140:10 203:13 205:1 235:18 235:20
distinctions 68:14
distinguish 51:14 68:20 215:6
distinguishing 204:16
distorting 124:20
distractions 231:5
distraught 246:14
distribution 175:21
district 1:1,2,3 8:15,16 10:20 240:3
dive 219:1
division 4:4 8:9
dmiceli 3:13
doctor 21:17
document 1:7 5:8,12,15,19,22 6:3,6,11,15,20 7:3,5,8,11,14 7:18,20 14:4,8 28:19,22,24 47:22 48:1 55:8,9 79:6 174:16 175:3 176:13
documentarian 242:14
documentation 161:7 227:12
documented 164:25 211:22 242:6
documents 5:11 17:19 93:14,16 94:18
doing 173:15 220:3
domain 96:15
dominant 209:21
don 120:10
door 32:8
dopamine 96:7 165:7,10,13 168:3,6 215:4
dopaminergic 38:25 114:24 115:7 167:23 168:16 208:7 214:19 215:10 215:10 220:13
dorsey 15:6,9 15:15 16:13
dorsi 15:10
dosage 102:21
dosages 173:1
dose 61:8,11,20 71:7,9,14 72:4 80:9,24 103:3 103:20 165:24 183:13 190:8 197:4 201:8
doses 62:12 165:17 167:10
double 123:22 124:10 142:4 166:3 242:11
doubling 88:9
doubt 65:5 191:1
douglas 4:14 8:7
dowling 3:6,8 9:1,1
dowlingfirm.... 3:9
downstream 82:4,9,16 170:15 171:23
dr 6:4 10:3 14:2 15:6,6,19 16:5,13,23 17:1 18:4,18 18:21 19:4,7 22:17 23:21 28:11 46:25 72:18,19 75:13 77:25 78:1,6 89:13 93:12 94:10 97:9 100:15 105:25 115:10,21

118:10,23 123:13 127:10 132:4,20 142:23 150:23 154:3 157:11 161:7 165:25 166:5 174:14 178:9 179:9 180:25 184:2 185:2,8,13,15 185:19,20,20 185:25 186:23 188:19 189:1,3 189:10,16,24 190:3 192:12 192:19 194:4,7 194:14,18 195:7,21 198:8 216:3 217:24 233:17,18 234:11,24 235:7,17 241:2 242:3,5 243:2 244:10 245:15 247:17
draft 28:22 233:3
drafted 28:19
drafts 233:11
drank 65:18
draw 84:3 173:13
drinking 6:7,13 6:17,22 63:7 115:15 197:16 197:17 199:5 200:16 201:13 201:23
drive 10:13
driven 206:9
driving 165:14 206:12
drs 184:20 186:22
drug 38:22
dry 39:12,21 40:13 224:3,4
due 71:1 106:22 127:23 238:6
duly 2:11 9:23 249:6
duration 66:2 81:3 103:21,25 104:9 116:9,16 116:20
durations 167:12
duty 119:7
dysfunction 160:23
dyskinesia 229:15
dysphasia 244:11,12,14 244:18,20 245:9,11

e

e 4:12 8:2,2 46:8
e.g. 108:16
earlier 12:12 39:14 43:14,19 43:20 44:1 50:24 52:7 73:7 74:5 90:10 115:2 144:12 145:20 149:14 159:12 159:25 161:9 161:12 167:11 190:25 203:11 207:6 210:21 215:9 221:8 226:10 230:22 230:24
early 161:15 242:17
earned 26:13
easily 39:25
eastern 1:2 8:15 10:20
ecological 92:25 93:4
educate 48:3
education 29:17 134:25
educational 28:25
effect 81:9 108:15,15 130:14 168:13 177:21
effects 73:16 79:20 145:4 169:14 171:16 171:23
eight 99:7 162:21 163:7,9 163:20 165:2 165:10 166:20
either 15:6 27:25 32:9 34:23 43:2 44:15 45:9 58:25 60:13 61:1 120:11 124:19,23 153:18 154:12 162:17 221:7 222:21 229:21 240:12 249:12
elevated 111:23
eliminate 138:7
eliminating 209:8
elizabeth 3:20 9:6
elizabeth.k.pl... 3:22
ellison 4:11
email 3:5,9,13 3:16,21 4:7

emerging 150:3
emphasize 226:9
employ 34:4 219:24 239:11
employed 16:9 29:22 116:21
employees 6:21 121:17
employment 28:25 29:3 116:8,9 119:8
encephalopat... 149:12
encounters 237:22
endogenous 167:21,21
entered 23:23 203:21
entire 63:17 77:17 119:20
entirety 179:24
entitled 2:10 5:8,12,15,19,22 6:3,6,11,15,20 7:3,5,8,11,14 7:18,20 175:3
environment 217:9 224:4
environmental 4:4,9 91:10 135:25 154:11 205:12,18,23 210:3 217:23 226:13
enzyme 115:5 164:19,20
enzymes 82:7
epa 62:17,22,25 63:3,20 66:13 200:13 201:7
epa's 199:4
epidemiologic 32:2 33:25 64:24 67:25 68:17 109:3 113:7,10,19 114:19 194:17 195:15 211:18
epidemiologi... 30:25 31:12 33:21 67:17 68:9 85:13 89:14 98:13 99:16 100:10 112:15 194:13 194:20 195:6 195:10
epidemiologist 30:7,22 31:9 100:18 109:13 123:7 126:6 127:8 138:9 139:5 141:17 150:13 162:2 168:23
epidemiologi... 111:15
epidemiology 30:10,13,16,21 31:3,6 109:9 153:13
episode 243:15
equal 103:22 108:23 109:22 110:14 111:9 112:3 162:19
equally 49:6 185:21
equation 92:7
equipoise 48:18 48:24 49:5,15 50:16 57:21,23 83:13 97:18 98:25 99:11 100:2 146:6,9 146:14
equivalent 166:10
errors 127:12
especially 37:24 71:19 88:21 177:5 243:16
esq 3:4,8,12,15 3:20,20 4:6,6 4:11
essentially 178:22
established 83:14 204:7 221:10 222:1 227:16 228:4
establishes 63:4 200:13
estimate 26:14 108:16 197:12
estimated 158:22 190:7 198:25 227:4
estimates 108:15 143:2 144:5 158:7 166:6
et 45:19 68:24 80:20 98:11,12 111:12 151:10 161:18 183:13
ethical 65:6
ethylene 161:18
etiologies 222:8 222:11
etiology 44:25 45:3 51:12 203:4 217:14
evaluate 62:23 88:24 123:24 126:9 201:8
evaluated 37:21 73:16 161:25 187:14

evaluating 47:17 48:4,9 48:14 57:17 187:18,24 188:7,12 238:25
evaluation 6:11 6:16 17:23 115:13 182:18 228:20 233:7
evaluations 226:19 228:19
evans 45:19
everyone's 130:12
evidence 6:7 55:15 56:25 57:2 81:1 82:12 84:9,22 89:14 97:11 98:13,23,25 99:17 100:2 105:4 106:15 107:13,23 112:21 114:7 114:19 145:12 145:23 146:7 146:13 150:5 155:13 160:17 160:18 182:18 195:25 206:11 210:9
evolution 217:18
exact 80:11,16 173:14 203:23 203:24
exactly 20:3 38:10 62:13 66:5 70:19 87:22 135:20 187:10 193:5 201:6
exam 42:17,20 192:17 227:14 227:19,22,24 227:25 228:8 228:22 229:1,3 229:7,20 230:4 230:10 231:2 235:22
examination 5:4,5 10:1 228:2,11 232:2 242:1
examinations 5:2
examine 193:1
examined 2:11 9:24 37:17 227:8 232:6
examiner 228:7
examining 229:5
example 11:22 16:5 26:1 37:10 41:16 57:23 61:24 70:14,24 79:24 81:24 83:16,18 87:24 118:10 119:14,17 127:23 183:15 209:4,8 212:6 212:14 235:23
examples 48:22 62:3 83:13 187:9,11
exams 227:1
exceeding 155:12
excellent 79:24
exception 29:1
excess 201:24
exclude 95:9 129:11 131:6 152:3
excludes 151:20
excluding 94:11 145:10
exclusively 144:22
execute 22:10
executed 22:21
executing 22:25
exercise 208:11 208:14
exhibit 5:7,8,12 5:15,19,22 6:2 6:3,6,11,15,20 7:2,3,5,8,11,14 7:18,20 13:23 14:3 22:14,17 23:18,22 28:8 28:12,15 93:7 93:8,17 95:11 95:13 97:6,10 115:18,22 123:10,13 125:20,23 132:16,17 150:20,24 157:8,12 161:22 162:5 164:1,13 174:11,15 186:2,6,10 188:22,25 189:12,14
exhibiting 161:4
exhibits 5:6 6:1 7:1 93:13 94:11
exist 224:25
existing 96:17
exists 55:3 65:6 74:3 173:8 191:1
expand 69:25
expect 106:24 120:15 160:1
expenses 25:21 25:24 26:5

experience 29:19 34:12 39:20 123:6 209:3 237:18 246:5,7
experienced 234:2
experiences 135:22
experiment 183:13
experiments 62:10
expert 5:16 7:19 22:1 25:10 26:13,15 27:3,10 29:13 29:24 30:10 33:22,23 34:4 43:7 52:10 53:1 59:12 126:5 127:8 150:10 153:12 162:1 168:22 168:25 202:25 203:2,7,15 239:17
expertise 29:16 35:14 123:6 126:5 203:12 237:20
experts 196:11 241:2
explain 81:12 107:16 184:11
explained 140:14 203:11 209:19
explaining 111:16 124:2
explicitly 135:7 184:22 205:10
exposed 6:13 6:17,21 36:9 37:13 39:4 63:15 64:3,4,5 68:3,3 76:1,13 78:13 79:23 105:9 115:14 117:14 130:22 142:2 154:8 159:12,20 162:9 190:22 191:23 192:2,6 192:10 197:4 200:5 211:11 212:6 225:5,8 225:18
exposure 7:9 7:18 20:18 23:6 37:7 38:24 39:25 40:13 44:9 58:6,16 60:20 61:19 62:5 63:8,12 64:8 65:1,11,18 66:1,8,17,23 67:5,12,16 68:1,7,22 69:1 69:4,24 71:13 71:21,23 72:3 72:11,17,19,20 72:23 73:3 74:6,11,12,16 74:22 75:18,21 76:18 77:13 78:2,7,7 81:2,3 81:3,16,17,18 85:21 90:12,21 98:11 101:9,11 101:18,20,24 102:1,5,6,9,10 102:17,19,23 103:5,25 104:1 104:11,12,15 117:21 118:1 118:14 120:21 123:24 124:18 124:21 125:10 125:16 126:10 127:20,22 129:20 130:13 130:25 133:10 133:17,20,22 135:15,22 136:3 142:1 143:3 144:15 145:6 155:1,4 157:13 158:7 158:11,15,21 158:22 159:25 163:10 165:10 166:5,23 167:4 167:12,20 168:14,15 188:19 189:5 189:11,23 190:5,9,11,14 190:17 192:18 194:6,13,22 195:7,10,16,19 196:25 197:15 198:4,10 199:3 199:10,17 200:17 201:13 201:23 202:14 205:23 210:6 210:11,24 211:8 215:3 221:15,17 223:22 224:6 224:24 246:22 246:24
exposures 7:3 20:23 37:1 38:3,9,11,15 39:14 40:24 43:8,18 63:2 65:19,20 68:17 73:9 75:14 91:10 92:4 116:1,7 117:6 132:21 135:18 135:21,25

136:9 143:7 144:6,23 147:3 153:18,25 154:11 155:8 159:2 160:20 197:11,13 205:19 210:17 211:4,6,9,23 220:14 222:13 224:20
extension 209:16
extensively 136:15,18 137:8
extra 90:3
extrapolate 130:21
extremely 234:25

f

face 227:15,15
fact 153:2 154:8 155:7
factor 23:7 33:16 43:3 52:16 60:16 85:22 119:19 149:4,17,20,24 204:13,19,21 204:23,24 205:3,13,24 206:13,14,16 206:18,20 208:11,13,21 209:9 210:7,18 210:25 211:16 213:8 214:8 215:5 223:13 224:13 226:4
factors 42:22 42:25 60:6 61:4 118:20,24 119:4 124:16 124:17,25 126:19 165:14 168:4 183:5 204:9,17 205:2 205:5,9,12 206:5 208:17 208:18 210:4 216:9,19 217:23 218:24 220:16 223:11 224:20 225:23 226:13,14 230:24 238:6,7 238:8
failed 104:16
failure 244:24
fair 11:14,25 12:7,17 21:10 58:18 93:6 103:14 140:20 200:7 204:1
fairly 230:17
fall 242:8 243:8 243:16
fallen 243:3
falling 244:4
falls 242:20,21 243:23
familiar 10:23 109:5 128:18 170:25 171:7 182:14 183:21 186:1 217:10
family 133:6 135:13 209:11 222:22 230:9
far 27:23 64:5 64:22 72:13 179:17 180:8 199:4
farm 211:15,20
farms 212:6
faster 234:14 235:6
favorable 91:18
february 5:18 29:5
federal 53:21 53:25
fee 26:7 27:5,6
feel 30:3
feeling 56:22 56:25
feindt 53:1,20 240:9,10,21
felt 228:7
field 171:11 194:9 203:16 212:8
fifth 112:17
figure 162:24 165:8
fill 237:2
finalized 29:6
finally 156:14
find 33:6 50:4 140:22 231:4
finding 94:23 107:10,20 144:18
findings 85:9
fine 19:25 55:11 75:3 174:23
finish 12:9 13:12 241:5
finished 177:23
finishes 241:12
first 2:11 19:17 20:22 37:16 46:5 55:2 89:22 110:13 111:25 112:13 148:4 161:4 164:12 167:17 182:8 209:15 209:18 233:24 249:6

fit 70:19 221:9
five 27:11,17 28:1,3,5 62:18 142:3,9 162:21 165:1 166:19 215:19
flat 26:6,7
flu 212:20
fluctuated 116:19
fluctuations 77:22 78:1
focus 51:13 230:6
focused 98:11 136:8 147:16 196:25
focuses 177:4 179:14
follow 135:8 193:19
following 44:18 147:13 168:13
follows 9:24 76:9
food 26:4
foods 209:1,4
footnote 172:5
foregoing 248:2 249:7,9 249:13
forget 46:5
form 20:24 21:7 24:13,20 29:25 30:18 36:15 38:19 39:22 41:6 42:23 47:24 49:3,25 51:10 53:14 54:4,18 54:24 55:6 56:2,13,18,23 58:11,21 59:3 60:2,8,18 61:9 61:16,21,25 62:7 63:9,22 65:3 66:10 67:19 68:12 70:9 71:15 72:6 73:5 76:25 77:8,24 78:15 80:14 81:11,19 83:5 83:23 84:6,14 84:20 85:4,23 86:9 87:6,13 88:5,16 89:1 89:18 90:25 91:20 92:1,12 92:20 93:1 95:3 96:20 97:15 100:6,11 101:12 102:2 102:24 103:7 103:18 104:2 104:18 105:11 105:22 106:4 106:11 108:2 109:11 113:8 114:2 116:3 117:16 118:3 120:7,17 121:24 125:3 126:23 129:16 130:16,23 133:24 136:17 137:13,18,23 138:5,15 139:3 140:8 141:1,14 143:16 144:10 146:1,16,25 153:5,14 155:6 155:14 160:7 160:16 161:11 170:6,21 171:17 176:5,8 176:10 177:22 178:21 180:12 182:20 185:9 187:7,20 190:24 191:11 191:14,19 195:13 196:9 196:21 200:19 201:1,10,17 202:1,11,17,21 204:5 205:7,14 205:25 206:19 207:16 211:17 213:10 216:23 218:10,17 219:7 220:4,24 221:24 223:6 223:25 224:8 225:19 226:8 241:10 242:9 243:11,25 244:7,22 245:19 246:3 246:11 247:2
formal 31:8 184:20 229:22
formally 22:4 183:16
formation 167:21
formed 24:12
former 38:23 41:11
formulation 171:24
forth 33:19 229:6
found 70:8 71:13 72:3 80:3 96:18 97:2,3,18 99:11 114:22 115:6 138:22 139:10,19 140:1 146:9,19
foundation 21:7 176:6,16 176:19 179:23
four 18:19 145:10 188:14

200:2
fraction 26:21
fracture 243:9
frame 243:24
framed 74:5
framework 220:6
frank 100:15 245:3
franklin 4:10
freeman 240:4 240:6
frequency 104:10 206:22
frequently 239:12 242:8
friend 232:7
friendly 15:16
front 88:6
fryar 19:4
full 10:5 15:11 101:10,19,25 102:5,16 140:17 183:24 249:9
fully 22:21 178:1
function 229:12
functional 236:2,7
fund 117:4
fundamental 59:18 61:14
further 143:18 152:24 153:3 247:16 249:11
future 237:12

g

g 8:2 223:18
gait 229:6,13
ganglia 214:11
gaps 181:8
gary 185:2 232:8
gastrointestinal 213:7,20
gavage 162:10 162:20
gender 226:14
gene 209:21 223:8,14
general 15:25 17:2 32:13 37:6 59:13 60:25 61:18 83:8 86:19 111:2 121:6 122:20 179:20 183:11 187:1 193:15 206:25 219:19 225:10 228:21,23 229:3,19 238:13
generalization 170:5 173:13
generalize 180:5
generally 20:9 20:17 23:3,5 57:14 59:6 61:10 63:24 83:10 85:12,16 85:17 86:1 87:3 89:14 90:7 91:18 92:9 104:19,23 105:17 109:9 122:21 123:2 128:14 181:22 183:14 194:9 195:18 197:20 204:7 208:8 214:3 216:22 218:7 226:11 228:21 243:23
generating 83:1
generation 209:23,23
genes 223:2,10 223:17
genetic 91:9 124:25 126:19 182:25 209:13 209:25 210:1 216:9,14 217:22 218:22 220:16 222:15 222:18,24 223:1,11 226:12 236:9 236:12
genetically 236:22
geneticist 29:20 29:22 203:13
genetics 29:21 29:24 30:2 203:7,12,16,19 216:8 236:20
georgetown 3:4
germane 86:16 96:13 109:6 196:24
getting 244:18
gibbons 3:20 5:4 9:4,4,8 10:1,2,17 14:1 14:24 15:1 21:5,14,15 22:16 23:20 24:15,23 28:10 28:14,16 30:4 30:23 36:20 39:2 40:4 41:20 43:5 46:24 48:6 49:16 50:3 51:19 53:19 54:7,20 55:1 55:11,13 56:10 56:15,21 57:3 58:17,24 59:9

59:13,17,21,23 60:4,12,24 61:12,17,23 62:4,15 63:13 63:19 64:1 65:8 66:6,16 66:22 67:3,10 67:21 68:21 71:4,22 72:15 73:12 76:6,20 77:3,10 78:5 79:2,14 80:21 81:14 82:17,22 83:10,17 84:2 84:11,16,23 85:6 86:3,12 87:10,20 88:10 88:23 89:3,12 90:5 91:13,23 92:8,16,23 93:5,11,21 94:7,9 95:10 96:22 97:8,16 98:1,4 100:8 100:14 101:3,7 101:16,23 102:7 103:2,13 103:23 104:6 105:1,15,24 106:6,13 108:5 109:14 113:13 114:8 115:20 116:6 117:19 117:24 118:6 120:12 121:1 122:3 123:12 125:5,22 127:1 127:15,17 129:23 130:17 131:11,15 132:3,15,19 134:1 136:20 137:15,20 138:1,10,17 139:7 140:19 141:9,18 142:7 142:11 143:17 145:2 146:4,20 147:5 150:22 153:11,17 155:11,16 157:10 160:9 160:21 161:16 161:24 164:1,5 170:16,24 172:1 174:4,10 174:13,23 175:6 176:7,10 176:16,22,24 178:8,14,16,24 179:6,8 180:7 180:14,16,24 183:3 185:12 186:6,8,13,17 187:13,21,22 188:24 189:14 189:15 191:4 191:12,16,21 195:20 196:12 197:2,8,24 200:22 201:2 201:12,21 202:8,13,19,24 204:11 205:11 205:22 206:3 206:21 207:20 211:24 213:18 215:16,19 216:2 217:1 218:13 219:3,8 220:19 221:3 222:3 223:15 224:2,11 225:22 226:17 237:4 241:11 242:9 243:11 243:25 244:7 245:19 246:3 246:11 247:2,8 247:16

give 13:6 19:23 20:25 35:14 38:11 62:12 89:22 116:12 124:12 173:25 174:24 198:13 227:4

given 42:19 90:13 98:22 99:25 120:13 161:13 165:18 166:16 204:9 207:11

gives 37:6 57:22 90:2

gleaned 111:3

glutathione 175:16

gluten 209:5

go 10:24 12:16 13:16 33:8 39:10 42:13 44:21 46:17 80:23 83:11 90:24 107:15 112:12,13 114:9 132:4 135:21 136:21 147:6 148:4 164:21 170:13 173:11 174:23 175:1 180:16 186:14 199:9 200:24 201:4 242:19

goal 41:9 42:4 62:25

goals 63:11

going 10:24 11:12,17,19 12:6 13:11,15 14:3 19:9 21:1 21:5 23:22 28:12 38:16 46:18 55:7 58:14 76:4

89:4,6 93:25 97:10 111:19 131:17 132:15 136:22 150:24 155:1 174:19 178:10 179:23 180:18 198:21 210:3 215:21 228:25 241:18 247:8,12,21
goldman 15:6 15:19 16:5 67:23 68:14 69:19,22 72:8 79:25 80:7,8 80:24 81:7,24 98:11 131:13 132:12,20 136:2 138:22 139:10,19 140:1,21 141:25 142:13 142:23 145:13 145:22 146:12 146:23 147:1 150:25 151:4 151:15 154:3 155:5,7,21,25 156:6,13 158:4 158:10,15 159:1 161:25 199:20 200:1
goldman's 150:15 157:5 157:12 158:6 199:22
golkow 8:8
good 8:6 9:17 10:3,4 38:8 89:4 108:18 109:22 160:18 182:25 210:13 212:17 215:11 229:7 242:14
google 95:19
gradients 71:7 71:9
graduate 35:13
grams 166:14 166:15,22
granular 41:1 73:11 217:6
granularly 211:22
graph 162:25
graphs 165:5
great 18:16 93:20
greater 72:12 80:3 81:25 82:1 87:17 88:1,2 143:14 143:25 168:2 236:15
grocery 243:4
grossman 3:10
ground 10:25 37:14 212:5,21
groundwater 125:13
group 3:3 8:23 8:25 9:3 16:13 80:19 116:25 117:8 225:11
groups 209:4
growing 211:15
guaranteed 190:23 201:14 201:18,20
guarantees 204:13
guess 21:1,1,3 21:12
guessing 27:16
guidance 57:12 57:18 62:17
guide 182:18
guided 229:7
gut 56:22,24 213:13,15 214:4

h

hadnot 128:4 128:15 129:1,9 129:14,20 130:1,5,8,13,18 130:22
hand 9:21 177:11 211:10 249:20
handed 23:21 28:11 97:9 123:13 125:23 150:23 157:11 162:4 174:14 188:25
handing 176:13 189:14
hanley 3:20 9:4 10:17 14:19 55:7 59:4 97:25 174:2 186:4
hanley.w.gib... 3:21
happen 227:6
happens 38:1
happy 25:1 27:24 134:4 145:15 164:21 182:4 213:12 218:19 230:23
hard 26:14 41:18 43:1 56:14 71:17 84:15 86:2 165:22 208:9 210:10
hazard 70:17 86:14 112:9 124:19
head 39:1 91:6 91:6 105:16 125:2 126:21

134:2,9,12,13 134:19 135:6,9 148:2,21,22,23 148:25 149:3,7 149:8,9,14,15 149:17,18,22 149:23 150:1,6 206:15 220:15
health 62:24 118:20,24 119:21 151:17 151:17 152:1 152:19,20 195:22 196:20 200:23 201:3 201:15,25 202:10,16
healthcare 152:21 157:25
hear 12:3 20:22
heard 8:15 10:16 20:14 171:1
heavy 211:10 211:13
held 8:11
hello 15:21 16:8
help 105:7 232:11
helped 232:20
helpful 195:19
hereunto 249:19
hierarchical 51:5 221:8
hierarchy 89:14
high 50:25 80:3 103:3 112:16 112:24 113:3,6 113:9,11,16,16 113:20,23 139:8,9 156:17 198:11
higher 112:10 114:4 120:16 148:1 167:10 197:14,21 202:6,9 223:22 224:5
highest 110:15 111:9 156:16
highway 8:12
hill 53:4 182:14 182:17 183:4 183:16,18 184:4,6,19,20 184:23 185:7 185:14 186:21 186:25 240:7
hired 202:25
historic 38:3
historical 125:11
historically 136:25
histories 135:13 136:12 192:15
history 36:17 37:3,4 38:7,8 38:17,21 39:12 42:17,20 73:3 119:6 133:11 133:15,15,20 133:21,22 134:8 136:4,7 192:19 209:11 215:12 219:21 230:5,8,9,15,20 232:15 242:22
hmm 81:5 108:6
hobby 133:11 133:15,21 136:9
hoehn 229:15
holcomb 128:16
hold 29:12 30:9 142:7 186:8 243:20 245:12 245:25
hopefully 189:10
hormonal 206:12
hospital 10:13 149:23
hospitals 47:16
hour 2:4 12:13 17:13 25:11 46:16 68:4 89:5 230:17 232:23
hours 17:13 176:23 195:16 215:18 241:16
huh 11:24
human 62:24 64:19 92:3 112:21 113:11 165:22
humans 64:13 64:17 167:14 184:17
hurt 4:6 9:9
hydrate 177:13
hydrocarbons 188:4
hydrogen 169:22 172:14 172:19,25
hydroxylase 164:13,19
hypothetical 66:11,20 71:16 84:10
hypothetically 131:3

i

icu 149:10,22
idea 37:6 216:10,13
identical 135:18
identifiable 219:9
identification 13:24 22:15 23:19 28:9 93:9,13 97:7 115:19 123:11 125:21 132:18 150:21 157:9 161:23 174:12 188:23 189:13
identified 134:23 151:16 152:16 218:21
identify 8:18 71:12 72:2 122:24 190:21 225:1 226:15
idiopathic 217:11,14,16 217:18,25 218:2,7 226:11
idiosyncratic 216:19
ii 133:2
illustrative 64:23
imaging 230:7
imbalance 243:15
ime 229:25 230:19 231:8 231:11,14 232:23 233:1 235:8
imes 230:11
impact 33:16 99:8 160:14 209:2 228:22 232:17 238:22
impaired 133:14 134:17 134:24
impairment 235:9,11,12,14 235:23
implication 203:5
imply 217:21
important 70:10 86:7,17 88:14 92:7 103:25 104:11 107:22 114:14 119:11 129:6 140:13 155:20 156:8 179:25 211:7 215:6 217:17
impossible 41:7
imprecise 143:2
incidence 160:2 206:24 225:12
incidentally 238:21
incidents 207:3
include 29:10 38:22 70:14 88:20 118:8 198:14 229:5 244:24
included 64:6 65:16 68:1,3 68:19 69:8 78:1 116:5 129:5,7 131:5 142:2 145:14 152:1,7,17 166:1 169:6 184:18 185:17 193:7 229:10
includes 38:8 160:20 184:14 212:24 220:6 230:6
including 31:25 44:13 58:15 104:20 119:13 129:8 132:12 168:12 198:14 212:4 229:12 235:16
inclusion 129:11 149:22 154:20
income 26:13 26:25
incomplete 66:20 71:16
increase 67:17 68:9 160:11 170:10 206:1,5 206:22 244:4 245:6
increased 65:22 87:9,11 103:11 140:2 170:9 205:20 206:24 208:20 208:24 209:12 212:12,16 223:3
increases 61:19 207:22 236:17
increasing 208:6
independent 17:22 33:1 54:12 60:14 192:13 226:18 228:11
independently 33:8 61:1 122:13 126:2 127:2 191:22 192:1,5,9,21

index 5:1,2,6 6:1 7:1
indicate 85:14 87:4 108:13 141:19 152:24
indicated 62:22 64:9 73:3 109:22 142:20 163:16 193:20 222:4,21
indicates 86:21 86:24 137:16 137:21
indicating 168:3
individual 153:19 154:1 155:2 156:7 190:22 204:9 218:25 226:16
individualized 118:13 125:16 158:7
individually 73:17 80:2,18
individuals 15:4 36:8 105:9 123:25 126:10 129:4 151:20 152:1,8 152:18 153:25 154:12,21 159:13,21 224:4 226:23
induced 175:24 215:7,14
industry 39:13
infections 212:20 245:2
infectious 212:15 220:14
inferences 143:3
inferred 133:10 158:10,15
inferring 133:20
inflammation 171:23
inflammatory 220:11,15
influential 205:9
informant 133:14 134:18 135:16 230:15
informants 134:22 135:5 143:13
information 11:10 20:1 25:1 29:9 36:13 43:4 48:4 85:20 99:18 102:3,10 102:15 124:15 127:24 134:18 135:5,9,15 136:3 143:23 144:20 153:9 232:12,21 242:22
informative 90:3,9 114:5
ingested 194:5 194:9
ingestion 74:15 167:1 195:22 196:2,7 212:3
inhalation 74:12 167:2,4 212:2
inherent 144:6 144:24 191:2 246:16
inherited 209:25
inhumane 62:11
initial 20:2 21:25 95:12
injuries 37:2 125:2 126:21 134:2,19 135:6 148:2 149:18 149:23 150:7
injury 39:1 134:9,12,13 135:9 148:21 148:22,23,25 149:3,7,8,9,14 149:15,17,22 150:1
innocent 90:11
inside 197:18
insight 90:3 101:10,20,25
insofar 129:20
instability 242:24
instance 57:17 70:1 96:18 102:21 214:17 224:3
institute 16:10 78:16 79:3
institution 33:17
institutional 78:22
institutions 41:11 235:16
instructed 54:2 237:12
instruction 20:25 21:11
insufficient 168:1
insurance 152:3
intake 208:24 244:25 245:1
integrate 42:17
intensity 103:24 104:4

intentionally 95:9
interactions 80:12
interest 32:19 32:22 33:18 178:9
interested 38:2 249:14
interesting 89:20 91:2 210:13
interests 33:11
interference 82:21
intern 4:15
international 16:6
interns 9:12
interpret 54:17 113:16 164:16 176:13
interpretation 110:19
interpreted 111:17 116:17
interpreting 138:2
interrupt 59:4 82:19
interstrain 178:3
interval 70:19 107:25 108:1,4 108:22 109:2 109:10 110:6 111:11 137:16 137:21 138:2 138:12,19 139:2,12 141:10,13
intervals 70:14 88:15,20,24 107:9 108:12 137:11 141:23
interview 135:2 230:4
interviews 134:3
intolerances 209:6
introduce 85:2 132:15
introducing 14:2 22:17
introduction 177:3,17 178:5
inverse 202:6
investigator 30:25 34:15
involve 145:5
involved 43:17 105:3 239:13 240:23
involving 43:7 240:12
irrespective 102:22
isolating 145:3
isolation 213:4
issue 176:17
issues 20:10 245:21

j

january 6:9
job 133:20 144:21
joel 18:4
joey 9:10
joined 9:8
joseph 4:6
joseph.b.turner 4:8
journal 33:16
journals 21:21
judgment 237:19
july 1:14 2:3 8:1,9 247:22
justice 3:18 4:3 4:10 9:9 10:18 50:2,8 52:8 53:16 54:3,9 54:13,21 55:15

k

k 3:20 46:8 223:18
kathleen 1:24 2:5 9:18 249:2 249:23
keep 31:23 247:18
keeping 38:7
keller 3:14
kellerpostma... 3:16
kelly 7:19 72:18 188:19 189:1,3
key 181:7
kidney 119:14 177:6,8
kilogram 162:18 165:1 166:17
kind 18:14 43:19 51:4 167:16 173:7 228:4 230:15 243:14
kinds 32:22 33:13 36:13 38:15 228:1
know 14:13,15 14:16 15:8 18:4 20:15 21:1,4,20 26:17 29:15 32:15 36:24 37:2,10,13 38:7,9 39:6 40:2,21,22 41:1,11 45:22 52:11 62:8,13

65:9,24 66:12 66:14,21 67:14 70:7 71:2,6 79:19 82:10 91:3 101:5 102:11 104:24 113:5 120:21 130:4 131:5 136:23 149:6 165:17 167:14 173:8 183:9 196:6 200:2 202:3 206:13 219:12 222:12 225:21 226:11 238:15
knowing 37:5 37:17 66:4 84:1
knowledge 36:10 39:3 56:8 94:15 124:24 126:18 135:12 187:3 222:20 232:14
known 13:20 21:13 62:21 64:18 78:13 80:13 99:25 151:9 170:18 173:11 191:2 199:12,18,23 200:8 210:18 218:16 222:12
krause 46:6
kristin 1:13 2:2 2:9 5:9,13,16 5:20 6:4 8:17 9:22 10:7
ks 5:18

l

l 3:19 8:13 167:22 223:18 240:4
labs 230:8
lack 117:21 118:1 119:3 124:15 127:24 155:4 176:19 176:19 179:22 208:11
lacks 176:5
laid 220:7
lake 3:7
lamacchia 19:20 20:11 21:25
language 49:24 50:4 52:10 53:24 139:14 141:6 205:21 238:18
large 62:18 130:21 131:8 143:13,23 203:20
largely 130:2 206:9
late 247:10
latency 120:20 120:22 159:24 160:5,19 167:15
lawsuit 11:11
lawyer 54:19
lay 21:18
lead 9:2 133:22 135:24
leadership 8:23 8:25 9:3 19:20
leading 100:17
leads 208:6
leave 26:21 93:25 247:9,13
left 119:2
legal 3:3 8:8 15:11 47:22 52:12,14,15,23 53:16,17 54:3 54:17 203:18 217:5 219:2
lejeune 1:6 6:8 6:14,18,22 7:7 7:10 10:19 17:24 19:17 20:10,15,18,22 21:19 23:7 36:9 37:18 50:2,8 52:8 53:16 54:3,9 54:13,21 55:15 58:7 59:1,25 64:3,21 65:17 65:19 74:6,17 74:23,24 75:10 75:25 76:12 77:18 78:18 79:8 89:25 104:16,20 105:10,14 115:16,23 116:1,5,10,22 117:7,13,22 118:2 119:8 120:2,6,14 121:4,7,20 122:9 123:15 125:25 128:1 128:12,20 131:6 145:13 150:18 152:9 153:20 154:7 154:22 156:15 157:14 158:17 158:19,22 165:19 181:20 190:6 191:24 192:3,7,11,14 192:18,23 193:1,3,15,18 199:3 221:15 221:18 222:4 223:23 224:7 224:13 225:6,9

225:15 246:23
lejeune's 120:15
lengthy 227:17
letter 47:25 48:1,4
letters 47:18,21 50:11
level 35:14 41:1 62:5 64:11,12 64:25 65:6 67:11,16 68:7 77:7 85:14 86:22,24 87:11 103:5,16,17 108:13 110:16 111:9 202:15 235:19
levels 40:15 62:23 63:1 64:18 65:23,25 74:15 75:17 78:2,22 116:19 118:9,13 125:9 129:20 130:25 154:9 155:9 156:18 157:2 165:8,18,23 190:10 199:5 199:10,17 200:2 202:4,6 202:9 223:22 223:23 224:6
li 240:4,16,17 240:19
liability 45:21
license 9:19 249:4
licensed 9:19 249:3
licenses 30:13
licensing 46:10
life 19:3,4 51:3 236:8 237:5,9 237:25
lifespan 123:1 167:13
lifetime 63:8,12 63:17 134:8 159:2 200:17 201:8 204:15
light 175:22
likelihood 129:18 144:17 216:15
likely 20:3,4 37:13 42:19,19 42:22 47:5,8 47:13,15 48:8 48:10,12,16,19 49:6,6,19,23 50:12,17,23 51:1,1,2,6,14 51:15,15,17,18 51:20,21,25 52:2,5,15 53:8 53:12,13 55:5 55:17,21 57:13 57:19 65:12 68:10 71:21 74:7 76:18,23 77:6 81:16 127:12 135:22 200:8 210:20 221:17 222:24 223:4 224:14 243:12 245:24 246:21
limit 106:25 108:20,21 176:7,10 178:10 198:17 227:25,25
limitation 124:14 127:11 127:19 142:21 143:5 153:4 155:5,10
limitations 33:18 88:19 107:16 117:20 117:25 118:7 142:25 191:3 208:10 227:21 246:14
limited 21:6 98:15,17 99:17 114:19 146:7 156:4 157:24
limits 151:24
line 16:8 217:18
lines 218:4
link 96:19 150:6 213:14 213:24
links 213:20 214:4
lipophilic 170:12
list 6:4 15:3 38:21 42:18 50:24,25 51:6 51:9 69:9 93:19,23 94:13 105:8,20 106:3 106:9 169:7 173:22 174:9 181:17 182:3,4 182:5,10 186:16 193:8 221:8
listed 29:3 96:14 120:2 173:21 185:4 230:24
lists 182:8,12 182:13
liter 67:24 68:16 72:10,13 194:15,21,23 195:3 197:5,6 198:25

literature 17:9 21:20 31:3,11 31:19,23,24 32:1,11,14 33:21,24 34:19 35:8,18,22,25 35:25 36:4 50:18 56:16 57:14 62:19 76:21 79:20 95:6,9 97:17 99:7 109:5 110:22 115:9 147:4 160:25 169:2,5,8,16 173:15 181:4 196:14 207:2 211:19 213:19 213:23 214:2,3
litigation 1:6 4:9 10:19 15:14,18,23 16:22 17:3,24 19:18 23:15,24 27:15 45:21,23 49:21 53:5 57:10 231:24 240:7,23
little 18:10 36:25 68:4 74:4 82:20 210:4,8
liu 161:17 162:5 167:5,17
live 135:23 227:7,8
lived 129:5,12 192:24 200:3
liver 177:5
loaded 141:16
located 128:1
location 36:23 158:11,16,18 193:2
locations 37:9 127:25 135:24
long 18:17 40:18 41:22,24 100:9 160:19 230:11 232:7 232:23
longer 40:19 41:18 64:5 72:14
longitudinal 41:10
look 32:14 33:10,13 64:20 70:13 75:9 87:17 89:25 97:21 100:23 120:23 134:4 145:15 160:24 173:24 211:21 213:13 218:19 238:22
looked 14:13 24:8 68:18 80:1 107:24 157:2 174:21 188:4 210:8,15 211:19 224:17
looking 62:20 65:15 70:15 71:10 106:21 107:1 119:12 129:18 131:8 140:9 142:6 147:14 156:9 159:14 164:20 167:11 182:23 217:5
looks 23:25 164:3 179:13
loss 115:7 134:14 149:9 149:15,19 163:17 165:6 165:10,13 168:2,6 208:7 243:13 244:24 245:1
losses 164:25
lost 144:3
lot 40:22 210:9
louis 18:8
lower 51:21 52:1 103:5 108:1,21 147:19,23 148:17 198:17 236:10
lowered 148:8
lowest 67:15 68:7
lrrk2 223:18
lucio 185:25
lunch 131:16 131:19

m

m.d. 1:13 2:2,9 8:17 9:22
madam 9:14
made 90:10 157:3
magnitude 139:6 145:1 155:9
magnitudes 223:22
mail 4:12
main 128:8 146:22 229:11 230:10
mainside 128:3
maintain 11:1
major 142:25
majority 71:19 172:6 228:8
make 10:25 11:18 13:17 21:12 68:10 102:12 117:12 131:10 139:13 147:13 153:9

164:15 173:3,7 192:20 215:12 228:14 235:18 235:20
makes 61:8 170:8
making 21:7 49:18 50:24 51:4,9 77:20 78:18 140:10 156:10 183:14 203:13 221:7
male 206:12
maltbie 1:24 2:5 9:18 249:2 249:23
manages 239:7
maneuvers 230:4
maps 192:22 193:1,5,6,10,17 193:21,25
marie 10:9,10
marine 7:7 150:17
marines 6:12 6:16 104:17 115:13 121:11 121:14 123:15
mark 97:24
marked 13:24 22:15 23:19 28:9,12 93:8 93:13 97:7 115:19 123:11 125:21 132:18 150:21 157:9 161:23 174:12 188:23 189:13
marking 115:21
mass 194:5,8
material 182:8 182:9,11
materials 5:23 6:3 14:17 20:7 69:9 79:11,17 93:18,18,23 94:2 114:1 174:8 181:16 185:5 187:4 193:8 247:11
math 166:9
matter 8:13 22:1 237:13
matters 236:21
maximum 62:22,25 197:15 199:4
mayo 236:14
mcl 63:4 155:9 155:12 200:24 201:5,14,24 202:3,4,6,9,15
mcls 62:18 63:6 200:13,15
md 5:13,16,20
mean 15:8 18:24 32:6 48:10 51:23 52:14 57:16 63:12 70:20,23 74:22 77:14 79:22 86:10 87:1,22 93:3 94:24 103:12 104:4 113:7,16 118:8 164:10 171:6 173:4,5 189:9 203:9 216:14 234:8
meaning 34:2 49:11 63:14 69:25 71:5 73:17,24 106:24 140:13 156:6,10 168:15 169:12 170:12 176:11 204:18 209:3 234:10
meaningful 70:21 88:1,3 122:6 138:12 154:9
means 48:19 56:7 70:25 85:25 102:19 163:9 164:17
measure 64:21 208:9
measured 71:18 116:2 119:21 125:9 137:11 157:2
measuring 162:25 163:2
mechanism 99:19
mechanisms 181:6 223:20
mechanistic 83:14 92:6 98:23 155:19 182:25 184:17
media 21:18
median 62:20 64:19 67:23 68:14 72:10 155:8 199:10 199:17 200:1
medical 13:2 17:22 29:2 47:9,17 48:2,9 48:14,15,22 49:2,7,10 53:18 55:25 57:5 161:6 206:10 212:11 226:19 227:18 228:11,19,24 237:21 238:11 238:14,19,23 238:25 239:3,7 239:18 243:20

245:13 246:1
medically 233:15 237:10 239:18
medicare 151:17,21 152:2,20 157:25
medication 38:25 215:7,14
medications 12:24 215:4,10 220:13
medicine 56:5 78:17 79:3 92:5 201:19 203:22 210:14
meet 17:10,15 57:22 140:16 145:24
meeting 19:9 19:11,15,24 21:25 22:9 50:7
member 19:19 135:13
members 7:6 123:15 133:6 150:17
memorized 55:10
memory 12:25 13:3 21:3,22 229:19
men 206:7
mental 229:20 229:22 230:13 235:22
mention 21:18 216:10
mentioned 33:15 38:6 40:2 71:11 74:10 88:17 95:8 114:15 115:2 130:25 135:7 159:23 162:5 179:13 185:23 188:14 195:15 196:1 198:20 206:7 215:9 219:18 243:23
mentions 26:4
mentor 18:7,9
merely 60:15
messing 101:5
met 16:15 17:12,17 20:11 146:6,9
meta 80:20 83:15 89:15,21 112:23
metabolic 171:22
metabolism 7:15 173:17 174:5 175:15 180:2 181:6,12
metabolisms 175:12
metabolite 82:5 82:9 114:21 115:3 146:18 146:22 177:10 177:12
metabolites 82:16 170:15 175:20,22 177:7,11
metabolized 82:5
metals 211:11 211:13
methodology 63:5 95:15 196:7,18 237:15
methods 33:9 33:12,16 69:21
metric 194:6,14 195:6,22 196:13
micalane 231:15
mice 162:16 164:25 165:17 166:16 180:4
miceli 3:12 8:24,24 19:14 19:23
micro 210:11 214:4
micrograms 67:24 68:16 72:10,12 166:3 166:10 194:15 194:21,23 195:3 196:24 197:5,6 198:5 198:5,11,12,14 198:15,18,25
mics 247:18
middle 10:8
midway 110:13 164:2
mike 3:8,9 9:1
milberg 3:10
milberg.com 3:13
mild 87:16 235:11,23
milestones 20:5
mili 192:17
milieu 149:25 213:4
military 36:17 38:24 123:20 154:14
miller 185:2
miller's 185:8 185:13,15,19
milligrams 162:17 165:1 166:10,16

mimic 214:14 215:13
mimicking 168:5
mimics 215:2 220:11,12
mind 109:16 118:21 125:8 148:4 158:13 179:2 186:11 187:12 226:2 242:10
mine 33:24
mini 235:22
minimum 66:4
minute 23:9 109:17 215:19
minutes 19:14 176:23 215:18 241:16
mischaracteri... 200:1
misclassificat... 127:21,23 130:21 133:23 144:16
misclassify 129:14
misheard 142:10
missing 215:13
mistake 198:16
mistaken 159:8
misunderstand 196:4
mitochondrial 171:25
mix 41:17 74:17,20 129:7 204:8 226:12 227:3,10
mixed 149:6 211:21 238:15
mixture 73:23 77:12,15 78:12 79:21,22,22 188:13 191:17
mixtures 78:11 156:20
mm 81:5 108:6
moca 229:22 236:6
model 65:19 78:2 92:3 189:23 198:3 198:10,17
modeled 72:11 72:19 75:14 118:10
modeling 125:13
models 64:17 78:7 82:11 129:20 178:4
moderate 87:16
modern 210:13
modest 87:12
modifications 91:10
modified 135:1
modifiers 218:24
modify 178:14
molecular 184:14
molecule 66:17 67:5,13 82:10 171:16 172:15 172:16 173:8
molecules 82:4 170:8 173:6,9 173:10,13
moment 53:10 103:4 116:12 144:11 164:7 173:25 247:19
moments 11:2
month 163:20 167:13
monthly 67:24 68:15 116:18 155:8 239:10 239:15
months 40:1,9 72:9,14 162:16 162:21 163:6,7 163:9,19 165:2 165:10 166:20 195:11 200:4 234:5,13 243:3
montreal 229:16,18 235:21
morgan 3:11
morning 5:4 8:4,6 9:17 10:3 10:4,25
mortality 6:11 6:16,20 106:21 107:1 108:17 115:13 118:16 120:25 121:3 121:16 122:8 122:23 123:3 123:14 124:3 125:24 126:12 129:17 131:2,3 131:4 151:13
motor 160:23 160:23 229:9 229:12 230:7,7 233:25,25 246:17
mountain 10:13
mouse 7:16 173:19 174:6 178:4
move 88:6 181:2
movement 16:6 16:14 18:13 29:18 30:2,19 31:22 38:21

217:19 227:17 237:19
moving 121:15 148:21
mppe 115:8
mptp 82:10 210:22
mri 214:25 215:1
msph 7:19
mucosal 213:7 213:20,25
multifactorial 204:4
multiple 45:19 65:14 89:23 145:6 209:22 214:7 222:8
muscle 228:6
mutation 209:25 216:14 216:16 218:22
mutations 209:14,22 210:1 216:9 236:12
myriad 32:7

n

n 8:2
name 8:7,21 9:17 10:5,8,16 46:2,4,5 128:21 181:9
207:18
named 249:13
names 45:16 68:23
nano 210:12
narrower 137:16
narrowing 152:25 153:3
nastasia 240:4 240:6
national 181:19
natural 209:6 243:10
naturally 208:6
nature 222:16
navy 6:12,16 115:13 121:10 121:14 123:15
near 112:4 212:7 242:20
necessarily 37:8 61:22 87:16 103:17 120:19 135:18 145:19 148:14 201:25 214:20 222:6 224:1 225:24 227:25
necessary 62:6 65:1 233:15 237:10,17 239:19
necessitate 37:8 66:4
need 66:14 94:1 102:3,15 145:19 164:11 176:21 213:21 224:9
needed 25:2 64:11 168:5
needs 70:22 179:5
negate 224:23
negative 95:1
nephro 179:21
nephrotoxicity 177:7 179:11 179:15
nerves 229:5
neuro 179:20 208:15
neurodegener... 167:24
neurologic 96:7 229:3,20
neurologist 29:18
neurology 5:14 18:15,19 73:1 210:14
neuron 164:24 165:6 208:7
neurons 114:24 115:7 163:18 165:4 168:3,6
168:16 214:19
neurotoxicity 96:8 179:10
nevada 1:24 2:8 9:20 249:4 249:24
never 30:24 31:2 34:15,18 35:7,10,17,21 35:24 36:3,7 110:9 113:3 151:21
new 24:25 94:2
nexus 47:18,21 47:25 50:11
nielsen 68:25
night 93:23 94:3,12 182:6 247:10
nigra 82:6,8 115:6 173:11 177:15 214:18
nigral 168:3
nine 80:3,4 82:1
nissl 163:3
nod 11:24
nods 105:16
non 178:4 187:5,23 188:7 188:11 230:7 233:25 246:17
nonspecialist 227:13

normally 84:3 229:25
north 1:2 3:3,8 3:12 8:16 10:20
notable 70:17
note 24:25 75:11 180:1 183:20 232:12 243:6 244:17
noted 155:25 243:2 244:9
notes 19:10 114:17 232:25 233:10 234:11 244:10 245:16
notice 5:8 14:7
noticed 172:2
noting 182:5
notion 171:15
notwithstandi... 218:22
november 19:21 22:6 28:23
nrc 181:22
nrc's 181:23
nuanced 149:5 205:1,16
nuances 171:8 217:16
null 111:24 112:4,5 135:14 144:17,25 154:23 249:18
number 9:15 9:19 45:12 56:20 62:11,14 65:7,10 66:5 106:22 119:24 120:15 124:4 148:2,8 163:2 163:11,24 186:4 195:11 219:17
numbers 120:11 142:4 158:4 192:21
numerical 238:9
nw 4:5

o

o 4:10 8:2 167:22
oath 11:3,5
object 13:10 20:24 24:13,20 29:25 30:18 36:15 38:19 39:22 41:6 42:23 47:24 49:3,25 51:10 53:14 54:4,18 54:24 55:6 56:2,13,18,23 58:11,21 59:3 60:2,8,18 61:9 61:16,21,25 63:9,18,22 65:3 66:10 70:9 71:15 72:6 73:5 76:25 77:8,24 78:15 80:14 81:11,19 83:5 83:23 84:6,14 84:20 85:4,23 86:9 87:6,13 88:5,16 89:1 89:18 90:25 91:20 92:1,12 92:20 93:1 94:4,6 95:3 96:20 97:15 100:6,11 101:12 102:2 102:24 103:7 103:18 104:2 104:18 105:11 105:22 106:4 106:11 108:2 109:11 113:8 114:2 116:3 117:16 118:3 120:7,17 121:24 125:3 126:23 129:16 130:16,23 133:24 136:17 137:13,18,23 138:5,15 139:3 140:8 141:1,14 143:16 144:10 146:1,25 153:5 153:14 155:6 155:14 160:7 160:16 161:11 170:6,21 176:5 177:22 178:21 180:12 182:20 185:9 187:7,20 190:24 191:11 191:14,19 195:13 196:9 196:21 200:19 201:10,17 202:1,11,17 204:5 205:7,14 205:25 206:19 207:16 211:17 213:10 216:23 218:10,17 220:4,24 221:24 223:6 223:25 225:19 226:8 241:10 247:14
objection 13:12 62:7 65:13 66:19 67:1,7 68:12 101:21 146:16 171:17 179:22 201:1 219:7 224:8 242:9 243:11

243:25 244:7
244:22 245:19
246:3,11 247:2
objections
14:21 21:6,8
observation
228:9
observations
233:3
observe 159:11
228:23
observed 9:11
71:7,9 85:14
86:22,25
156:22
obtain 215:1
occupation
38:23 211:12
occupational
119:6 133:11
133:15 136:8
211:3,6,9
220:13 223:21
224:6
occupations
38:2,23 73:9
occur 29:4
143:7 210:1
occurred
127:13 194:2
odds 70:17
80:1,3 81:23
81:24,25 85:8
86:14,18 87:8
87:8,12,17,25
88:9,11 103:10
108:16 139:16
139:17 140:11
141:7 200:4
offer 24:18
61:24 65:10
offered 43:7
44:4,5,7,7,25
59:9 237:25
246:19
offering 59:15
75:24 76:11
office 19:7
239:6,9 240:4
officer 29:2
oftentimes
32:11
oh 28:5 39:19
44:20
oil 162:19
okay 10:8,10
12:10,11,19
13:9,14 14:8
14:11,14,16,24
15:8,19,24
16:12 17:1,5
17:10,15,19
18:1,6,16,21,25
19:9,13,16,22
20:9,13,17
21:14,24 22:7
22:10,13 23:9
23:17 24:1,24
25:3,16 26:6
26:12,20,23
27:2,5,18 28:1
29:12,20 30:5
32:5,20 33:1
35:3 36:13,21
37:5,20 40:11
41:2,21 42:1,6
42:10,13,21
43:16 44:1,7
44:15,24 45:7
45:16 46:4,13
47:4,21 48:20
48:24 49:17,23
50:4,8,11,15,20
51:4,20 52:7
52:19,22 53:10
53:20 54:2,8
54:21 55:19,24
56:11 57:4,25
58:25 59:20,21
60:5,25 61:7
61:13 62:5,16
63:3,6 64:25
65:9 66:7,17
67:4,15 68:6
69:2,11,14,18
70:7 71:12,23
72:20 73:1,13
73:15,21 74:4
74:16,21 75:1
75:9,16 76:21
77:4,11,20
78:6,10 79:19
80:6,22 81:4
82:18 83:1,18
84:3,17,24
85:12 86:4,21
87:4,21 88:2
88:11,24 89:3
90:6,16,22
91:17,24 92:9
92:19,24 93:16
93:20 94:16,22
95:11,22 96:9
97:3,17,20,22
98:2,5,20 99:5
99:10,14,23
100:9,15 101:8
102:21 103:14
103:24 104:10
104:14 105:2,6
105:25 106:7
106:19 107:18
107:24 109:1,8
109:15,20
110:2,5,12,21
110:25 111:19
112:6,12 113:6
113:14,18
114:9 116:7,13
117:3 118:19
119:23 120:13
121:2 122:4,7
122:19 123:5,9
123:23 124:2,7
124:24 125:2,6
125:15,19

126:4,8,15,18
127:10 128:11
128:22,25
129:13,24
130:7,12
131:12 133:10
133:13,17
134:2,5,11
135:7,17
136:13,14
137:1 138:11
138:18,22
139:1,10,19
140:5 141:10
141:25 142:17
143:12,20
145:3 146:12
146:21 147:6
148:1,16,21
149:18 150:9
150:15 151:3,7
151:20 153:2
153:18,23
154:2,17
155:17,21,25
156:25 157:5
157:23 158:3,5
158:19 159:6
159:11 160:22
161:9,17,21
162:4 163:14
164:6,24 165:7
165:21 166:9
166:13,25
167:3,8,17
168:10,21
169:8,17
170:17 171:3
171:10,14
172:2,11,14,22
173:15 174:18
175:7 176:2,25
177:18 179:6
180:8,15 181:4
181:19,23
182:1,7 183:7
183:18,24
185:1,18,24
186:2,7,18,20
186:25 187:17
187:21 188:6
188:18 189:7
189:23 190:14
190:21 191:5
191:22 192:13
192:22 193:6
193:10,19,25
194:8 195:2,9
195:21 196:13
196:18 197:3
197:25 198:21
199:22 200:12
200:15 201:7
201:13,22
202:14,20,25
203:6,9,22
204:12,18
205:5,12,23
206:4,17 207:5
207:10,21,24
208:3,11 210:3
210:11,17,24
211:13,25
212:9 213:7,19
214:7,22 215:3
215:16 216:17
217:2,10,13,24
218:14 219:4
219:14,24
220:2 221:19
221:22 222:4
222:10,14
223:1,21 224:3
224:12,22
225:1,4,14,23
226:18 227:4
228:1 229:16
229:24 230:11
230:18,25
232:1,2,22,25
233:5,10,13
234:8,15,19,23
235:7,25 236:9
236:19,22
237:1,20,24
238:3,10 239:3
239:12 240:21
241:7,11,14
242:22 244:3
247:16
old 8:12 161:2
161:3 182:24
206:17
olive 162:19
omit 227:24
228:6,7
once 41:12,15
one's 206:1
236:7
ones 44:15
52:22 69:14
71:11 128:14
157:24 182:17
221:9 240:15
ongoing 15:3
onset 160:22
161:14,15
207:7 225:13
open 94:1
247:9,13
opens 32:8
opine 23:4,5
53:11 54:2
237:12
opined 57:25
58:19 239:17
opining 49:9
131:2
opinion 43:7
44:25 52:10,13
58:6,25 59:19
59:24 60:25
65:10 66:3,7
66:23 74:5
75:24 76:11,17
83:1 110:19

146:19 155:12 156:10 164:10 180:9 224:12 237:24 238:23 243:8,19,19 245:10,12,22 245:25
opinions 24:11 24:17,21 25:2 58:2,15 59:10 129:22 131:10 178:7 190:4 246:19
opposing 241:13
opposite 51:23 86:1
option 247:13
options 57:22
oral 162:9,20
orange 37:11 37:14
order 50:24 62:13 63:1 66:14 102:12 108:12 198:1 221:11
ordinarily 238:25
organ 181:5
organic 211:13
organs 179:14
original 249:17
originally 182:22
originating 236:3
outcome 25:18 249:14
outcomes 118:20,24 119:13,21 196:20
outdated 217:19
outset 21:13
outside 15:13 17:6 48:22 50:11 57:10 75:17,22 77:1 96:15 102:10 104:21 197:18
overall 129:22
overbroad 66:19 71:16 205:15
overcome 221:22
overlap 229:4 229:14
overreporting 144:9
oversight 239:9
overstepping 165:5
own 32:21 50:5 91:12 97:2,4 110:12,22 112:7 183:22 184:8 192:17 216:18
oxidated 175:16
oxygen 171:24 172:16

p

p 4:10 8:2 70:12,19,24 140:10,11,13 140:15 141:4 142:17 148:14 187:9
p.m. 131:18,20 131:20 132:2 180:19,21,21 180:23 215:22 215:24,24 216:1 241:19 241:21,21,23 247:22,25
pace 11:21
pacific 8:1
page 5:3,7 6:2 7:2 11:1 13:17 15:2 24:6 44:21 45:18 58:3 75:4 80:25 95:19 96:2 97:23 98:7 99:15 104:22 107:2,4 107:5,7,19 109:16 110:12 111:5,20 112:1 112:12,13,17 113:18,21 114:10 116:13 118:7 119:1,23 121:2,15 122:17 124:13 125:10 126:16 127:15 134:5 136:21 137:2 142:5,24 146:2 147:7 152:5 154:2 156:12 159:17,18 162:13,24 163:24 164:12 164:16 165:8 166:4 167:17 169:18 172:7 174:20,21 175:7,8 183:23 186:3,4,6,7 190:1 197:3,10 198:22 220:8 230:3 233:9,21 233:23
pages 115:1 174:22 217:15
pairs 132:25
panel 106:8

paper 33:11,14 110:20 111:12 111:18
paragraph 98:8,21 99:14 107:7 108:8 109:25,25 110:4,13 112:1 112:17 113:19 114:15,16 116:14 119:2 127:16 134:6 134:20 143:18 144:12 154:3 156:12 172:6 172:10 198:22 233:24
parameter 110:9
parameters 112:7,8 113:24
paraphrasing 113:12 218:3
paraquat 45:21 45:23
pardon 242:20
parkins 31:18
parkinson 7:4 7:6 23:8 58:13 58:22 60:23 76:19 79:9 96:6 106:25 161:15 178:4 200:5,8 205:4 205:19 206:8 212:19 213:5 214:13,14 215:15 217:21 218:5,25 219:11,19 220:6 246:17
parkinson's 7:9 7:12,21 13:21 16:3,10,15 21:17,19 27:19 28:2 29:13,16 30:16,21 31:22 32:12,13,18,23 35:19,22 36:1 36:5,8 37:10 37:11,21 38:1 38:4,13 39:15 39:18 42:2,8 43:13 44:8 45:1,9 55:22 58:1,8,10,20 59:2 60:1,7,15 60:16 61:2 62:6 64:7,13 65:2,12,21,22 65:22 66:9,18 66:24 67:6,18 68:10 69:7 71:13 72:3,21 72:24 73:2 74:8 76:2,15 76:24 78:14 79:21 80:5 81:10,17 82:7 82:11 88:22 95:7 96:19 97:18 98:14,16 99:1,12 100:3 105:13 106:22 119:15,16,18 119:22,24 120:15,22 122:21,22,23 122:25 123:2,6 124:5,6 126:5 127:7 129:19 132:21 139:21 140:2,7,23 143:6 147:25 148:18 149:4,7 149:20 150:2,7 150:11,16 155:22 157:13 159:10,12,24 160:2,5,12,19 160:23 161:1,4 161:19 162:1 165:15 168:7 168:22,25 170:10 173:12 177:15 184:15 187:6,15,19,25 188:8,13 189:18 190:23 199:13,19,24 200:9,10 203:1 203:2,18,19 204:4,6,14,14 204:20,22,24 204:25 205:6 205:13,20,24 206:6,14,18,23 206:25 207:4,7 207:12,22,25 208:12,18,21 208:24 209:3 209:10,12,13 209:14,16,17 209:19,24 210:7,15,18,20 210:25 211:2,5 211:16 212:13 212:16,21,25 213:3,8,14,15 213:21,25 214:4,8,16,20 215:5,12 216:12,22 217:14,25 218:8,15 219:5 219:16 221:14 221:16 222:5 222:13,15,22 223:3,5,11 224:15,21 225:7,12,15,25 226:6,12,24 228:12,23 229:1,4,7,8,12 229:25 230:6 230:10 233:14

233:18 234:16 234:20,25 235:5 236:10 236:14,16,19 236:23 240:13 242:24 243:10 243:13,16 244:5,15 245:7 245:23 246:6,8 246:21
parkinsonian 220:16
parkinsonism 149:12 212:18 214:13,19 215:7,13,15 220:12,12 224:18
parkinsonisms 203:20 212:22 212:23 213:1
pars 214:18
parsed 74:1
part 31:13 33:22,23 36:24 45:23 55:12 65:25 90:19 92:7 114:14 179:14 181:11 192:18 203:3 214:25 229:9 229:17,19 240:6
participating 20:8
particular 15:22 16:4,25 38:9 40:6 42:16 60:20 84:10 85:20 86:16 90:4 91:6 114:6 119:19 217:3 238:20
particularly 48:21 135:18 244:4
particulars 91:3,14 92:22 103:1
parties 240:2 249:12
parts 37:2 62:18 75:6 194:16 198:24 199:6,11,18
party's 239:18
past 234:14
pat 3:4 8:21
pathology 184:15
pathways 171:22 175:17 177:10
patient 33:7 37:16 38:10,17 41:15 216:18 220:1 225:24 226:5 228:12 230:5,13 231:3 237:22
patient's 37:21 42:2,8,16 48:2 196:19 219:16 221:10 225:25 226:6
patients 32:11 37:25 38:2,11 39:4,11,17,24 40:2,6,8,12,16 40:17,18,20,25 41:4,5,10,12,13 41:23 42:11,14 47:18 55:20 73:2,8 216:24 219:18 227:6,8 235:3,4 239:1 239:3 246:6,7 246:13
patterns 209:20
pause 177:2
paused 208:25
pay 238:19
payment 25:18
pc 5:13
pce 13:21 23:6 35:22 39:7,11 42:6,8 58:7,16 58:19 59:1,24 60:6,13 61:1 64:8,22 65:1 66:24 68:6,7 68:19 71:20,23 72:2,11,23 73:13,17,24 74:11,18,25 75:6 77:12,16 78:12,17,21 79:23 80:2,4 80:12 81:9,16 81:18,25,25 82:3 95:7 96:3 96:23 98:14 99:11,16 100:3 114:22 115:3 117:6 120:13 130:3,10,20 131:1,9 136:14 136:24 137:6 139:11 140:6 140:11,22 141:10,20 142:1,3,14,15 145:23 146:7 146:13,24 156:1,9,18 166:3 169:4,10 169:19,22 170:4,5,10 171:21 172:4 172:18 173:8 175:13,19,24 176:4 177:5,8 178:20 179:20

180:10 184:16 187:15 189:11 190:10 191:5 192:2 198:11 198:15,23 199:3,10,17,23 200:2 222:9 223:24 224:5,6 224:17,24 246:25
pcp 166:6
pd 13:22 147:10,19,21 147:24 148:2,8 148:9,22,24 156:4 159:19
peek 230:1
peer 31:2 34:18 35:7,17,21,24 36:4 105:3,5 196:14
peg 56:11
pelvis 243:5,9
penalties 11:7
penalty 248:1
pendleton 115:24 116:24 117:3,6,10,13 117:22 118:2 119:9 120:3,6 120:14 121:4,8 152:10 153:20 158:18 159:7
penetrants 216:11,16 218:22 223:9 223:11,20
pennsylvania 4:5
people 37:12 62:12 64:5 65:15 72:8,20 72:23 90:11 106:24 116:5 122:22,25 123:1 129:11 129:19 131:6,6 152:3 160:20 200:3,6 209:2 209:5,7 211:21 212:6 225:14 226:12 235:16 236:15 240:16 240:17,19 243:15 245:1
peoples 235:5
perc 13:21 96:3 137:6 142:14 145:8,11
percent 26:15 27:1 43:1 56:6 70:25 71:2 85:25 108:11 108:21 138:24 139:11 147:21 147:21 148:23 148:25 163:11 163:18,19 165:9 168:2 216:16 218:15 218:21 223:12 225:11,12 236:15
percentage 26:12,19,25 56:11 147:23 148:13,17
percentages 218:20 219:4
perchloroeth... 13:21 96:6 181:7
perfectly 124:4
perform 22:24 42:10 53:18 94:22 100:10 109:7 184:22 203:3 222:24 228:19 229:25 231:7 236:4
performed 95:6 95:16 183:22 184:7,12 198:7 228:9 231:14
performing 120:9 231:1
period 103:6,10 160:6,19
periods 40:19
perjury 11:7 248:1
perlmutter 18:4,18,21 19:7 217:24 233:17 235:17 243:2
perlmutter's 161:7 234:11 234:24 242:5 244:10 245:15
permanence 168:13
permit 230:25
peroxide 172:15,19,25
person 16:22 17:15,17 204:13 209:14 211:11 222:21 222:22 226:16 227:2,8,14,19 231:1
personal 41:10 75:21 109:3 238:8 240:24
personally 2:8 19:6 32:9 109:7 196:15
personnel 6:12 6:17 115:14 122:8
persons 68:1,3 129:5,7 149:11 209:22

pertinent 16:3
pesticide 38:24 210:24 211:19
pesticides 211:1,15,22,23 212:4
peterson 231:17
ph.d. 1:13 2:2,9 8:17 9:22 30:1 171:6
phd 5:13,16,21 7:19
phillips 3:10
phrase 55:25 57:4 85:24 113:6,9
phrased 74:9
phrasing 74:16 141:3
physical 228:21
physician 18:13 49:13
physicians 18:2
pick 173:13
picture 89:22 102:5
piece 131:8
pieces 155:20
pigeonholed 195:17
pillars 229:12
pizoli 68:24
place 144:3 169:21 247:19
placebo 90:13
places 156:7
plaintiff 3:2 6:5 43:24 46:6 78:24
plaintiff's 8:22 8:25 9:2 45:25
plaintiffs 17:24 22:8 27:21,23 43:23 45:19 231:23
plan 12:13 19:3 19:5 24:18 100:10 237:5,9 237:25
plane 16:7
plans 237:16
plant 128:5
plants 193:2
plastics 210:12 210:12
platt 3:20 9:6,6 247:7
plausibility 115:4 183:12
plausible 99:19
play 75:20 160:1
played 203:20
please 8:18 9:14,21 10:5 10:11 11:23 12:4,9,16 21:7 76:7 97:23 107:5 112:13 121:2 139:24 144:11 175:8 196:5
pllc 3:6
plus 115:8 163:1 234:14
pneumonia 245:4
point 17:11 21:24 90:10 94:6 103:21 112:18 113:18 128:4,15 129:1 129:9,14,21 130:1,5,8,13,18 130:22 131:5 135:13 141:5 144:4 167:11 197:19,21 207:24,25 212:19 217:17
pointed 149:14 195:4
points 230:10
poison 61:8 62:13
pollution 210:6
poor 90:18 245:1
population 65:15,25 70:18 72:8 88:21 121:6 123:20 133:18 138:3 138:13,14,20 138:21 151:3,4 151:12,24 152:13 155:19 157:16,20 158:2 225:10
populations 62:20 63:1 65:23 70:16 104:24 117:13
portion 143:14
posed 32:10
positive 95:1 163:17 164:24 209:11 212:2
positivity 164:16
possibility 85:2 221:23 225:24 226:5
possible 42:15 83:18 84:25 85:17,19 103:9 103:22 133:25 135:11 179:18 193:4 229:3
postage 26:1
postman 3:14
postulated 213:14

postural 242:23 243:13
potential 40:13 62:23 195:22 204:8,8 220:20 220:22 221:1,7 226:4
practical 70:22
practically 70:17
practice 25:4 31:13 32:1 33:22,23 34:5 36:7,11 37:20 39:24 41:8 42:1,5 50:13 51:13,16 55:20 57:8 73:1 183:8 196:19 196:25 213:17 215:1 216:25 219:14,25 228:5 238:24
practices 34:3 235:18 237:21
precise 88:25 216:21 225:25
precision 108:14,19 109:22 110:16 111:10 137:12 137:17,22 141:20
preclude 225:24 226:5
predates 99:7
predicate 176:6 176:19
predisposes 245:1
predominant 129:25 130:5,9 130:11
predominantly 233:6
prepare 16:18 16:20 17:7,11
prepared 77:2 237:6
preparing 16:24 54:14
presence 120:13 221:25 225:23 226:4 232:17
present 4:13 19:13 117:10 131:10 156:15 223:23 232:5,8
presentation 218:5 236:23
presentations 221:11
presented 111:18
presenting 242:17
presently 99:25
presumption 105:8,20 106:2 106:9
presumptive 47:23 105:13
prevalence 209:17 236:10 236:17 246:16
prevalent 52:1
previous 47:1 67:11 132:5 196:23 216:5
previously 52:9 52:12,20 151:8 158:23 188:14 198:20 231:10 247:15
primarily 130:18,20 179:11 246:25
primary 165:14
principal 30:24 34:15
principle 61:7 61:14
principles 115:2 183:11
print 93:24
prior 22:25 39:20 80:23 125:9 177:24 181:1 226:19 239:16
priori 219:12
prioritized 51:5
priority 42:18 50:25
privileged 219:1
probability 243:21
probable 78:13
probably 10:23 55:23 57:9,11 129:10 182:3 226:22
probe 36:18
problematic 242:21
problems 242:15 244:11
procedures 10:24
proceed 9:16
proceeding 249:13
proceedings 249:10
process 33:3 220:11 221:5 228:11
processes 220:15
produce 14:17 96:17 169:13

171:16
produced 94:12
producing 168:1
production 5:10 14:18 136:24 247:10
productions 14:12
products 45:21
professional 15:16 21:21 47:9 48:2 237:18
profile 103:5 103:12
profound 168:6
prognosis 233:24
progress 235:6
progresses 235:6 244:5 245:7,9 246:8
progressing 234:25 235:1 242:25
progression 7:9 157:13 159:9 159:15 208:16 218:5 233:24
progressive 234:3
projects 26:18
prolonged 103:6 167:25
prompted 178:1
prone 69:5
pronounce 162:11
proof 52:13,14 54:22
properly 11:19
proportion 143:13,24,25
pros 91:11
protect 63:1
protective 119:18 200:23 201:4 206:14 208:15,15
proven 202:22
provide 41:9 85:13 101:9,19 101:25 107:22 137:11 195:10 195:14 232:11
provided 17:19 20:2 50:9 54:9 96:25 108:12 112:22 133:14 134:3,18 182:5 189:24
providers 152:4
provides 83:12 175:11
proxies 133:8 135:8
proximate 211:8
proxy 133:14 133:22 134:18 134:22 135:5 135:13 143:13 143:23 144:4,7 144:8,19
psychiatry 5:14
ptelan 3:5
pubic 243:5
publications 31:12 33:2 57:6 172:11
published 31:2 34:18 35:7,17 35:21,24 36:3 57:12 110:20 122:7 171:10
pubmed 95:17
pull 228:7
pure 81:17,18 214:14
purely 85:9 183:17 227:9
purported 193:11
purpose 11:9 105:6,19 205:17 219:11
purposes 83:7 237:23
pursuant 5:9
push 227:13
pushed 106:8
put 70:22 188:16
putting 219:4
puzzle 131:8 155:20

q

qualified 29:23 48:1 203:6
qualify 54:16
qualitative 175:18
quality 33:10 33:14
quantified 207:1
quantify 190:17,19
quantitative 175:18
quarterly 116:23
queried 209:2
question 11:23 12:4,10,15,16 13:13 31:17,20 32:4,9,15 33:7 34:1 35:6 38:13 42:6

44:23 49:4 55:7,8,9 59:8 59:17,22 60:9 60:11,21 62:1 62:11,14 63:23 65:4 66:12,15 67:4,9,11 68:6 71:23,25 73:13 73:21 76:10 80:15,24 81:20 83:6,9,24 84:7 87:14 88:7 89:19 90:1,12 90:21 91:1,3 91:21 92:2 93:2 95:4 96:23 101:1,13 101:15 102:18 103:8,19 104:3 113:15 117:17 118:4 120:8,18 120:23 121:25 125:9 135:4 142:9 145:17 148:5,7 171:19 176:14,15,20 177:19,24 178:1,10,13,15 178:22,22 185:11 187:17 193:19 195:1,2 196:5,23,23 203:11,14 210:13 211:7 212:17 213:12 214:15 223:7 225:20

questionnaire 18:22 19:4

questionnaires 134:7 135:10

questions 11:12 11:20 12:5 31:24 32:5,7 32:12 40:24 54:5 94:1 109:18 132:5 164:22 174:20 175:2,5 216:5 229:21 230:19 232:9 236:6 241:4,12 242:3 247:4,11,16

quick 111:19 166:9 215:20 241:14

quickly 41:13

quite 38:1 41:17 141:8 160:19 193:4 216:11

quote 91:6 112:4,4 114:20 183:24 207:3 214:2 218:3,20

quoting 75:4 107:19 232:13

r

r 8:2 46:8 223:18,18

raise 9:21

raleigh 3:8,12

random 71:1

randomized 89:16 90:6

range 160:24 197:10

ranged 198:11

ranked 50:24 51:21,24 52:1

rapid 234:4,8

rare 56:6 90:2 207:11

rarely 73:10

rate 166:7 194:25 195:18 195:18 197:21

raters 144:14

rates 121:3 159:9,14 197:14

rather 11:24 78:23 121:11 123:6 144:21 180:2 200:8 219:9

rating 229:8 236:5

ratio 70:17,17 80:3 81:24,25 86:10,14,14,18 86:21,24 87:2 87:8,12,25 88:9,11 103:10 107:25 108:16 108:17,18,20 109:2,10 111:11 112:9 121:7,19 122:5 138:23,24 139:6,10,14,16 139:17 140:11 141:7 200:4

rationale 111:17

ratios 80:1 81:23 86:8 87:8,17 108:4 110:6 121:16 124:19

ray 15:9,10,15

rdr 1:25 2:6 249:24

reached 77:7 247:20

reaching 75:10

reactive 171:24

reactivity 164:8

read 14:11,14 14:15 17:8 21:21 31:23 75:19 76:6,8 96:2 98:9,18

98:21 99:15,21 99:22,24 100:5 100:7 107:7,14 107:19 108:9 108:24 110:13 111:20 112:20 114:16 116:15 117:1 119:2,10 124:13 127:18 128:9 134:6,12 134:21 135:4 137:5,9 142:24 143:10,19,19 143:21 144:2 144:13 152:6 152:15 154:4 154:15,16,19 154:24,25 156:13 162:15 162:22 163:14 167:19 168:8,9 169:5,16,18 170:1 171:1 175:9,25 176:1 176:15 179:1,2 179:5,17,23 180:8 183:25 185:6,15 190:2 190:12 198:22 199:7,9,14 232:13 234:1,6 234:11 247:5
readily 169:22 170:13
reading 32:8 75:2 96:2 109:5 116:11 152:5 177:16 181:11 249:21 249:21,22
readings 70:4
reads 98:8 99:15 107:7 167:19 175:9
real 111:19 177:18 215:20
really 21:16 119:20 120:23 213:2 219:1 236:6 238:7
realm 211:19
reason 13:5 36:21 37:5 40:5 42:9 112:11 120:19 146:22 153:6 180:9 208:25 212:1
reasonable 11:21 26:5 49:2,7,10,15 55:25 56:7 57:5 86:5 190:7 233:15 243:20 245:12 246:1
reasonably 202:4 220:20 222:1,17 237:9 237:17 239:18
reasons 111:10 129:10 218:11
recalculate 192:21
recall 20:3 53:9 68:22 69:11,16 70:6 71:10 79:10,15 96:15 134:4 143:4 165:24 173:20 174:17 181:10 181:25 185:16 189:4 245:17
recapitulate 167:13
receive 238:13
received 25:6 26:23 93:22 151:21 157:24 162:17 247:10
receiving 164:25
recent 150:3 227:12 234:4
recess 46:20 89:8 131:19 180:20 215:23 241:20
recessive 209:21
recognize 14:4 87:24 93:14
recognized 78:11 244:14 246:15
recollection 22:5
reconstruction 125:12
record 8:7,19 10:6 11:1,17 11:18 12:13 13:16 21:6 46:17,18,23 76:8 89:7,11 93:21,22 94:8 131:17 132:2 174:24 175:1,4 180:19,23 215:21 216:1 227:18 241:15 241:19,23 247:21 249:10
recorded 151:10
records 161:6 238:14,19,21 242:6 244:21 245:15
recover 243:14
recovery 245:2
red 53:4 240:7
redirect 31:21 241:13
reduce 85:8

reducing 144:17
reductive 29:15
refer 141:4 164:11 166:2 171:8 194:18
reference 19:5 58:14 116:25 124:11 145:18 145:20 163:23 197:10 199:20 213:2 218:19 220:8
referenced 39:14 43:19,20 81:8 115:9 169:16 181:12 181:13 193:8
references 156:6 171:20 172:9
referencing 43:23 45:18 108:7,8 146:2 159:16 182:6 203:10 223:17 245:16 246:24
referred 43:14 122:10 240:8
referring 58:2 97:12 105:12 111:4 156:25 189:2
reflect 87:11 124:4
reflexes 243:14
reframe 21:7
regarded 18:12
regarding 35:18 94:2 104:15 122:8 244:10 245:10
regardless 102:14 148:16
regions 82:6 214:12
registry 13:19 151:9
regrow 168:16
regular 147:16 160:25
regularly 42:10
regulatory 78:10
reimbursed 25:21,24
reiterate 247:8
related 11:10 16:24 53:4 105:17 169:19 211:3,20 245:22
relatedly 33:20
relates 1:7 178:3
relationship 18:10 41:24 55:3,5 69:6 71:1 80:12,16 81:2 82:14 170:19 171:4 187:6,15,18,24 188:8,12
relationships 40:16,25 41:17 41:18,22 124:21 171:11
relative 77:16 86:11 87:1,2 138:23 139:10 139:14 197:15 209:15,19 222:23 246:20
relatively 136:19,22 159:23 160:3 247:12
relaxed 52:6
relevance 70:22 113:17 115:4 119:17
relevant 33:24 37:1,2 39:15 42:16 61:11 64:6 71:21 74:10 75:13,18 82:2,7 87:18 88:1,3 92:4 95:7 104:8,9 117:7 128:14 129:21,21 138:21 143:7 147:3 164:23 177:15 183:14 186:25 205:17 216:11 220:18 221:7 223:9
reliable 89:16 89:20 90:7 195:9
reliance 5:22 93:17 110:21 113:25 143:22 144:19 169:7 173:22 182:2,4 185:4 186:16 247:11
relied 69:3 94:13 97:1 115:12 131:12 133:5 147:1 150:9,15 168:11 187:4 188:18 192:12
rely 90:14,16 90:18 94:19 146:23
relying 144:21 144:22
remaining 195:25
remember 19:25 21:2 39:25 40:3,5,7 45:16 46:3

54:1,8 80:10 96:10 142:8 156:8 193:4 238:12,17
remembered 2:3
remembering 45:12
remind 21:5
reminisced 16:9
remote 228:2
remotely 227:22
removal 116:24
remove 233:10
render 249:17
rendering 52:10,13
repeat 76:5 101:14 139:23 230:23
repeated 149:23
repeating 158:13 226:2
rephrase 24:16 58:18 59:21 187:21 201:22
rephrasing 118:21
replacement 175:14 176:3 178:18 179:18
replicated 84:18,21 85:8
report 5:16 6:5 7:19 17:2,8,9 17:14 18:22 19:1 23:10,23 24:2,11,17 27:13 29:6 43:20,21,25,25 44:4,16,22 45:3,18 47:4,6 49:24 50:18 53:1,7 54:14 58:4,13 69:4 71:8 72:17 78:2,17 79:5 79:11,16,18 81:1 87:25 94:14,20 95:5 95:12,13,20 97:1 99:11 105:20 106:18 110:12 111:20 112:13 113:10 114:1,10 115:1 115:10 118:25 122:18 123:16 123:17,23 132:12 145:16 145:18 146:3 156:3 163:23 166:1 168:11 168:12 169:17 171:21 172:3 173:16 181:20 181:24 183:19 184:23 185:1,8 185:15,17,19 185:23,24 187:4 188:2,18 189:1,3,6,17,21 190:1,15,18 194:14 195:16 196:3 197:3 198:21 213:2 216:12 217:15 218:4 223:19 229:11 230:1 231:11 232:12 233:4,6,8,21,23 244:9
reported 1:23 70:2 133:22 144:23 148:24 158:23 234:12 249:7
reporter 1:24 2:7 9:14,17,19 11:16 76:6,8 79:13 117:23 249:1,3
reporter's 249:18
reporting 133:5
reports 23:14 27:14 44:5,8 45:14,15,17 49:20 50:15 79:1 111:3 184:2 185:20
represent 8:20 10:18 100:24 136:11 165:5 182:7 190:10
representation 28:24
representative 138:14
represented 163:3
represents 88:9
reputable 227:16,18
reputation 33:17
request 12:15 13:11 14:18 36:21
requested 14:12 23:10 249:21,22
requests 5:10
requires 55:15 220:22
research 16:16 32:13 54:12 95:22 109:3,4 109:6 181:19 196:24 228:3 236:4

researching 32:23
resided 154:6
residence 192:15
residency 18:15,19
residential 135:24 136:3,6 136:12 158:11 158:16,17
respect 168:10
respecting 228:14
respective 91:15
respond 11:23 178:1
responded 14:20,23
respondents 145:5
response 71:7,9 80:9,24 81:2 81:22 124:21
responses 14:21 233:3
responsible 167:23 214:12 238:25
responsive 14:17
rest 172:6
restate 59:22 60:10 71:25 86:23 139:23
restrict 153:7
result 84:18,21 111:22 149:18 246:22
resulted 134:14 149:15
results 85:2,8 85:13 86:8 88:15 89:23 94:23 95:2 96:17 116:16 141:20 154:23 159:18 164:2
retained 22:1,4 22:8 23:4 27:4 203:15
retainer 22:10 22:18,20,25 26:4,6,7
retention 5:12
retrospective 6:18,23 62:9 62:19 71:20 143:4
retrospectively 64:20
reveal 54:5
reverse 168:18
review 17:14 20:6 25:1 31:12 33:2,21 53:24 54:16 77:5 93:24 94:23 97:18 100:16 110:22 122:13 126:2,4 127:2 145:14 173:16 174:24 175:4 176:21 181:5,23 182:3 185:8 186:18 189:20 192:22 193:10,21,25 230:7,7,8 238:10,14 239:10 242:5
reviewed 17:8 31:2 33:9 34:18 35:7,17 35:21,24 36:4 50:18 52:7,9 56:17 76:22 94:19 105:5 123:5,16 150:10 168:22 181:15 182:9 182:11 184:1,3 189:5 193:7 194:20 196:14 237:5
reviewing 34:4 105:3 146:12 177:1 238:19
reviews 31:16 89:15
revising 233:11
revisited 109:16
reynolds 7:19 16:23 17:1 72:18,19 75:13 77:25 78:1,6 118:10 165:25 166:5 188:19 189:1,3,6,8,10 189:16,24 190:3 192:12 192:19 194:4,7 194:14,18 195:7,21 198:8 241:2
richard 15:6
ridge 3:3
right 9:21 25:16 30:13 31:3 46:16 75:23 84:5 85:22 94:7 95:12,16 104:7 109:25 126:13 126:14 131:16 132:9 134:6 139:13 147:12 163:4 215:16 226:1 241:11 241:17
rigorous 69:21
risk 7:4,5,12 32:24 34:23,25

35:1,4,8,10,15 42:21,24 43:3 60:6 61:18 65:21,22 67:18 68:9 85:22 86:8,10,11,17 86:18,21,24 87:1,2,9,11,16 88:9,12,25 101:10,20 103:4,10,11,12 103:16,17,21 104:1 108:16 111:22 118:20 119:4,19 120:23,24 121:7,19 124:6 124:16,16 132:21 138:23 139:10,14,21 140:2 143:2 145:1 149:3,17 149:19,24 150:16 157:3 160:11 161:19 170:5,10 194:6 200:5 201:9,15 201:25 202:10 202:16 204:17 204:18,21,23 204:24 205:2,3 205:5,8,12,20 205:24 206:1,4 206:5,13,13,16 206:17,20 207:21,25 208:11,13,17 208:20,24 209:9,12 210:7 210:18,25 211:16 212:12 212:16 213:8 214:7 215:5 216:19 218:24 223:3 224:13 224:20 225:23 226:4,14 238:6 238:7 244:4,20 245:2,3,6

risks 62:23 70:8 85:14 101:25 102:17 102:23 104:12 118:24 244:23

rmr 1:25 2:6 249:24

rodent 167:14

role 61:3 203:20

room 231:1

root 211:7

rothchild 5:17 6:5 7:21 8:22 65:11

rothschild 8:13 9:2 23:7,11,24 29:7 37:16 44:17 47:4 54:14 58:15 59:5,7 64:2 65:16,17 71:20 74:13,20 75:11 75:15,21 76:1 76:13 77:18 78:12 79:23 90:4 118:11 129:15 155:2 161:3 165:25 189:9 190:4,9 191:23 192:2,6 192:10,16,23 193:11,22 195:23 197:4 197:16 198:4 199:1 217:7,25 220:7 222:12 222:18 223:1 225:5 229:10 230:16 231:11 232:2,6 233:1 233:25 234:2 234:15 235:7 237:6 238:13 238:16 242:7 243:3 246:20

rothschild's 18:2 49:24 65:24 72:11 74:7 75:18 77:13 128:23 131:3 166:22 189:17 192:14 198:10 221:14 222:15 224:15 224:18 232:18 232:22 233:2 233:14,18 234:24 237:16 238:3,11 244:11,21

roughly 26:24

route 213:14

routes 74:11,12 74:13 167:2 212:3

routine 41:4

rpr 1:25 2:6 249:24

rule 42:21,25 52:4 82:13 215:2 220:23 221:1,11

ruled 51:7,7

rules 10:25

ruling 86:1 220:20,22,25 221:8

run 231:6

s

s 8:2 46:8

safe 30:15

safer 212:21

sample 132:24 132:24 138:13 138:20 141:22

142:2,21 143:1 151:3
samples 133:18
samuel 15:6 131:13 132:12 150:15
santa 240:3
satisfies 66:3
satisfying 38:12
saw 40:18 244:21
saying 58:9 60:5 76:22 224:16
says 98:21 108:9 112:20 116:15 134:21 137:5 143:19 152:6,15 179:3
scale 229:8 236:5
schedule 27:5
schedules 27:6
scholar 95:19
science 56:5,24 92:5 100:13 141:16 201:19 203:23,24,25
scientific 35:14 49:8,11 53:18 56:1 57:5 79:19
scientist 29:19 40:23 49:13 153:16 169:1 203:12
scientists 153:7
scope 77:1 188:2 203:3,21
score 229:9,15 235:21
scoring 134:25
screen 213:16
search 32:1,18 94:25 95:6,8 95:16,25 96:10 96:16 169:2,8 181:4
searched 31:18 96:13
searches 31:15
second 48:25 55:3 109:24,25 116:14 124:12 134:5 137:5 143:20 154:2,3 156:12 186:9 199:16
secondarily 246:25
section 4:9 95:15 184:14 193:17 233:6
sectional 91:18
see 15:15 22:21 40:17,21 41:5 41:15,23 44:19 84:18 106:20 113:9,11,22 119:11 121:19 134:10 145:7 160:1 162:24 178:6 180:9 186:15 209:20 214:24 227:14 233:21 234:21 238:18 242:6 243:6
seeing 41:12 85:7
seeking 206:10
seen 14:8 39:17 40:12 48:15,17 50:1,16 168:7 174:16 175:3 193:5 194:15 213:3,4
seep 212:5
select 42:22
self 69:4 70:2 133:5,21 144:23
senators 106:8
send 19:6
sensation 229:6
sense 41:14 77:5 78:3 170:8 243:12
sentence 98:8 98:20 99:23 109:24 152:5 167:18 199:21
separate 45:20 157:1 233:4 239:6
separated 142:14
separately 60:14 73:19 74:3
separates 142:13
september 243:2
serious 127:19
serve 219:11
served 27:9 37:12 79:7 128:4 152:8,9 239:16
service 7:6 36:18,22 37:6 38:24 78:19 79:7 105:13 119:7 123:15 150:17 152:20 154:14
serviced 128:23
services 25:9 151:18,21 152:21 153:8
serving 26:13 27:2

session 5:4 8:4
set 230:18 237:9 249:19
sets 62:25
setting 38:10 53:17 183:17
settings 145:5
several 15:3 67:25 106:8 112:23 127:22 129:10 131:12 132:12
severe 87:16 149:18
severity 245:8
sex 205:3 206:7 206:12,12
share 73:8
shared 50:6
shed 175:22
shifting 216:17
short 40:9 41:17,22 247:12
shortened 122:25
shorter 40:19 167:12
shorthand 2:7 249:3
show 55:8 65:20 81:24 150:6 175:13 186:11 189:23 193:11 197:3
showed 69:6 193:2,22,25 200:1 223:2
showing 83:19 93:12
shown 64:7 67:16 68:8 178:17
shows 83:2 119:24
sic 31:18
side 27:25
signature 22:22 24:6 248:6 249:23
significance 106:15,25 107:12,21 122:2 139:20 140:6,24 141:3 148:16
significant 20:12 69:6 70:8,12 83:3 83:19 107:11 120:5 121:23 140:24 144:18 147:9 148:15 163:17 187:5 187:24 188:7 188:12 224:20
significantly 121:5,5 141:12 148:1,8
signing 249:21 249:21,22
signs 235:8
similar 35:6 53:15 68:13 71:14 72:4 82:3,15 96:14 120:11 135:22 159:20 169:13 169:24 170:8 171:15,16 172:4 180:11 203:10 218:6
similarities 169:3,9,11 171:4,12 172:3 181:8
similarity 170:4 171:22 180:10
similarly 170:9 170:9
simple 83:2
simply 62:13 113:11
single 66:17 67:5,12 84:4 84:12 85:1 103:4 145:4 177:20 178:18 179:18 204:12 222:23 224:13
site 117:4 128:8
sitting 52:24 57:24 96:9 185:16 189:4
situation 222:24 225:13
situational 246:13
six 145:9
size 141:22 142:21 143:1
skewed 138:20
skimmed 14:15
skimming 177:2
skip 55:11
slightly 207:8 216:17
slow 234:25 235:1
slower 235:2,6
slowing 208:15
small 26:21 70:16,18 88:21 106:22 138:20 141:22 142:21
smaller 89:23
smith 232:8
smoked 147:24 148:9,10
smokers 148:17
smoking 38:22 119:5,17 124:9 124:15 126:16

134:9 147:12 147:17 148:5 206:13
social 36:16 37:4 38:7,8,17 38:21 192:19 219:21 230:8 230:19
sole 100:16
solely 108:13 153:19
solid 227:18
solo 100:19
solvent 7:3 132:21 133:10
solvents 68:18 69:7 73:25 80:18 96:14 136:19 145:10 145:10 159:3 211:14 212:4 224:5
someone's 205:20 206:5
somewhat 140:16
sorry 15:10 28:4,5,14 31:16 47:12 48:12 78:11 80:7 82:19 87:2 95:14 111:1 112:12 116:13 122:15 139:15 142:12 142:12 143:20 150:9 162:7 164:24 166:15 166:19 176:9 186:5 198:16 208:5 212:10 233:22 240:18
sort 170:17 172:5
sound 25:16
sounds 29:15 102:20 186:1
source 74:19 110:25 111:1,8 111:11 199:16
sources 31:5 64:21 127:22 138:7 171:14
south 3:4
southern 1:3
spanish 212:20
sparks 231:19
speak 22:7 59:6 177:6
speaking 21:8 58:5 136:25 139:13 204:16 207:17
specialist 29:18 30:20 31:23 227:17 237:19
specialists 16:14 217:20
specialty 30:16
specific 6:8 15:17 21:22 31:24 37:8 45:22,24 55:20 58:6 59:6,11 59:16,18 60:20 61:4 62:2 64:11,12,23,25 65:9 67:12 69:5 73:11 75:25 76:12 78:11,22,24 83:8 89:25 90:3 104:22 105:19 108:10 110:25 111:1 115:5 145:4 155:2 173:1 175:21 179:21 181:21 190:21 195:17 210:5 210:17 213:11 213:21 217:6 219:4,13,20 228:16,18 229:1 236:19
specifically 13:10 23:11 30:17 31:20 38:16 42:21 58:9,14 69:17 75:14 79:10,15 79:20 82:8 96:12 106:1 117:5,11 123:22 131:1 165:3 177:6,14 178:24 183:10 184:13,14 185:16,22 188:1 189:5 193:13,21 199:1,23 201:11 203:8,9 210:16 211:21 213:5 218:16 226:15 229:9 236:17 246:20 246:25
specificity 64:10 183:12
specifics 20:16 36:12 60:22 66:14 84:1 85:11 89:22 227:11 238:17
specified 44:2 121:10 179:9 210:21
specify 42:25 113:19 194:25
specious 171:24
spectrum 150:1 161:14 207:7 235:5

speculate 21:3
speculating 227:10
speed 230:14
spell 46:7
spent 40:21 128:2 230:16
spinmed 29:2
spoke 192:16 192:19
spoken 16:22 106:20 209:17
spontaneous 216:8
spontaneously 210:1
spread 213:15
st 18:8
staff 24:4
stages 234:22 243:17
staggering 141:8
staging 229:15
stain 163:3
standard 22:20 31:25 42:4 47:5,9,17,20 48:9,16,25 49:20 50:13,17 51:16 52:6,15 52:23,25 53:8 53:12,13,17 54:3 55:17 57:13,15,16 77:5 145:24 146:10 194:6 213:17 214:25 218:4 222:7 230:18
standardized 108:17 121:3 121:16
standards 54:17,22
standpoint 242:24
start 228:21
started 13:15 47:2
starting 197:13 220:7 233:9
starts 121:13
state 2:8 8:19 9:15 10:5,11 151:25 248:3 249:3,4,5
stated 80:15 109:21 111:21 154:3 156:13 161:12 163:14 231:10 247:15
statement 88:6 111:2,4 153:1 172:3 191:5,8 191:13 199:17
statements 238:15 239:10
states 1:1 3:18 5:17 8:14,15 9:5,7,10,11 10:18,19 53:2 53:20 240:5,10
static 77:17
station 4:10
stationed 36:12 36:19 116:5,21 153:20
statistic 70:21
statistical 85:13 86:15 107:21 120:10 122:2 139:20 140:15 163:15
statistically 70:8,12 83:3 83:19 107:11 120:5 121:5,22 140:24 141:15 147:9 187:5,23 188:7,11
statistician 139:5
status 135:2 144:15 229:20 229:22 230:13 235:22
statute 50:2
statutory 52:10 53:24
stay 32:17
stenographic 1:24 2:7 9:18
step 212:21
steps 229:24
stimulate 167:20
stimulation 238:4
stock 4:14 8:7
store 170:13 243:4
strains 7:17 173:19 174:7 180:3
street 3:3,11,19
strength 183:11 229:6
strengths 33:18 88:19 144:24
stretch 173:5
strike 85:18 88:6 150:4
stroke 214:10 214:17,24 220:10
strokes 214:7 214:15
strong 98:22 201:18
strongly 209:24
structural 169:24 170:3 170:17 171:4 180:10 214:25

220:9
structurally 82:3 171:15
structure 170:18 171:11
struggling 102:18
studded 74:2
studied 65:24 71:1 72:8 74:1 79:20 80:18 88:21 104:23 149:21 152:14 157:20 207:1
studies 31:12 32:3 33:2,9,21 33:25 34:4 48:9,14,17 57:17 62:9 64:6,10,14,16 64:19,24 65:7 65:15 67:25 68:2,17,22,23 69:8,11,12,12 69:13,23 70:7 70:15 71:6,8 71:12 72:2 73:24 80:19,20 81:7,7,21 85:13 86:8 87:18 89:17,25 90:7,8,15,16,17 90:20 91:8,17 91:19,24,25 92:10,14 96:18 96:25 98:10 104:14,20,20 108:3 112:15 112:22,24 113:11,25 114:4,4 120:24 122:7,23 124:7 131:12 132:11 132:12 151:5 153:24 155:19 155:19,21 157:21 158:5 167:10 168:10 168:12 171:10 184:15,17 187:9,14,17,24 188:3,7,12 193:7 194:13 194:17,20 195:6,10,15 212:1 213:22
study 6:14,19 6:20,23 30:25 33:6,19 34:16 48:15 67:17 68:9 69:3,23 79:3 80:1,7,8 81:2 83:2,19 84:4,9,12 85:1 85:21 86:16,19 87:24 88:19 90:23,23 91:4 91:4,7,11 92:11,15,24,25 93:4 97:3,12 100:10 113:7 113:10,20 115:12,16,21 115:23 117:20 117:25 118:14 118:16,19,23 119:3 120:21 120:24,25 122:13,15,15 123:14,14,17 123:20 124:3,8 124:8,24 125:24 126:2,9 127:2,11,12,20 129:3,5,14,17 129:18 131:2,5 131:7 132:21 132:24 133:2,5 136:12 138:8 138:22 139:19 140:1,21 142:21 143:1 143:12 145:13 145:22 147:2 147:24 150:10 150:16,25 151:4,7,13,24 153:4,19 155:3 155:5,23 156:8 157:6,12,16,17 157:23 158:6 158:10 159:2,6 159:8,11,14 161:17 162:1,5 162:9 167:3,5 167:18 168:21 173:16 175:10 177:19 178:17 178:25 179:5 179:11,13,17 181:23 199:22 210:10 213:12 236:13
study's 84:18 85:7 88:15
sub 229:9
subgroups 209:7
subject 11:6 29:24 46:9,13
subjects 78:19 118:14 133:2 133:14 134:18 134:24 143:14 143:15,24 144:1 153:7
subpoena 5:10 14:12
subpopulations 122:24
subscribed 248:2
subsequent 207:18 245:4
substance 16:1 83:4,20,21

substances 13:18 151:9
substantia 82:6 82:8 115:6 173:11 177:15 214:18
substantial 52:16 190:5,11 190:14,17,20 199:12,18
substantially 175:15 176:3 178:19
suffer 246:6,9
suffering 245:22
sufficient 55:2 55:4,16 59:2 59:25 60:14 61:2 66:8,18 66:24 76:1,14 102:20 112:21 145:24 150:5
suggest 87:8 176:2 177:19 180:11 206:11
suggesting 102:4 172:4
suggests 169:24
suitable 134:22
suite 3:7 10:13
summarize 184:16
summarizing 110:18 111:8
summary 175:10
sunnyvale 16:10
super 117:4
supplemental 93:22
supply 156:16 212:7
support 18:22 47:19 110:3 160:18 171:15 184:18 190:3
supporting 24:4 98:23
supports 170:4 170:7
suppose 103:22
supposed 195:12
sure 10:25 11:18 13:17 14:13 18:11,24 21:16 24:22 33:5 34:1 57:15 63:11 72:1 93:3 97:22 100:12 101:17 103:11 105:23 109:18 118:22 125:4 136:23 139:13 147:13 148:6 149:6 158:14 164:9,15 165:20 185:10 192:20 193:24 194:3,19,25 195:8 201:6 215:12 226:3 228:13,14,17 242:13
surprisingly 230:12
sustained 154:13
swear 9:15
sworn 2:11 9:23 249:6
symptom 234:21 246:17
symptoms 161:4 213:3 221:11 232:15 233:25 234:12 234:16 242:17
syndromes 220:17
synergistic 81:9,13 82:13
synergy 81:23
syngenta 45:20
synthesis 89:23
system 128:17 129:1 130:1
systematic 89:15 94:22,24
systems 128:12 128:19,23 228:24 230:8

t

t 167:22
table 119:23 121:12,18 142:6,13 145:7 147:6,8,14 159:16
tables 108:11 189:20
taclo 82:5 115:2 167:22 177:14
tailbone 243:5 243:9
take 11:6 12:13 12:14 19:10 26:18 37:3 38:6 40:14,20 46:17 88:18 100:13 123:3 131:15 140:17 164:7 173:6,23 180:16 215:19 230:1,5 232:25 241:14
taken 35:13 46:20 89:8 131:19 180:20

215:23 241:20
takes 100:10
talk 16:23 19:1 48:24 106:23 136:2 149:7 165:7 211:8 217:17 219:20
talked 74:4 200:12 212:3 245:11
talking 59:5 79:4 119:20 131:4 145:25 149:8 173:10 177:12,14 197:7 204:1 216:9
talks 83:12 177:9
tarawa 128:15 128:24 129:4,8 129:12,21 130:7,10,19 131:7 193:14 193:17,22
target 181:5
tasks 144:21 236:6
taught 18:14 31:5 34:21 35:10 37:3
tautologies 204:1
tce 13:20 23:6 35:18 42:3 57:25 58:7,9 58:13,19,25 59:24 60:5,13 61:1 62:5,8 64:8,12,21 66:8,12,18 67:6,16,20 68:1,3,19 71:13,18 72:20 73:4,17,24 74:11,18,25 75:7 77:12,17 78:12,17,20 79:23 80:1,4 80:12 81:9,16 81:17 82:1,3 95:7 96:3,19 97:18 98:11,16 98:24 99:1,20 114:21 115:3 117:6 120:13 130:2,6,9,19 131:1,9 136:14 136:23 137:6 138:23 139:21 140:1 141:12 142:3,14,15 145:8,10,23 146:6,9 155:22 156:4,6,7,9,11 156:14 157:1 162:18 163:6,7 163:10,20 165:1,10 167:20,25 169:4,10,20,23 170:4,5,11,12 170:14 171:21 172:4,18 173:8 175:12,19,23 176:4 177:5,8 178:20 179:19 180:10 184:16 187:6 189:11 190:23 191:23 197:7,8 198:4 198:14 222:9 223:24 224:17 224:24 246:25
tcp 39:4
team 19:20
telan 3:4 5:5 8:21,21 14:19 17:12 19:10 20:24 21:9 24:13,20 28:13 29:25 30:18 36:15 38:19 39:22 41:6 42:23 47:24 49:3,25 51:10 53:14 54:4,18 54:24 55:6 56:2,13,18,23 58:11,21 59:3 59:11,15,20 60:2,8,18 61:9 61:16,21,25 62:7 63:9,18 63:22 65:3,13 66:10,19 67:1 67:7,19 68:12 70:9 71:15 72:6 73:5 76:4 76:25 77:8,24 78:15 80:14 81:11,19 83:5 83:23 84:6,14 84:20 85:4,23 86:9 87:6,13 88:5,16 89:1 89:18 90:25 91:20 92:1,12 92:20 93:1 94:4 95:3 96:20 97:15,24 98:2 100:6,11 101:1,5,12,21 102:2,24 103:7 103:18 104:2 104:18 105:11 105:22 106:4 106:11 108:2 109:11 113:8 114:2 116:3 117:16 118:3 120:7,17 121:24 125:3 126:23 127:14 129:16 130:16

130:23 133:24 136:17 137:13 137:18,23 138:5,15 139:3 140:8 141:1,14 142:6,8 143:16 144:10 146:1 146:16,25 153:5,14 155:6 155:14 160:7 160:16 161:11 163:25 164:2 170:6,21 171:17 174:1,8 174:19 175:1 176:5,9,12,18 177:22 178:12 178:21 179:1 179:22 180:12 182:20 185:9 186:4,7,11,14 187:7,20 190:24 191:11 191:14,19 195:13 196:9 196:21 197:7 200:19 201:1 201:10,17 202:1,11,17,21 204:5 205:7,14 205:25 206:19 207:16 211:17 213:10 216:23 218:10,17 219:7 220:4,24 221:24 223:6 223:25 224:8 225:19 226:8 237:2 241:10 241:14,17 242:1,2,12,18 243:18 244:2,8 244:22 245:5 245:20 246:4 246:18 247:3 247:14

telephone 3:5,9 3:13,16,21 4:7 4:12 17:16,18 135:1

tell 11:3 12:4 21:2 52:24 65:7 181:10

tells 13:11

temporality 183:12

ten 81:25 142:2 243:3

tend 154:22

tendency 197:11,12

term 20:14 41:18,22,24 48:18,22 81:13 113:3,16 136:18 141:16 170:22,25 212:18 213:2 217:10,19 226:10 246:23

terminal 16:8

terminology 215:8 235:10

terms 81:9 95:8 96:10,13 195:11 245:10 246:5

terrace 128:15 128:24 129:4,8 129:12,21 130:7,10,19 131:7 193:14 193:17,22

tertials 116:8 116:23

test 228:8 229:22

testified 9:24 106:1 239:23

testify 13:3,6 29:23 240:19

testimony 12:20,25 25:12 47:1 114:13 132:6 153:12 181:1 216:6 247:21 249:7

testing 106:15 107:1,12,17 222:19,25 223:2

tests 140:15 228:18

tetrachloride 136:15 137:7

tetrachloroet... 7:16 13:20 96:5 173:18 174:5,6

texas 1:24 2:8 9:20 249:5,25

th 163:1,1,3,4 163:11,17 164:7,16,24 165:4

th's 164:13

thank 9:13 10:15 12:2 47:3 81:4 82:21 93:10 109:20 113:22 114:11 132:7 137:4 176:20 181:3 189:10 241:6 247:17 247:22

thanks 247:3

thereof 2:4

thing 14:15 71:3 82:24 224:16 229:21

things 32:19,22 33:13,15 39:1 75:11 96:13 135:21 207:8

227:23 228:1
think 21:10,10 21:11 28:13 32:3 36:24 38:14 42:18 43:11 46:16 49:17 51:23 52:2 56:19 57:23,24 71:5 73:7 78:21 82:2,14 87:23 89:3 113:20 119:16 120:10 125:7 129:6,22 136:5,8 145:19 148:13 150:5 155:9 158:1 159:17 163:25 168:24 177:23 177:24,25 179:25 182:2 183:14 201:19 206:9 208:14 209:13 212:1 212:22 217:15 221:6 224:16 230:9 232:17 236:13 240:8 240:20 244:9
thinking 31:19 209:24
third 98:8 119:2 127:15 159:17
thorough 38:6 227:12 229:2 230:5
thought 77:6 142:9 208:8 211:2,4
thoughtful 38:5
thousand 158:2
thousands 39:19 40:11,15 41:3 162:25
thousandths 141:5
three 18:20 28:7 43:12 45:13,14,15,17 52:19 72:9,14 110:15 111:9 141:4 142:10 162:16 163:6 163:18 180:3 182:13 200:4
threshold 63:15 66:5 70:20 190:22 191:1
thrive 244:24
time 8:1,10 11:9 12:14,20 20:11 26:16,18 37:23,23 40:17 40:18,19,22 45:8 77:17,22 78:25 89:4 100:13 102:22 103:4,6 120:20 128:2 146:7,17 148:23,25 158:11,16,17 159:9,15 167:16 170:14 174:24 177:25 178:9 182:24 191:24 192:3,7 192:18 199:2 202:14 215:2 216:10 228:14 232:7 236:18 241:12,15 243:24 244:18 247:3,17,22
timeline 20:4
times 22:7 27:9 27:11 45:7 195:11 196:1
tissue 170:14 175:21
tissues 7:16 173:19 174:6
title 174:1 189:17
titled 115:12 161:18
titrated 103:21
titrating 62:12
today 8:16 12:21 13:7 16:19 19:24 21:12 24:22 52:24 57:24 62:3 77:2 93:25 96:9 128:21 168:25 182:6 187:8 188:3 189:4 191:1 195:15 205:17 206:16 211:10 212:3 214:2 218:23 219:12,17 222:9 237:3 241:5 246:20 247:21
today's 8:9 14:7 17:7 247:20
together 11:9 15:20 16:7 32:14 60:13 61:2 73:19,22 74:1 81:16 97:21 156:9
told 55:19 181:21
tone 228:6 229:13
took 11:3 216:3
tools 70:23
top 29:3 121:13 187:11
topic 149:5 210:10,14

216:8
topics 32:18 49:10
tort 53:21,25
torts 4:4,4,9
total 45:14 102:21 119:24 124:4 163:2 165:24 166:22 194:5,8,21,24 195:7,18,21 196:6,24 198:3 198:10
totality 82:12 84:9 106:17 114:7 155:18 195:25
touched 230:9
touching 82:20
toward 144:16 154:23
towards 135:14 140:6,23 141:3 144:9,25 227:14
toxic 13:18 20:18,23 23:5 43:7,18 44:9 63:1 66:12 90:12,14,21 117:15 151:8 160:20 172:21 172:24,25,25 177:4 190:5
toxicities 175:24 180:11
toxicity 171:5 176:4 177:21 178:19 179:19 179:20,21 180:1,6 181:6 184:16
toxicologic 182:25
toxicological 34:16 169:9,11 169:13 171:16 172:20
toxicologies 169:25 172:4
toxicologist 34:7,13 171:9
toxicologists 105:3
toxicology 34:10,19,21 61:14 194:10
toxins 90:11 210:18
trail 3:7
trained 18:14 235:17
training 29:17 31:8 34:12 38:8 41:16 171:3
transcribe 11:17
transcribed 11:19 249:8
transcript 249:17
transcription 249:9
transform 169:23
transitioning 19:16
trauma 206:16 220:16
traumatic 149:3,11
travel 26:1
treat 219:11
treated 36:8 233:18
treating 18:2
treatment 128:5,17 129:1 162:14 163:20 193:2 197:1 209:9 227:13 233:14 237:8 237:13,16 239:21
treatments 219:13
tremor 229:13
trending 140:5 140:23 141:3
trial 16:15 28:3 129:11 239:23
trials 89:23
tribunal 46:14
trichloroacetic 177:12
trichloroetha... 177:13
trichloroethyl... 7:12,15 13:19 96:4 161:18 173:18 181:7
triple 72:12
tripling 88:12
true 39:7,9,10 53:11 128:6 172:17 191:5,8 191:13,17 206:12 208:8 227:15 228:3 248:2 249:10
truth 11:3
truthful 13:6
try 138:7 209:8
trying 51:8 167:12
tuesday 1:14 2:3
tumor 220:10
turn 15:2 24:8 97:23 104:21 107:2 109:19 121:2 175:7 241:12
turner 4:6 9:10

turning 162:24 169:17
twice 41:15
twin 79:25 80:7 80:8 91:8 136:7 147:2,10 147:10,19,22 148:18,19
twins 7:4 132:22,25 135:17,23 147:23,24 148:2,3,8,9,23 148:24
two 17:13 18:20 39:24 40:6,12 43:23 44:8 45:9 54:22 59:9 71:8 75:11 88:9 98:10 108:23 109:22 119:25 122:7 173:6,13 182:8 207:8 213:24 223:10,16 224:24 227:5 240:1
type 57:1 109:6
typed 233:2
types 38:25 57:22 62:9 64:10 150:6 206:15 213:15 220:15
typical 161:9 216:24 217:8 228:10 230:11 230:17 235:4 243:15
typically 38:14 84:17 110:6 209:18 220:6,9 227:1 230:22 230:25 231:7 243:13
typo 113:20
tyrosine 164:13 164:18

u

u 18:18 46:8 236:3
u.s. 4:3,10 121:6 136:24 151:8
uh 11:24
ultimately 221:9,13
umbrella 213:2
unable 154:10
unaffected 147:10,22 148:3,9,18,24
unavoidable 143:5
unaware 144:15
unbinding 249:16
uncertain 227:11
uncertainty 108:14,19
under 14:18 45:20 53:21 63:20 98:7 112:18 148:21 158:21 159:18 162:13 240:8 248:1
underestimati... 144:25
undergone 222:18
underlying 121:17 138:3 138:14
underreport 144:5
underrepresent 144:5
understand 11:2,5 12:3,22 36:25 49:17 51:8 71:5 82:2 119:11 176:11 183:21 184:5 185:10 192:23 193:16 195:1 207:10 217:21
understanding 31:17 55:14,24 75:5 81:15 102:16 111:2
understood 12:6 21:9 48:7 59:15 164:14 192:20 198:2
undoubtedly 128:7
unethical 62:12
unexposed 154:21 159:20
unfair 176:14
unified 229:8
unique 89:24 217:8
united 1:1 3:18 5:17 8:14,15 9:5,6,10,10 10:17,19 53:2 53:20 240:5,10
units 194:16 195:17
universal 209:9
university 18:8
unknown 216:22 218:8 221:20,23 222:2 226:1,6
unquantified 68:2
unquote 91:7

unsealing 249:16
unusual 161:13 234:20
updated 182:5
upper 107:25 108:20 213:7
uptick 234:21
urinary 245:16 245:21
usdoj.gov 3:21 3:22 4:7,8,12
use 38:23,25 47:11 48:4 50:16 51:2,8 51:24 95:22 106:14 107:9 107:12 109:9 110:6,22 113:25 141:2 148:15 153:8 167:10 170:23 183:16,18 194:4,21 195:6 196:2,6 208:20 212:18 213:17 215:3 217:20 226:10 235:20 237:15 246:23
used 33:9 47:8 47:20 48:8 49:19 50:12,17 53:7 57:4 79:6 81:13 96:11 108:4 109:1 110:5,9 111:10 112:6 135:8 136:15 137:7 141:6 149:22 151:13,16 152:1,3,18 166:7 183:4,7 185:18,19 186:20 194:14 195:7,21 196:13,18 200:16 206:9 207:6 224:5
useful 92:5 114:5 122:24
usefulness 113:17
uses 19:4 48:16 62:22 201:7
using 41:21 48:18 87:19 95:17 111:16 113:23 125:11 125:12 144:7 145:22 198:19 215:8
usmc 6:14,18 6:22 115:15
usually 36:11 36:16 217:22 236:10 239:5,6
utility 112:16 112:25 113:4,6 113:10,12,16 113:20,23 114:4

v

v 5:17
va 47:22 48:3,3 50:12 78:20 79:6 106:8 151:21
va's 47:16 106:2
validate 179:4
valuable 71:2
value 70:12,19 70:24 112:3,4 140:11,14 141:4 148:14 187:9
values 140:10 140:15 142:17
variability 178:3
variables 104:22 205:9
variation 209:7
varies 26:17 100:12
variety 179:14
vary 57:2 173:6 204:10
vascular 220:10,12
vast 228:8
vehicle 162:18 162:20 163:5 163:11,21
verbally 11:21
verifiable 214:22
verification 192:14
verify 33:8 179:4
veritext 8:8
versed 30:3,20
versus 8:14 34:5 41:4 42:19 44:3 45:20 53:2,12 53:20 85:22 140:12 147:21 148:18 158:18 240:4,5,10,16 240:17,19
veteran's 152:1
veterans 36:11 47:16 79:7 80:1 105:8,21 133:3,6 151:17 152:19
vha 157:25
video 8:11 227:24
videoconfere... 3:7,11,15 4:3,9

videographer 4:14 8:6,8 9:13 46:18,22 82:19 89:6,10 131:17 132:1 176:23 180:18,22 215:18,21,25 241:16,18,22 247:6,18
videotaped 1:12 2:1 5:8
vietnam 37:12 37:15
view 10:14 49:9
viewpoints 182:21 183:9
vinyl 36:1 39:9 39:10 80:13 156:18 187:18 187:25 188:1 191:13 192:10
virtually 143:4 227:2 232:3
vitae 5:20
voc 156:15
vocs 156:1,9 158:22 188:14 191:18
void 249:18
volume 162:20 194:25 195:7

w

w 3:11,20
wait 167:16
waived 249:21
walk 228:10
walked 230:22
walking 242:16 242:16 243:4
want 21:3,11 59:4 84:18 102:11 145:18 147:13 149:16 163:23 164:15 165:4 175:2 179:1 189:7 210:4
wanted 21:12 106:10 114:9 114:14 132:6 144:4
war 133:2
wash 18:18 236:3
washington 3:19 4:5,11 18:7
water 1:6 6:8 6:14,18,22 7:10 10:19 17:24 19:17 20:10 36:9 58:7 59:1,25 63:7,11 64:3 64:21,22 65:18 74:6,14,17,19 74:22,24 75:10 75:14,22,25 76:12 77:12 78:18 90:1 104:16 115:15 118:9 128:11 128:17,19,22 129:1 130:1 155:25 156:15 156:17 157:2,3 157:14 172:16 172:20,21,24 172:24 193:2 197:16,17 199:6 200:16 201:13,23,23 212:5,7 221:15 221:17 222:5 223:23 224:7 224:13 225:5,8 225:18 246:22 246:23
way 15:21 37:3 55:9 97:25 118:10 120:24 153:18 172:18 200:13 206:25 207:1 249:13
ways 196:3 215:14 235:15 236:1
we've 14:20,23 52:20 81:6 89:4 99:8 106:20 122:20 141:21 147:4 158:5 163:3 188:2,17 190:25 200:12 206:15 209:16 211:14 212:3 218:23
weakness 119:3 143:12,22
weaknesses 144:24
week 17:13,18 68:4 162:21 165:2 166:19 195:16
weight 84:22 92:11,14,25 244:24 245:1
welch 231:21
went 23:9 28:3 28:5 43:24
whereof 249:19
wide 138:20 139:1 141:23 160:24 161:14
wider 137:21 141:12,15
wilson 220:14
window 147:14

wish 12:14 114:12 241:5
withdrawing 178:12
witness 2:9,12 9:16 22:1 24:14,21 26:13 27:3,10 30:1 30:19 33:22,23 34:5 36:16 38:20 39:23 41:7 42:24 47:25 49:5 50:1 51:11 53:15 54:19 56:5,14,19,24 58:12,22 60:3 60:10,19 61:10 61:22 62:2,8 63:10,24 65:5 65:14 66:11,21 67:2,8,20 68:13 70:10 71:17 72:7 73:7 76:17 77:1,9,25 78:16 80:15 81:12,21 83:11 83:25 84:8,15 84:21 85:5,24 86:10 87:7,15 88:8,17 89:2 89:20 91:2,22 92:3,13,21 93:3,10 95:5 96:21 98:3 100:7,12 101:14,22 102:3,25 103:9 103:20 104:4 104:19 105:12 105:23 106:5 106:12 108:3 109:12 113:9 114:3 116:4 117:18 118:5 120:9,19 122:1 125:4 126:24 129:17 130:24 133:25 136:18 137:14,19,24 138:6,16 139:4 140:9 141:2,15 144:11 146:2 146:17 147:1 153:6,15 155:7 155:15 160:8 160:17 161:12 164:4 170:7,22 171:18 174:3 176:20 178:2 179:25 180:13 182:21 185:10 186:15 187:8 190:25 191:15 191:20 195:14 196:10,22 197:9 200:21 201:11,18 202:2,12,18,22 203:16 204:6 205:8,16 206:1 206:20 207:17 211:18 213:11 216:24 218:11 218:18 220:5 220:25 221:25 223:8 224:1,9 225:21 226:9 239:17 242:10 243:12 244:1 244:23 246:12 248:6 249:19
women 206:8
wonderful 217:7
word 14:14 33:4 89:21 148:15 173:23 190:19 201:18
wording 51:3
words 90:18 214:11
work 18:17 22:24 25:3,7 26:15,16,24 27:25 34:5 40:13 47:9 108:3 109:2 110:10,23 112:7 128:8 183:5 192:15 194:1 224:4
workday 128:3
worked 129:12 145:5 193:11 193:16,23 200:3
workers 6:13 115:14 128:1,7 197:12
working 39:12 39:21 129:4 197:18 199:2
workplace 127:24 128:23
works 94:13 169:6
world 133:2
worldwide 136:16 137:8
worse 244:18
writing 24:2 94:19 181:24 189:20
written 23:14 27:13,14 47:18 48:1 79:1 230:21
wrote 53:1 110:13 169:18 181:20 190:1 198:22 234:1

x

x 5:22 249:22

y

y 162:25 163:2
yahr 229:15
yeah 136:10 139:25 142:12 186:13
year 26:17,17 26:24 68:5 181:22 234:13
year's 16:6
years 18:19,20 21:22 27:12 40:3 63:21 64:3,4 65:16 75:9,13,17,17 75:22 99:7 159:19 161:1 182:24 195:16 200:17 234:14 234:17 236:14
yesterday 17:13,17
yielded 143:2
young 159:23 160:3 161:14

z

zina 3:15
zina.bash 3:16
zoom 3:7,11,15 4:3,9 8:24

(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.

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VERITEXT LEGAL SOLUTIONS

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Veritext Legal Solutions represents that the foregoing transcript is a true, correct and complete transcript of the colloquies, questions and answers as submitted by the court reporter. Veritext Legal Solutions further represents that the attached exhibits, if any, are true, correct and complete documents as submitted by the court reporter and/or attorneys in relation to this deposition and that the documents were processed in accordance with our litigation support and production standards.

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