

# Exhibit 598

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

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

This Document Relates to:  
ALL CASES  
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- - -  
June 30, 2025  
- - -

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

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GOLKOW, a Veritext Division  
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A P P E A R A N C E S:

ON BEHALF OF PLAINTIFF:

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A P P E A R A N C E S : (Cont.)

ON BEHALF OF DEFENDANT:

UNITED STATES DEPARTMENT OF JUSTICE

CIVIL DIVISION TORTS BRANCH

ENVIRONMENTAL TORTS LITIGATION SECTION

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VIDEOGRAPHER:

Jonathan Juarez

TRANSCRIPT INDEX

PAGE

APPEARANCES.....	2 - 3
INDEX OF EXHIBITS.....	5 - 10
EXAMINATION OF	
MATTHEW J. WEISS, M.D., MBA:	
BY: MR. BU.....	13
BY: MS. SULPIZIO.....	298
AFTERNOON SESSION.....	201
SIGNATURE PAGE.....	301
ERRATA.....	302
REPORTER'S CERTIFICATE.....	303

EXHIBITS WITH ORIGINAL TRANSCRIPT

FEDERAL STIPULATIONS

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

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

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

## E X H I B I T S

NO.	DESCRIPTION	PAGE
Weiss Exhibit 1	Specific Causation Expert Report for David Fancher	22
Weiss Exhibit 2	Plaintiffs' Designation and Disclosure of Phase III Expert Witness With Respect to Kidney Cancer. Materials Considered List For Matthew J. Weiss' Report on Plaintiff David W. Fancher	24
Weiss Exhibit 3	Dr. Matthew Weiss - Supplemental Materials Considered List	25
Weiss Exhibit 4	Dr. Matthew Weiss - Second Supplemental Materials Considered List	25

- - -  
E X H I B I T S  
- - -

NO.	DESCRIPTION	PAGE
Weiss Exhibit 5	National Academies Sciences Engineering Medicine Article Review of the Department of Veterans Affairs Presumption Decision Process	52
Weiss Exhibit 6	National Cancer Institute article Cancer Stat Facts: Kidney and Renal Pelvis Cancer	107
Weiss Exhibit 7	ATSDR Assessment of the Evidence For the Drinking Water Contaminants At Camp Lejeune and Specific Cancers and Other Diseases January 13, 2017	130



## E X H I B I T S

NO.	DESCRIPTION	PAGE
Weiss Exhibit 8	Aschengrau study	142
Weiss Exhibit 9	Moore study 2010	148
Weiss Exhibit 10	Andrew study 2022	157
Weiss Exhibit 11	Woburn Center Incidence and Environmental Hazards 1969-1978 January 23, 1981	164
Weiss Exhibit 12	Bove study 2014	166
Weiss Exhibit 13	Additional File 2: Table 11: Categorical Cumulative Exposures and Underlying Cause of Death	167

## E X H I B I T S

NO.	DESCRIPTION	PAGE
Weiss Exhibit 14	Bove study 2014	178
Weiss Exhibit 15	ATSDR study April, 2018	183
Weiss Exhibit 16	Bove study October 2024	188
Weiss Exhibit 17	Appendix 7 David William Fancher (Kidney Cancer)	225
Weiss Exhibit 18	ATSDR Toxicological Profile For Trichloroethylene June 2019	246
Weiss Exhibit 19	U.S. Department of Health and Human Services Toxicological Profile For Benzene August 2007	253

- - -  
E X H I B I T S  
- - -

NO.	DESCRIPTION	PAGE
Weiss	Invoices and fee schedule	282
Exhibit 20	of Matthew J. Weiss, MD, MBA	
	Bates	
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(REPORTER'S NOTE: All quotations from exhibits are reflected in the manner in which they were read into the record and do not necessarily denote an exact quote from the document.)

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DEPOSITION SUPPORT INDEX

DIRECTION TO WITNESS NOT TO ANSWER

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REQUEST FOR PRODUCTION OF DOCUMENTS

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STIPULATIONS

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QUESTIONS MARKED

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9:26 a.m. EDT

New Hyde Park, New York

- - -

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

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

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

The deponent is Dr. Matthew Weiss.

Counsel, please identify yourselves for the record.

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

MR. BU: Nathan Bu for the United States.

MS. SPRAYREGEN: Sharon

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Sprayregen also for the United States.

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

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

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

THE WITNESS: I do.

THE STENOGRAPHER: Thank you.

- - -

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

EXAMINATION BY

MR. BU:

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

A. Sure. Matthew John Weiss.

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Q. And can you spell your last name?

A. W-E-I-S-S.

Q. You're a physician here at Northwell Health, right?

A. I am, correct.

Q. And our address -- or, Northwell's address is 1111 New Hyde Park, New York -- or, Marcus Avenue, Hyde Park, New York.

A. So, that's where we're located right now.

Q. Okay.

A. Obviously there's a lot of sites at Northwell Health.

Q. What's the address for the primary site where you practice?

A. 1111 Marcus Avenue.

Q. Okay.

My name is Nathan Bu. I'm a trial attorney with the Department of Justice. I represent the United States in this lawsuit.

The purpose of our time together

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

You understand that?

A. Yes.

Q. All right.

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

Do you understand that?

A. I do.

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

A. No.

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

A. No.

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

A. I have.

Q. About how many times have you been deposed?



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

Q. Were all of those as expert  
witnesses?

A. They were.

Q. Okay.

Have you ever been deposed as a  
fact witness?

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

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

A. No.

Q. Okay.

You understand that your  
deposition today is being recorded?

A. I do.

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

You understand that?

A. Yes.

Q. And you're doing a very good job

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so far.

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

A. Okay.

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

A. Sounds good.

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

A. Okay.

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

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

Q. Okay. Perfect.

And if you need to correct an

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answer, you will do so, right?

A. Sure.

Q. Okay.

Do you understand that you'll have the opportunity to review the transcript and correct any responses?

A. Yes.

Q. And do you understand that if you correct responses, the United States may reopen this deposition or question you at trial about those corrections?

A. Sure.

Q. Okay.

And you understand that your answers today are being given under oath under penalty of perjury?

A. Yes.

Q. You understand that your testimony today has the same force and effect as if you were testifying in a courtroom with a judge and a jury present?

A. Yes.

Q. All right.

Would you agree that physicians

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

A. Yes.

Q. Okay.

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

A. Yes.

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

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

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

Q. Sure.

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

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experience and knowledge?

A. Yes.

Q. Okay.

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

MS. SULPIZIO: Object to the form.

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

Q. Sure.

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

MS. SULPIZIO: Object to the form.

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

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Q. Okay. If a theory --

A. Yeah.

Q. If the physician's testimony is based on a theory that's not widely accepted, should the physician acknowledge that the theory is not widely accepted when giving that testimony?

MS. SULPIZIO: Object to the form.

A. I mean, I -- I think that makes sense. I think then they should probably give both sides of the -- of the theory or, you know, what is believed to be true versus what is -- what is fact versus what is theory.

Q. Okay.

And do you agree to hold yourselves to -- yourself to those standards as best you can when giving your testimony today?

A. Yes.

MR. BU: Okay.

So I'm going to introduce our first exhibit. This will be tab --

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or, Exhibit 1, tab 1.

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

BY MR. BU:

Q. Do you recognize this document?

A. I do.

Q. Okay.

Can you tell me what it is?

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

Q. Okay.

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

A. Correct.

Q. Okay.

And the curriculum vitae was  
also submitted earlier this year?

A. Yes.

Q. Okay.

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

A. Sure.

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

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

Q. Okay.

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

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

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

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



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Q. Okay.

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

BY MR. BU:

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

A. Yes, I believe so.

Q. Okay.

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

A. No.

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

BY MR. BU:

Q. Okay. All right.

I'm handing you what's been

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marked Exhibit Number 2.

A. Okay.

Q. Do you recognize this document?

A. Yes, I do.

Q. What is it?

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

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

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

Q. Okay.

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

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

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BY MR. BU:

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

A. Okay.

Q. Do you recognize these documents?

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

Q. Okay.

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

A. Yes.

Q. And --

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

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

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

Q. Okay.

Do you remember reviewing other witnesses' deposition testimony?

A. Yes.

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

A. Yes.

Q. Okay.

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

A. Yes.

Q. Okay.

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

You can take some time to look over them.

A. I don't think so. Although

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

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

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

Q. Okay. That's fine.

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

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

Q. Okay.

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

A. Okay.

Q. All right.

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

A. Yes.

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

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

Q. Okay.

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

A. There are.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Is your opinion based on the

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

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

Q. Sure.

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

MS. SULPIZIO: Object to the  
form.

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

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

Q. Okay.

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

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

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

10 Q. Okay.

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

17 MS. SULPIZIO: Object to the  
18 form.

19 A. No.

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

25 MS. SULPIZIO: Object to the



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form.

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

Q. Okay.

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

A. No.

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

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

Q. All right.

Did you consider applying any other standard of causation?

MS. SULPIZIO: Object to the form.

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

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

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

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

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

A. I did not.

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

A. I did not.

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

MS. SULPIZIO: Object to the form.

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

Q. Sure.

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

MS. SULPIZIO: Note my  
objection.

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

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

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

MS. SULPIZIO: Object to the

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form.

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

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

Q. Okay.

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

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

1  
2 would say the only way that you can -- you  
3 know, with a hundred percent certainty,  
4 you know, show direct causal -- show a,  
5 like, inference, if we have an  
6 intervention and we want to show is there  
7 a benefit to this intervention versus not,  
8 the only way you can definitively show a  
9 benefit is through a prospective  
10 randomized trial. But the reality is is  
11 that in medicine, we frequently can't  
12 perform prospective randomized trials in  
13 order to see if there's an exact  
14 correlation.

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

24 Q. So let me put it this way.

25 Would you agree that prospective

1  
2 randomized trial is better at controlling  
3 for bias in general than retrospective  
4 studies?

5 A. I agree.

6 MS. SULPIZIO: Object to the  
7 form.

8 BY MR. BU:

9 Q. Okay.

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

15 MS. SULPIZIO: Object to the  
16 form.

17 A. I would agree with that.

18 Q. Okay.

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

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

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Q. Okay.

What about random error?

A. What about random error?

Q. Would a prospective randomized trial be better at controlling -- or accounting for random error than a retrospective study?

A. It would as long as the prospective randomized trial is -- is powered to show -- is powered properly to show a significant difference.

Q. Would you agree that determining whether an association is causal includes evaluating the quality of the studies reporting an association?

A. I would agree with that.

Q. Okay.

And part of that evaluation should look at whether the study is able to eliminate the role of bias with reasonable confidence?

A. I would agree with that.

Q. Okay.

Would you also agree that part

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

A. I would agree with that.

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

A. Yeah, I would agree with that.

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

A. Sure.

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

A. Sorry, which page are you on now?

Q. I'm sorry, page 4.

A. It goes on the back, sorry.

Q. Yeah.

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

Q. So at the top of the page for



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

Do you see that?

A. Sorry, point 2 and point 3?

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

A. All right.

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

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

Q. Okay.

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

A. Correct.

Q. Okay.

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

Do you see that?

A. I do.

Q. What does reasonable confidence  
mean to you?

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

Q. Okay.

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

A. I'm not aware.

Q. Okay.

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

A. I would.

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

A. Agree.

Q. All right.

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

A. I do.

Q. Okay.

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

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A. I do.

Q. Do you ever serve as a peer reviewer for medical literature?

A. I do.

Q. How long have you been acting as a peer reviewer for medical literature?

A. Probably 15 years.

Q. When you serve as a peer reviewer for medical literature, do you consider a study's ability to rule out bias?

A. We do.

Q. And when you peer review medical literature, do you also consider the study's ability to rule out chance?

A. I do.

Q. Have you ever peer-reviewed literature that applies an "at least as likely as not" standard?

A. Have I ever peer-reviewed literature? No.

Q. Have you ever published literature that uses an "at least as likely as not" standard?

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

Q. Have you ever published literature?

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

Q. Okay.

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

Q. Okay.

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

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

A. It was part of this litigation.

Q. Okay.

Do you remember the study author?

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

Q. Okay.

But you think they were a defense expert?

A. Yeah. I reviewed so many, you know, documents, you know, over the last -- and when I -- when I started this, I'll be honest, I just can't remember who it was, but I know I took note of it because I just hadn't seen it before.

Q. Other than this one study that was provided to you as part of this litigation, are you aware of any other literature that uses an "at least as likely as not" standard?

A. I am not.

Q. Okay.

Have you ever offered opinions in another case using an "at least as likely as not" standard?

A. I have not.

Q. I apologize because I think I may have asked this already, but other than the ATSDR Assessment of the Evidence,

1  
2 are you aware of any published guidance in  
3 how to apply an "at least as likely as  
4 not" standard?

5 A. I am not.

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

8 A. I did see that.

9 Q. Okay.  
10 Have you seen "equipoise and  
11 above" used in other scientific contexts?

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

1  
2 show that the new treatment is better than  
3 the old treatment, but they want to show  
4 that it's at least as good as the old  
5 treatment, and we call that a  
6 noninferiority trial.

7 Q. Okay.

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

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

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



1  
2 standpoint that you're having an  
3 at-least-as-good-as-desired outcome.

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

7 MS. SULPIZIO: Object to the  
8 form.

9 A. Say it one more time, sorry.

10 Q. Sure.  
11 Is noninferiority looking at  
12 more than just the probability that an  
13 intervention works?

14 MS. SULPIZIO: Object to the  
15 form.

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

23 Q. Okay.

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

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used in any other context?

A. I don't think so.

Q. Would you agree that the term "equipoise" denotes a lack of consensus across the medical community?

MS. SULPIZIO: Object to the form.

A. I don't think I would agree with that. I think equipoise means -- I equate it to they're equivalent. It doesn't mean that we don't either all agree that they're equivalent. So I don't think I -- I agree with that statement.

Q. Okay.

A. There may be consensus that there is equipoise.

Q. Are you familiar with the National Academies of Science?

A. Yes.

Q. What is -- what are the National Academies of Science, or are?

A. I mean, the National Academy of Sciences is essentially a -- a group that, you know, people are accepted to -- into

1  
2 as members based upon their either  
3 academic reputation or advances in their  
4 field of expertise and it's a -- it's a  
5 relatively prestigious, essentially like a  
6 club, like a society.

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

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

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

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

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

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

1  
2 authoritative, meaning it's, you know,  
3 it's the bible.

4 I guess I shouldn't say that.

5 But, I mean, it's -- it's --  
6 it's a well-respected, you know,  
7 publication that should be taken into  
8 account, but like any publication, you  
9 know, has to be interpreted, you know,  
10 with a grain of salt and, you know,  
11 there's -- there's lots of publications  
12 in, you know, very strong journals that  
13 maybe are not as well done as we would --  
14 as -- as we would like in the medical  
15 community and vice versa. So I wouldn't  
16 say that it's authoritative like you have  
17 to follow what's in the National Academy  
18 of Science, but it should be -- it should  
19 certainly be taken into account that it's  
20 a peer-reviewed publication and that, you  
21 know, it's a well-respected journal.

22 MR. BU: Okay.

23 Could we pull tab 30, please?

24 MS. SPRAYREGEN: 30?

25 MR. BU: 30, yes.

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

BY MR. BU:

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

Have you seen this document before?

A. This is a particular document. I'll be honest, I don't -- I don't -- I don't think I recall reviewing this.

Q. Okay.

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

Q. All right. So, earlier we were talking about the term "equipoise" and whether or not it indicates a lack of consensus. Do you recall that?

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A. I do.

Q. Okay.

Are you aware that the National Academies of Science concluded that the term "equipoise" denotes a lack of consensus across the medical community?

MS. SULPIZIO: Object to the form.

If you're going to ask him questions about it, I'd like for him to review it.

MR. BU: I'm just asking if he's aware of that.

A. I am not aware of that.

Q. Okay.

And are you aware that the National Academies of Science concluded that the term "equipoise" is inconsistent with current scientific use?

MS. SULPIZIO: Object to the form.

A. I'm not aware of it.

Q. Okay.

Could you turn to page 10 for

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me, please?

A. Okay.

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

BY MR. BU:

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

A. I do.

Q. Okay.

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

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

Q. Okay.

Do you have any reason to

1  
2 disagree with that conclusion by the  
3 National Academies of Science?

4 MS. SULPIZIO: Object to the  
5 form.

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

12 Q. Okay.

13 And why are you not in  
14 agreement?

15 MS. SULPIZIO: Object to the  
16 form.

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



1  
2 have the same outcome, that -- that --  
3 that doesn't mean that the medical  
4 community wasn't able to come up with a  
5 consensus. So I think you can have  
6 equipoise without -- and have a consensus  
7 that there's equipoise.

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

12 Q. Okay.

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

18 MS. SULPIZIO: Object to the  
19 form.

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

1  
2 equivalent. I mean, one of the groups can  
3 be correct. I would find it hard to  
4 believe that both could be correct.

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

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

20 Q. Okay. You can set that aside.  
21 Can you go back to Exhibit 1,  
22 your report, please?

23 A. Okay.

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

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

A. I do.

Q. Okay.

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

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

Q. Okay.

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

A. Not in my academic publications.

Q. Okay.

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Have you ever used that phrase  
in your clinical practice?

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

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

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

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

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MS. SULPIZIO: Object to the  
form.

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

Q. Okay.

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

MS. SULPIZIO: Object to the  
form.

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

1

2 Q. Okay.

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

10 MS. SULPIZIO: Object to the  
11 form.

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

24 Q. Okay.

25 A. So you could -- you could base

1  
2 lots of things on a reasonable degree of  
3 medical certainty.

4 Q. Okay.

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

8 Do you recall that?

9 A. Yes.

10 Q. Okay.

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

13 A. Correct.

14 Q. Okay.

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

17 A. Correct, for the most part.

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

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

1  
2 Well, cancer is a -- is a tumor that has  
3 the ability to spread, metastasize and  
4 either, you know, harm you permanently  
5 harm you or -- or kill you.

6 The roots of cancer, you know,  
7 in -- in theory, all cancer to some extent  
8 is -- is genetic, but those genetics  
9 are -- you know, we -- we define as all  
10 cancers have -- have genetic mutations in  
11 them. That doesn't mean that genetics  
12 causes the cancer. All cancer cells  
13 because there's a dysregulation of cell  
14 proliferation will frequently have genetic  
15 defects in it. And -- and cancer in -- in  
16 my opinion, is always related to either  
17 environmental exposures, some of which we  
18 know about, some of which we don't know  
19 about, or genetics. We know that certain  
20 patients have a genetic predisposition to  
21 developing cancers.

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



1  
2 patients that have good old-fashioned bad  
3 luck are probably all environmental  
4 exposures or genetics that we just don't  
5 know about yet.

6 Q. Okay.

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

10 A. They are.

11 Q. Okay.

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

16 A. I think obesity is an  
17 environmental exposure.

18 Q. Okay.

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

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

1  
2 risk when patients are obese. I'm not  
3 sure we know mechanistically why that is.  
4 Could it be the obesity itself or could it  
5 be the process by which patients get  
6 obese? I'm not sure we really know, to be  
7 honest with you.

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

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

19 Q. Okay.  
20 Would you agree that for all  
21 cancer, there's either some genetic -- or,  
22 germline or somatic mutation that starts  
23 the cancer process?

24 A. Say that again, I'm sorry.

25 Q. Sure.

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

MS. SULPIZIO: Object to the form.

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

Q. Is the mutation a necessary condition for cancer?

MS. SULPIZIO: Object to the form.

A. Explain that a little bit better to me.

Q. Sure.

A. I'm sorry.

Q. No, it's okay.

Can you have cancer without a mutation?

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

1  
2 you don't think that the mutation is  
3 always the cause of the cancer.

4 MS. SULPIZIO: Object to the  
5 form.

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

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

20 Q. Okay.

21 Does DNA repair play a role in  
22 cancer formation?

23 A. Some. Some cancers, yes.

24 Q. How so?

25 A. I mean, the way our cells work,

1  
2 our cells are dividing all the time at  
3 different rates. When cell division  
4 occurs, sometimes there are mutations in  
5 the DNA as the cell divides and -- and our  
6 body has a -- has an inherent mechanism to  
7 identify genetic defects that occur when  
8 cell division takes place and has a way to  
9 kind of protect ourselves against  
10 developing cancers.

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

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

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

25 So for instance, the most

1  
2 natural process by which cells die is  
3 apoptosis, but if they die in another way,  
4 for instance with a noxious stimuli like  
5 they're -- you know, they're exposed to a  
6 carcinogen, that's a different mode of  
7 cell death.

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

10 MS. SULPIZIO: Object to the  
11 form.

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

15 Q. The causes.

16 MS. SULPIZIO: Objection.

17 A. That the causes of cancer are  
18 multifactorial?

19 MS. SULPIZIO: Object to the  
20 form.

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

1  
2 by a genetic defect. If there's, you  
3 know, a -- a clear, clear exposure to a  
4 carcinogen, I don't -- I don't think that  
5 makes it multifactorial. I think you have  
6 one factor that leads to cancer.

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

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

14 Do you recall that?

15 MS. SULPIZIO: Object to the  
16 form.

17 A. I do recall that.

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

21 MS. SULPIZIO: Object to the  
22 form.

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

1  
2 and I say this to the patients and their  
3 families all the time, is that I'm going  
4 to -- I'm going to provide the best level  
5 of care that I possibly can, that I'm  
6 going to be, you know, committed to them,  
7 and that I'm going to optimize things for  
8 the best potential outcome possible.

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

11 Q. Okay.

12 And why can't you guarantee  
13 those other things?

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



1  
2 published and are at the, you know, I  
3 would argue are as good as anywhere else  
4 in the country or in the world, there's  
5 still a known complication rate.

6           So for instance, if I do an  
7 operation that has a 1.4 percent mortality  
8 rate, you know, there's no guarantees.  
9 1.4 out of a hundred will have a mortality  
10 after that operation no matter what is  
11 done. So I can't guarantee those things.

12           Q.       Okay.

13           Have you published literature  
14 addressing risk factors for postsurgical  
15 complications?

16           A.       I have.

17           Q.       Okay.

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

21           A.       I have.

22           Q.       Okay.

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

1  
2           A.       Some of the -- comorbid  
3 conditions. For instance, obviously  
4 cardiovascular disease a big one is  
5 chronic renal insufficiency or renal  
6 failure. There -- those are -- represent  
7 very high postoperative, you know,  
8 complication rates and even mortality  
9 rates. You know, underlying liver  
10 disease. Basically medical conditions  
11 that make people poor operative  
12 candidates. You know, pulmonary status.  
13 If they're on oxygen at home before  
14 surgery, they frequently don't tolerate,  
15 you know, a big abdominal operation very  
16 well, and they have a risk for being on a  
17 ventilator after surgery and so forth. So  
18 the list is, like, pretty clear in terms  
19 of medical comorbidities.

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

25           Q.       Okay.

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

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

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

A. Correct.

Q. Okay.

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

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

Q. Okay.

A. So, you know, there -- there

1  
2 are -- you know, there are publications  
3 showing that, like for instance, increased  
4 blood loss during the operation has a  
5 little bit worse outcome, but -- but those  
6 aren't -- because they're occurring during  
7 surgery or after, they're not really  
8 modifiable. We're -- we're -- we're  
9 really looking at preoperative available  
10 factors.

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

14 A. On surgical mortality?

15 Q. Yes.

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

20 Q. Okay.

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

1  
2 surgery, and I'm unaware of the margin  
3 affecting that.

4 Q. Okay.

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

7 A. Correct.

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

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

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

21 Q. Okay.

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

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or recurrence?

A. Yeah, I would agree with that.

Q. Okay.

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

MS. SULPIZIO: Object to the form.

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

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

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

THE WITNESS: All right. Sounds good.

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

(Recess taken.)

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

BY MR. BU:

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

A. I did not.

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

A. I don't think so.

Q. Okay.

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

A. Yes.

Q. Okay.

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

MS. SULPIZIO: Object to the  
form.

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

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resected, correct.

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

MS. SULPIZIO: Object to the form.

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

Q. For the development of the cancer.

A. Yes.

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

A. Yes, correct.

Q. Okay.

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

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



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so I treat lots of different cancers.

Q. What are the other cancers that you treat?

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

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

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

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

A. Yes.

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

A. Probably less -- less than ten percent.

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

A. Probably greater than 60 percent.

Q. Are there difference types of liver cancers?

A. There are.

Q. Okay.

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

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

1  
2 recurs in the liver. That's the most  
3 common liver tumor.

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

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

15 Q. Okay.

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

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

1  
2 can occur in the same part of the liver  
3 that a cholangiole carcinoma occurs even  
4 though they're coming from different  
5 cells.

6 Q. Okay.

7 Are there also different types  
8 of histologies?

9 A. There are.

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

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

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

22 A. Yes.

23 Q. And do they have different  
24 clinical characteristics?

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

1  
2 they frequently present differently. They  
3 frequently look different on -- on  
4 radiographic imaging. They -- they behave  
5 biologically differently.

6 Q. Okay.

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

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

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

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

1  
2 the liver's entire purpose -- well, not  
3 its entire purpose. But one of the main  
4 jobs of the liver is to essentially  
5 detoxify our bodies, and when it  
6 detoxifies our body, it does so by kind of  
7 breaking down whatever toxin is -- is  
8 there, creating inflammation. And then  
9 the liver is one of the few organs in our  
10 body that regenerates; kind of grows back.  
11 It doesn't grow back. What happens is it  
12 kind of swells and increases in volume.

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

19 Q. Okay.

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

23 A. Yes.

24 Q. What other types of cancer?

25 A. I mean, many cancers. Gastric

1  
2 cancers we know can be related to, you  
3 know, infections by bacteria called  
4 H pylori that causes chronic gastritis.  
5 We know that even something as simple as  
6 skin cancers related to some exposure and  
7 repeated inflammation related to that.

8 We -- we believe that pancreatic  
9 cancer can have -- you know, can be  
10 related to chronic inflammation. We know  
11 that patients that have chronic  
12 pancreatitis, or inflammation of their  
13 pancreas, have a higher risk of developing  
14 pancreas cancer. So -- so many, many  
15 cancers, we believe, are related to  
16 chronic inflammation.

17 Q. Okay.

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

22 A. Yes.

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

25 A. Correct.

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

A. Yes.

Q. And different clinical characteristics?

A. They can.

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

A. Clear cell is the most common form.

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

A. Sure.

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

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

Q. Okay.

And what are those potential



1  
2 causes?

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

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

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

1  
2           We then looked at -- let's see.  
3       Then we looked at, you know, obviously  
4       environmental exposures and we -- and we  
5       know from -- you know, from -- from these  
6       materials that he had had a significant  
7       exposure to, you know, multiple volatile  
8       organic compounds that are -- that are  
9       known and documented to increase a risk of  
10      developing a kidney cancer.

11           Q.       Okay.

12                    Were there any other risk  
13      factors that you considered?

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

1  
2 been -- had any of those types of  
3 exposures.

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

9 Q. Okay.

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

13 A. One tumor.

14 Q. Okay.

15 And when you were explaining --

16 A. Can I can I just correct myself?  
17 I should have said unifocal.

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

21 Who is "we"?

22 MS. SULPIZIO: Object to the  
23 form.

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

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

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

MS. SULPIZIO: Object to the form.

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

Q. Okay. All right.

How did you develop this list for your differential?

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

1  
2 differential, what are the possible causes  
3 of this condition. So I did it based on  
4 experiential knowledge. I did it based  
5 upon my review of the, you know,  
6 literature, existing literature related to  
7 clear cell carcinomas of the kidney and  
8 renal cell cancers and comparing the two.

9 Q. Okay.

10 Were there any guidelines or  
11 checklists that you consulted?

12 MS. SULPIZIO: Object to the  
13 form.

14 A. Not that I'm aware of.

15 Q. Okay.

16 A. No, I don't think so.

17 Q. All right.

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

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

23 Q. Sure.

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

1  
2       attributable to the risks that you  
3       identify in your differential?

4               MS. SULPIZIO:   Object to the  
5       form.

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

12      Q.       Okay.  
13               Do you have a general sense of  
14      what those percentages are?

15      A.       I do have a general sense.

16               MS. SULPIZIO:   Object to the  
17      form.

18      A.       I do have a general sense.

19      Q.       Okay.  
20               And you may want to pause to  
21      allow Gabby time to lodge her objection.

22      A.       Sorry.

23      Q.       That's fine.

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

1  
2 BY MR. BU:

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

5 MS. SULPIZIO: Object to the  
6 form.

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

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

1  
2           So in a lot of these exposures,  
3       it's just -- it's difficult to -- it's  
4       difficult to quantify it as "yes, smoking,"  
5       "no, smoking."

6           Q.       Okay.

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

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

24          Q.       Right.

25               Do you have a sense of what



1  
2 percentage of kidney cancer cases have no  
3 identifiable risk factors?

4 MS. SULPIZIO: Object to the  
5 form.

6 A. I would -- I would guess that --  
7 or I would think that -- that as many as  
8 half of -- of kidney cancers may not have  
9 a -- a clear identifiable -- identifiable  
10 cause or source.

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

18 Q. Okay.

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

22 A. There are.

23 Q. Okay.

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

1  
2 that the patient will necessarily develop  
3 kidney cancer?

4 MS. SULPIZIO: Object to the  
5 form.

6 A. I would agree with that.

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

10 MS. SULPIZIO: Object to the  
11 form.

12 A. I would agree with that.

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

17 MS. SULPIZIO: Object to the  
18 form.

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

1  
2 a high level of exposure and never develop  
3 a cancer. So -- so even in -- with  
4 exposure to the same carcinogen, like --  
5 like tobacco, you can have people that,  
6 you know, smoke their entire lives and  
7 don't die of lung cancer, they die of  
8 aortic aneurysms and peripheral vascular  
9 disease, and heart attack's probably  
10 related to the smoking, but not cancer,  
11 and then you can have -- you can have  
12 individuals that have a relatively low  
13 exposure to the carcinogen. Maybe they  
14 just, you know -- you know, had secondhand  
15 smoke while they were in -- you know, for  
16 years and they developed a cancer.

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

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

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cause of the cancer?

MS. SULPIZIO: Object to the  
form.

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

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

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

A. I think both.

Q. Okay.

I guess what's -- what's the

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

MS. SULPIZIO: Object to the form.

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

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

MS. SULPIZIO: Object to the form.

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

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phrase that you're saying.

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

A. Sure.

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

A. No. Dose-response.

Q. Okay.

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

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

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

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

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practice?

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

Q. Yeah.

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

Q. Okay.

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

A. I don't think so.

Q. Okay.

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Have you reviewed mathematical models about the cancer slope factors?

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

Q. Okay.

Earlier you were describing the term "idiopathic."

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

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

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

MS. SULPIZIO: Object to the form.

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



1  
2 unknown cause.

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

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

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

23           Q.       Okay.

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

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potential causes of kidney cancer?

MS. SULPIZIO: Object to the  
form.

A. Correct.

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

MS. SULPIZIO: Object to --  
BY MR. BU:

Q. Is that fair to say?

MS. SULPIZIO: Sorry.  
Object to the form.

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

Q. Okay.

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

MS. SULPIZIO: Object to the

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form.

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

Q. Okay.

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

Q. Okay.

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

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

A. I think.

Q. Okay.

A. Approximately.

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Q. All right.

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

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

BY MR. BU:

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

A. I am.

Q. Okay.

And what is SEER?

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

Q. Okay.

Is SEER data considered reliable  
in your field?

A. It is.

Q. All right.

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

A. Okay.

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

A. Okay.

Q. Do you see that?

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

Common types of cancer, yes.

Q. All right.

A. Yep.

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

A. About 80,000.

Q. Okay.

A. Yeah.

Q. Does that number sound about  
right now?

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

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with you, I was thinking of deaths.

Q. Okay.

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

Q. Right.

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

MS. SULPIZIO: Object to the form.

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

We could, you know, look at

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

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

Do you recall that?

MS. SULPIZIO: Object to the for.

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

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

A. Okay.

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

A. I believe so.

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

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

Q. Sure.

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A. I was looking at the sheet when you were talking. I apologize.

Q. No, that's fine.  
Do you need more time to review that page?

A. No, it's okay.

Q. Okay.  
SEER also reports that approximately 1.8 percent of men and women will be diagnosed with kidney and renal pelvis cancer at some point during their lifetime, correct?

MS. SULPIZIO: Object to the form.

A. They do.

Q. Okay.  
Do you have any reason to disagree with that figure?

A. No.

Q. So this means approximately for every 100,000 people, about 1,800 will develop kidney cancer at some point during their lifetime?

MS. SULPIZIO: Object to the



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form.

A. Yes, 1.8 percent.

Q. Okay.

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

A. That's correct.

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

MS. SULPIZIO: Object to the form.

A. Yeah, I think so.

Q. Okay.

And here the background risk would be approximately 1.8 percent?

MS. SULPIZIO: Object to the form.

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

Q. Sure.

1  
2           Would you agree the background  
3 risk for renal cancers is about 1.8  
4 percent?

5           MS. SULPIZIO: Object to the  
6 form.

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

11           Now -- now, that being said,  
12 the -- the vast -- that's lifetime risk  
13 and -- and most kidney cancers are  
14 diagnosed later in life, people in their  
15 60s, people in their 70s.

16           So the background of all-comers  
17 developing it in their entire lifespan is  
18 1.8 percent, yes.

19           Q. Do you have any opinions about  
20 the likelihood of someone developing  
21 cancer before the age of 40 -- sorry,  
22 developing renal cancer before the age of  
23 40?

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

1  
2 produced. If you look at all -- all  
3 patients develop -- developing kidney  
4 cancer -- being diagnosed with kidney  
5 cancer before the age of 40 is pretty  
6 rare, like single-digit percentage, less  
7 than 5 percent chance. So it -- it is a  
8 bit of an outlier. Many of the patients  
9 develop kidney cancers, you know, in their  
10 sixth and seventh decades of life. That's  
11 much more common. But to develop it  
12 before the age of 40 is -- is pretty rare.

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

18 MS. SULPIZIO: Object to the  
19 form.

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

1  
2 quick, but -- but you're talking about a  
3 very, very, very small percent chance that  
4 it's going to occur in the first four  
5 decades of life.

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

8 MS. SULPIZIO: Object to the  
9 form.

10 A. Probably even lower because --

11 Q. Okay.

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

22 Q. The likelihood of someone  
23 developing renal cancer before the age of  
24 40, absent a genetic predisposition, is  
25 not zero percent though, correct?

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MS. SULPIZIO: Object to form.

A. Correct.

Q. Okay. So let me put it this way.

Do you have an opinion about how much more than zero percent that likelihood is?

MS. SULPIZIO: Object to the form.

A. Rephrase that for me. I'm sorry.

Q. Sure.

A. Yeah.

Q. So, the likelihood of someone developing renal cancer before the age of 40 absent a genetic predisposition is greater than zero, right?

A. I agree.

Q. Okay.  
And it's probably less than five if we think that all cancers -- for all cancers, only 5 percent are diagnosed before the age of 40, right?

MS. SULPIZIO: Object to the

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form.

A. I think it's less than five.

Q. Right.

A. Correct.

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

MS. SULPIZIO: Object to the form.

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

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

1  
2 compounds related to kidney cancer. So I  
3 think, like, for another patient who's  
4 under the age of 40 with no genetic  
5 predisposition, their chances of -- and --  
6 and no environmental exposures, if you  
7 have that patient, the chance of them  
8 developing a kidney cancer are  
9 extraordinarily small.

10 Q. Okay.

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

14 MS. SULPIZIO: Object to the  
15 form.

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

19 Q. Okay.

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

22 Do you recall that?

23 A. I did.

24 Q. Okay.

25 And you reviewed the general

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

A. I did.

Q. And Dr. Steven Bird?

A. I did.

Q. Okay.

Did you review the report of Dr.  
Timothy Mallon?

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

Q. Okay.

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

Q. Sure.

A. I don't remember that name.

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

A. Correct.

Q. Okay.

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



1  
2 Hatten and Dr. Bird were offering in their  
3 reports?

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

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

Q. Okay.

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

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

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

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

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

Q. Okay.

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

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

1  
2 specifics of it.

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

6 MS. SULPIZIO: Object to the  
7 form.

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

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

1  
2 opinion based upon the material, but you  
3 can't necessarily cite back and say oh,  
4 yeah I got this idea from this, you know,  
5 source or...

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

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

19 Q. Okay.  
20 When you were talking about the  
21 levels in the Reynolds report, you're  
22 saying -- you're referring to the levels  
23 that Mr. Fancher was exposed to, correct?

24 A. Correct.

25 Q. Okay.

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

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

Q. Okay.

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

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

1  
2 compare it to.

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

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

18 A. Yes.

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

21 A. I do.

22 Q. Okay.

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

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

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

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

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

A. I don't.

Q. Okay.  
Is the level of our understanding about the relationship

1  
2 between TCE and kidney cancer the same as  
3 it is for our understanding of PCE, vinyl  
4 chloride, and benzene and kidney cancer?

5 MS. SULPIZIO: Object to the  
6 form.

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

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

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

25 Q. And why is the time period



1  
2 relevant?

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

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

25 Q. Do you know if the literature is

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more consistent for TCE than for the other chemicals?

A. I don't know.

Q. Are you familiar with IARC?

A. I am not.

Q. Okay.

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

MS. SULPIZIO: Object to the form.

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

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

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Did you review the ATSDR  
Assessment of the Evidence?

A. I did.

Q. Okay.

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

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

BY MR. BU:

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

A. Okay.

Q. Do you recognize this document?

A. I do.

Q. Okay.

And what is it?

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

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Q. All right.

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

MS. SULPIZIO: Object to the form.

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

Q. Okay. So your understanding -- sorry.

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

MS. SULPIZIO: Object to the form.

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

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

Q. Okay.

ATSDR issued several publications about Camp Lejeune, right?

A. Mm-hm.

Q. Okay.

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

MS. SULPIZIO: Object to the form.

A. Sorry, say that again.

Q. Sure.

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

Can you turn to page 13 for me, please?

A. Okay.

Q. So, this is a table with the

1  
2 overall summary of the evidence for ATSDR;  
3 is that right?

4 MS. SULPIZIO: Object to the  
5 form.

6 A. Yes.

7 Q. Okay.

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

11 MS. SULPIZIO: Object to the  
12 form.

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

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

23 Q. Sure.

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

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A. For PCE --

Q. Yeah.

MS. SULPIZIO: Object to the  
form.

A. For PCE, for PCE it says below  
equipoise evidence for causation.

Q. Okay.

And then for TCE, what was the  
conclusion?

MS. SULPIZIO: Object to the  
form.

A. It says for TCE it says  
sufficient evidence for causation.

Q. All right.

And is your understanding that  
these conclusions are related to the  
levels of contaminants at Camp Lejeune?

MS. SULPIZIO: Object to the  
form.

A. I think these are based upon  
the -- like again I'm looking at just this  
one figure out of context of the entire  
document, but I think these are -- these  
are trying to summarize the existing

1  
2 literature on what the evidence is, not  
3 specifically looking at the Camp Lejeune  
4 data. It's -- it's talking about what is  
5 the evidence in the -- in the published  
6 literature and summarizing that.

7 Q. Okay.

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

13 A. They could be.

14 Q. Okay.

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

18 A. I did.

19 Q. All right.

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

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



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Q. Okay.

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

MS. SULPIZIO: Object to the form.

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

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

1  
2 high-impact journals because in theory the  
3 peer review process is a little more  
4 stringent.

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

10 Q. Okay.

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

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

16 A. Okay.

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

20 A. Okay.

21 Q. The last page of the exhibit.

22 A. Okay.

23 Q. Okay.

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

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

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

A. It does.

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

A. I see that.

Q. Okay.  
Did you review those articles?

A. I don't specifically remember reviewing them.

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

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

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

Q. Okay.

All right. You can set that  
aside.

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

A. Okay.

Q. All right.

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

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

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

A. Here we go, yep.

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

A. Correct.

Q. Okay.

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

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that right?

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

Q. Okay. Let me ask this. Did you identify the Bove article yourself, or how did you -- how did you come across the Bove article?

MS. SULPIZIO: Object to the form.

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

Q. Okay. And how did you develop this list of 23 levels?

MS. SULPIZIO: Object to the form.

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

1  
2 papers, and just basically putting  
3 together a list of, you know, exposure  
4 levels from the literature that looked to  
5 be, you know, consistent with  
6 carcinogenesis.

7 Q. Okay.

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

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

12 Q. Okay.

13 Did you review the Aschengrau  
14 article?

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

18 MR. BU: Okay.

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

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

24 (Recess taken.)

25 THE VIDEOGRAPHER: The time

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

BY MR. BU:

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

A. I did not.

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

A. I don't think so.

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

BY MR. BU:

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

Do you recognize this document?

A. I do.

Q. And what is it?

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

Q. Okay.

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

A. Yes.

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

A. Okay.

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

A. Correct.

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

MS. SULPIZIO: Object to the form.

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

Q. Okay.



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Can you turn to page 289 of  
Exhibit 8?

A. Okay.

Q. All right.

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

Do you see that?

A. I do.

Q. Okay.

Does that paragraph describe a  
27 to 44 milligram range?

MS. SULPIZIO: Object to the  
form.

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

Q. That's fine.

A. (Witness reads document.)

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

Q. Okay.

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

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A. I do.

Q. All right.

And do you see that last sentence for this -- the first paragraph of "Discussion"?

A. I do.

Q. All right.

And so, Aschengrau and the co-authors concluded that no kidney cancer cases were considered exposed when latency was taken into account; is that right?

MS. SULPIZIO: Object to the form.

A. In their discussion, they say: No kidney cancer cases were considered exposed when latency was taken into account.

Q. Okay.

And they also say: No meaningful increases in the risk of kidney cancer were detected without latency.

Is that right?

MS. SULPIZIO: Object to the form.

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

Q. Okay.

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

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

Q. Okay.

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

MS. SULPIZIO: Object to the form.

A. Say it one more time.

Q. Sure.

A. Sorry.

Q. Is considering latency one of

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

A. It is one way that you could.

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

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

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

MS. SULPIZIO: Object to the form.

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

MR. BU: All right.

Can we pull up tab 12, please?

And you can set Aschengrau aside.

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

BY MR. BU:

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

Have you seen this document  
before?

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

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

A. Yeah.  
Q. -- is that right?  
A. Yeah. I have seen this, yeah.  
Yeah, I've -- I've reviewed this.

Q. Okay.  
A. For sure.

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

A. Yeah, I believe -- yes.

Q. Okay.

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

Is that right?

A. Correct.

Q. And a ppb is a part per billion?

A. Yes.

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

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

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

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Q. Okay.

A. Correct.

Q. So this is measuring something a little bit different than Aschengrau which was a cumulative exposure?

A. I mean, this is getting back to the days of, you know, conversions and calculations and...

Yeah, I mean, they're -- they're just representing it as parts per billion as opposed to Aschengrau which represented it as -- as like a total mass of, like, 27 to 44 milligrams, total mass ingestion or exposure versus you know, a exposure to a -- to a cumulative concentration.

Q. Okay.

Would you agree that determining the duration of an exposure to a given concentration is relevant to whether that exposure can cause cancer?

MS. SULPIZIO: Object to the form.

A. I mean, I think it probably depends. Sort of like we talked about the

1  
2 dose-response curve earlier, it may be  
3 that, you know, certain high doses at a  
4 short duration may have a similar effect  
5 to a lower dose over a long duration, and  
6 I think it's -- I -- I think they're -- I  
7 think they're probably interrelated.

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

21 Q. Okay.

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



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a 20-year exposure and a 50-year exposure?

A. I mean, I would think so.

Q. And that's because of the dose-response relationship?

MS. SULPIZIO: Object to the form.

A. I mean, it just -- I would believe that if you had the same level of ex -- I mean the same amount of exposure for a longer period of time that it probably -- you know, a longer exposure to the same carcinogen you would think would have a higher impact.

Q. Do you know what the median duration of exposure was for the cases in Moore 2010?

A. I don't recall.

Q. Okay.

Can you turn to the table -- to Table 1 on page 6,531. I don't know why they're numbered that way, but they are.

A. They're numbered that way 'cause cancer research literally publishes all their papers in order for the year. So

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

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

Q.       Okay.

A.       That's a different journal.

Q.       Do you see the footnotes to Table 1?

A.       I do.

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

          Do you see that?

A.       I do.

Q.       Okay.

          What's an interquartile range?

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

1  
2 percent. So you divide the entire group  
3 into -- into fours, and then you can look  
4 at any one of those quartiles.

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

9 MS. SULPIZIO: Object to the  
10 form.

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

13 Q. Yeah.

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

16 Q. Okay.

17 Above that do they also measure  
18 exposure duration in years?

19 A. They do.

20 Q. Okay.

21 A. Sorry, yes.

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

24 A. The median was 19.5 years.

25 Q. All right.

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And what was the interquartile range?

A. 5.8 years to 31 years.

Q. Okay.

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

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

Q. Okay.

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

MS. SULPIZIO: Object to the form.

Q. -- or in another median?

MS. SULPIZIO: Sorry.

Object to the form.

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

Q. Okay.

A. I would assume, I mean, water

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

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

Do you see that? Line 3.

A. Okay. Yep.

Q. Okay.

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

A. Yes.

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

A. Yes.

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

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

MS. SULPIZIO: Object to the  
form.

A. I'm not sure.

MR. BU: Can we please pull tab  
13?

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

BY MR. BU:

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

A. Okay.

Q. Have you seen this document  
before?

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

Q. What is this document?

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

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

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page 8 of your Fancher report?

MS. SULPIZIO: Object to the  
form.

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

Q. Okay.

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

A. Correct.

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

Is that fair to say?

MS. SULPIZIO: Object to the  
form.

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

You know, whether you evaluate

1  
2 it as just total exposure or total  
3 exposure over time, honestly, I think I  
4 would have to rely on -- on people that  
5 evaluate, you know, environmental  
6 exposures and the development of cancer,  
7 you know, to -- to -- to found an opinion,  
8 which is what I've done.

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

12 MS. SULPIZIO: Object to the  
13 form.

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

17 Q. Okay.

18 Can you turn to page 5, Table 2?

19 A. Okay.

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

23 A. Yes.

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



1  
2 15-year exposure?

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

5 Q. Okay.

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

11 MS. SULPIZIO: Object to the  
12 form.

13 A. Yes.

14 Q. Okay.

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

18 A. I'm sorry, in Table?

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

21 A. Okay. Yeah, and the odds  
22 ratios.

23 Q. Okay.

24 A. Yes. Yes, I do.

25 Q. And what's your understanding of

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what an odds ratio's approximating?

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

Q. Okay.

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

A. It does.

Q. Okay.

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

A. Correct.

Q. All right.

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

MS. SULPIZIO: Object to the form.

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

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

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

Q. Sure.

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

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

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

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

A. Yes.

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

MS. SULPIZIO: Object to the form.

A. I mean, probably. Although as

1  
2 we talked about before, probably different  
3 individuals have different susceptibility  
4 to some of these carcinogenic, you know,  
5 agents. So what -- you know, some  
6 patients with a very, very low level of  
7 exposure may be more prone to develop a  
8 kidney cancer, whereas other patients may  
9 require a somewhat higher level.

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

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

18 A. It does.

19 Q. All right.

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

23 MS. SULPIZIO: Object to the  
24 form.

25 A. No.

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Q. Okay.

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

A. I believe it's capable.

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

And you can set that one aside.

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

BY MR. BU:

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

A. Okay.

Q. Do you recognize this document?

A. I do.

Q. What is it?

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

Q. Okay.

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

MS. SULPIZIO: Object to the form.

A. Yes.

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

A. Correct.

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

A. Yes.

Q. Okay.

Do you recall how long those residential exposures were for?

MS. SULPIZIO: Object to the form.

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

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this paper were.

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

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

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

THE WITNESS: Okay.

BY MR. BU:

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

Do you recognize this document?

A. I do.

Q. And what is it?

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

Q. Okay.

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

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right?

A. I do.

Q. All right.

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

A. It does.

Q. Like low, medium, high exposures?

A. Low, medium, and high, correct.

Q. Okay.

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

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

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

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



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BY MR. BU:

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

A. Okay.

Q. Do you recognize these tables?

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

Q. Okay.

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

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

A. Yeah. Yeah, it did.

Q. Okay.

And do you recall reviewing those supplemental tables?

A. I do.

Q. All right.

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

So, level 9 is cumulative

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

A. That's correct.

Q. Okay.

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

A. That's correct.

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

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

Q. Okay.

A. Yes.

Q. All right.

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

A. Correct.

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

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

A. Correct.

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

A. I do.

Q. Okay.

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

MS. SULPIZIO: Object to the form.

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

1  
2 something's not statistically significant  
3 doesn't mean it's not clinically  
4 significant. So the hazard ratios are --  
5 are clearly elevated, but they do cross  
6 one.

7 Q. Okay.

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

12 MS. SULPIZIO: Object to the  
13 form.

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

18 Q. Okay.

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

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

25 A. That's correct.

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Q. Okay.

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

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

Q. Okay.

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

A. No, I dis --

MS. SULPIZIO: Object to the form.

A. I disagree. You know, just

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

8 Q. Okay.

9 And these hazard ratios in the  
10 supplement are what you refer to for items  
11 7, 10, and 13 in your Fancher report; is  
12 that right?

13 A. Correct.

14 Q. Okay.

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

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

20 A. That's correct.

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

24 A. That is correct.

25 MS. SULPIZIO: Object to the

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form.

BY MR. BU:

Q. Okay.

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

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

Q. Okay.

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

MS. SULPIZIO: Object to the form.

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

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but I agree with that statement.

Q. Okay.

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

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

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

Do you see that?

MS. SULPIZIO: Object to the  
form.

A. I do.

Q. Okay.

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

A. Yes.

Q. Okay.

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

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



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Q. Yeah.

A. Repeat that question, 'cause I'm not sure I understood it. Sorry.

Q. Let me ask it this way.

Why do you associate 18 months of residence on base from 1975 to 1985 with an increased incidence of kidney cancer?

MS. SULPIZIO: Object to the form.

A. I'd have to look. To be honest with you, it's been a -- it's been a little while since I re-reviewed this, but I think -- I think that's -- I shouldn't say 'cause I'm guessing.

I think -- I think it's coming from the Bove report as somehow they zeroed in on that length of time. I don't know if that was the average length of time or -- I think -- I think it was the average length of time that a resident was felt to be on base at Camp Lejeune, if I recall correctly.

Q. Okay.

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

Q. Okay.

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

A. Okay.

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

A. I do.

Q. Okay.

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

A. Correct.

Q. Okay.

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

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right?

A. Yes.

Q. And the p-value is .19?

A. Yes.

Q. Okay.

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

Is that fair to say?

A. Yes.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Okay.

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

Is that fair to say?

A. Yes.

MR. BU: Can we please pull tab 9?

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

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BY MR. BU:

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

A. Okay.

Q. Do you recognize this document?

A. I do.

Q. And what is it?

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

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

MS. SULPIZIO: Object to the  
form.

A. Correct.

Q. Okay.

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

A. Okay.

Q. And this is reporting hazard

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

A. It does.

Q. Okay.

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

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

Q. Yeah.

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

A. It is.

Q. Okay.

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

A. That's fair to say.

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

A. That's fair to say.

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

1  
2 these effect estimates have limited  
3 statistical precision?

4 MS. SULPIZIO: Object to the  
5 form.

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

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

22 Q. Okay.

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

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significance with a smaller N?

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

Q. Okay.

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

A. Correct.

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

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

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

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

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



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BY MR. BU:

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

Do you recognize this document?

A. I do.

Q. And what is it?

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

Q. Okay.

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

A. Correct.

Q. Okay.

And items 18 and 20 relate to  
TCE exposures?

A. Correct.

Q. And 19 and 20 relate to PCE  
exposures?

A. Correct.

Q. Okay.

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Can you turn to page 76,  
Table 7?

A. Okay.

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

A. Yes.

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

A. It is.

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

A. I'm sorry, say that again.

Q. Sure.

A. Sorry.

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

MS. SULPIZIO: Object to the

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form.

BY MR. BU:

Q. Is that right?

A. Yes.

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

A. It would.

Q. Okay.

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

A. Yes.

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

A. Yes.

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

A. It does.

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

A. It does.

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Q. Okay.

All right. Can you turn to the next page of ATSDR 2018?

I'm sorry, two more pages to Table 8.

A. Is -- I'm sorry, on page 79?

Q. Page 78.

A. Page 78, okay.

Q. All right.

And this table describes odds ratios for cumulative exposure to PCE; is that right?

A. Yes.

Q. Okay.

And do you see kidney cancer in Table 8?

A. I do.

Q. Okay.

And similar to before, item 19 in your report exposure to 36 to 711 ppb months of PCE corresponds to the medium exposure in Table 8; is that right?

A. Yes.

Q. And item 21 cumulative exposure

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

A. Correct.

Q. Okay.

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

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

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

A. That is correct.

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

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

BY MR. BU:

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

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A. I do. I do.

Q. Okay.

A. Yes.

Q. And what is it?

A. It's another paper by Bove that came out in October 2024 "Cancer Incidence Among Marines and Navy Personnel and Civilian Workers Exposed to Industrial Solvents in Drinking Water At US Marine Corps Base Camp Lejeune: A Cohort Study."

Q. This is not a mortality study; is that right?

A. Correct. This is a cancer incidence study.

Q. Can you explain what the difference is between a mortality study and a cancer incidence study?

A. Sure. So, I mean, a mortality study is the, you know, the patients -- or, the -- the patients are -- you know, die of their disease. An incidence is -- is just that they developed the disease.

So it becomes very important in -- in cancers that have a, you know,

1  
2 have a large number of patients that  
3 survive their cancer. Frequently we look  
4 at incidences as opposed to survival. So  
5 they're just -- they're just, you know,  
6 different measurements. One is you die of  
7 the disease and the other is you developed  
8 the disease. You may be cured of the  
9 disease. You may not be cured of the  
10 disease. But if -- incidence is the  
11 disease developed.

12 Q. Okay.

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

16 MS. SULPIZIO: Object to the  
17 form.

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

23 Q. Okay.

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

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A. I'm sorry, Table?

Q. Page 7, Table 3.

A. Okay. Got it.

Q. It says "Comparison of cancer outcomes at Camp Lejeune versus Camp Pendleton among with the Marines/Navy subgroup" at the top.

A. Yes.

Q. And Table 3 reports some hazard ratios for kidney cancer; is that right?

A. Yes. I'm just trying to find it here.

Yes, there it is.

Q. Okay.

And then it also reports hazard ratios for different subtypes of kidney cancer?

A. Yes.

Q. The confidence intervals for all of these hazard ratios also crosses one; is that right?

A. Yes.

Q. Okay.

And the hazard ratios for clear



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2 cell only are less than one; is that  
3 right?

4 A. Yes.

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

9 MS. SULPIZIO: Object to the  
10 form.

11 A. Just repeat that, I'm sorry.

12 Q. Sure. Let me ask it this way.  
13 How would you interpret a hazard  
14 ratio of less than one?

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

23 Q. Okay.

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

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increased incidence of that type of  
disease?

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

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

A. Okay.

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

A. Yes.

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

A. That's correct.

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

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A. That's correct.

Q. Okay.

The confidence intervals for kidney cancer on the Table 4 are -- they also all cross one; is that right?

A. Correct.

Q. Okay.

And is Table 4 what you referred to for item 23 in your Fancher report, more than 21 quarters spent on base as a non-civilian worker?

MS. SULPIZIO: Object to the form?

A. Yes.

Q. Is Table 3 what you refer to for item 22, one to six quarters stationed on base as a service member from 1975 to 1985?

MS. SULPIZIO: Object to the form.

A. Yes.

Q. Okay.

For the levels described in items 1 through 23, were there any other

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articles that you relied on?

MS. SULPIZIO: Object to the  
form.

A. I don't think so.

Q. Okay.

And in all the Bove and ATSDR  
studies, the only statistically  
significant association was the high  
exposure group for PCE in the 2018  
morbidity study; is that right?

MS. SULPIZIO: Object to the  
form.

A. I mean, they -- they all had  
increased hazard ratios, but -- but the  
confidence intervals, 95 percent  
confidence intervals crossed one.

Q. Okay.

So there was only one  
association that we looked at where the  
confidence interval did not cross one.

Is that fair to say?

MS. SULPIZIO: Object to the  
form.

A. There was one where we can say

1  
2 with 95 percent confidence. The remainder  
3 we -- you know, had increased hazard  
4 ratios.

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

10 MS. SULPIZIO: Object to the  
11 form.

12 A. I mean, it's possible.

13 Q. Okay.  
14 But that's what we would expect,  
15 right?

16 MS. SULPIZIO: Object to the  
17 form.

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

20 Q. Sure.

21 A. Sorry.

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

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

MS. SULPIZIO: Object to the  
form.

A. Yes.

Q. Okay.

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

Q. One out of 20.

MS. SULPIZIO: Object to the  
form.

BY MR. BU:

Q. 5 percent.

MS. SULPIZIO: Object to the  
form.

A. I don't think I understand.

Q. Okay.

A. So let's back up.

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

MS. SULPIZIO: Object to --

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

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MS. SULPIZIO: Sorry.

Object to the form.

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

Q. Sure.

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

Q. No, it's okay.

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

A. Correct.

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

Is that fair to say?

MS. SULPIZIO: Object to the  
form.

A. That's correct.

Q. Okay.

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

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

MS. SULPIZIO: Object to the form.

A. That's correct.

Q. Okay.

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

MS. SULPIZIO: Object to the form.

A. Specifically Camp Lejeune water and kidney cancer?

Not -- no.

Q. Okay.

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

MS. SULPIZIO: Object to the form.



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A. Say that one more time. Sorry.

Q. Sure.

So, 1 through 23 you list several levels of exposure that have been found to be correlated with kidney cancer; is that right?

MS. SULPIZIO: Object to the form.

A. Correct.

Q. Okay.

Are you aware of any other levels that have been found to be correlated with kidney cancer other than the ones listed in 1 through 23 of your report?

A. I'm not aware of any, or I'm certainly not thinking of any right now off the top of my head.

MR. BU: Okay.

All right. I think this is a good place to stop.

THE VIDEOGRAPHER: The time right now is 12:55 p.m., and we're off the record.

(Luncheon recess taken.)

- - -

A F T E R N O O N       S E S S I O N

- - -

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

BY MR. BU:

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

A. I did not.

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

A. No.

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

Do you recall writing that?

A. I do.

Q. Okay.

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

A. I don't quantify it. I said

1  
2 substantial because based upon Dr.  
3 Reynolds' charts and graphs and  
4 calculations as to what his exposure level  
5 was, the amount that he was exposed in  
6 comparison to what the medium, low, and  
7 high risk exposure was considered in the  
8 Bove papers, I felt that it was a  
9 substantial amount that he was exposed to,  
10 but I don't quantify that.

11 Q. Okay.

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

16 A. I do not.

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

20 A. I did not.

21 Q. Okay.

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

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Q. Okay.

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

MS. SULPIZIO: Object to the form.

A. I did not.

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

A. Okay.

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

A. I do.

Q. And there are four items listed?

A. I do.

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

Do you see that?

A. I do.

Q. Okay.

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

1  
2           A.       Well, this -- this was based  
3       upon -- this was what Dr. Reynolds used to  
4       calculate this chart. It wasn't me. And  
5       she utilized what the ATSDR had -- had  
6       estimated a marine on base would have been  
7       exposed to, or actually just ingested,  
8       'cause it's actually an underestimate,  
9       just what the ATSDR data would have  
10      suggested that a marine on base for that  
11      amount of time would have been -- would  
12      have ingested.

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

20           Q.       Okay.

21                 Do you see the next full  
22      paragraph on page 10?

23           A.       Yes.

24           Q.       All right.

25                 And it reads: I formed my

1  
2 opinion that Mr. Fancher had substantial  
3 exposure solely based upon Mr. Fancher's  
4 deposition, the elevated levels of  
5 concentrations in the water, and  
6 additional documents from Mr. Fancher's  
7 file.

8 Do you see that?

9 A. I do.

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

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

25 Q. Okay.

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

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

Q. Okay.

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

Are you referring to the concentration or something else?

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

Q. Okay.

What concentrations would you consider intense?

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MS. SULPIZIO: Object to the  
form.

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

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

A. I don't recall.

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

A. Correct.

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

Is that fair to say?

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

Q. Okay.

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



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right?

A. She does.

Q. Okay.

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

MS. SULPIZIO: Object to the form.

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

Q. Okay.

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

A. It may.

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

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

1  
2 studies that talk about total exposure  
3 level as a -- I mean, I think they're a  
4 total mass, but I'll be honest, I'm not  
5 sure. I'm not sure your line of  
6 questioning.

7 Q. Okay.

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

12 A. Yes.

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

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

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

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

25 Like for instance, with Mr.

1  
2 Fancher, he -- his BMI was, like, pretty  
3 normal when he was -- you know, when he  
4 was exposed to these chemicals. You know,  
5 clearly these chemicals are metabolizing  
6 and distributed throughout the body. So  
7 if someone is in, like, an extreme of  
8 weight, it may have an effect on -- on the  
9 overall cancerogenic potential. But --  
10 but to be honest with you, I don't know if  
11 we know that either way.

12 Q. Okay.

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

16 A. Yes.

17 Q. What are some examples?

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

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

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

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larger dose for a larger patient.

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

A. That's correct.

Q. Okay.

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

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

A. That's correct.

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

A. Yes, I believe she did.

Q. Did you calculate that total VOC exposure?

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A. I didn't calculate it, but I --

Q. Okay.

A. I relied on Dr. Reynolds' calculations.

Q. All right.

And -- okay. I think you mentioned that you think that Dr. Reynolds's calculations underestimate the true exposure for Mr. Fancher; is that right?

A. I do.

Q. Okay.

And that's because it -- Dr. Reynolds's calculations account only for ingestion and not for inhalation and dermal exposure; is that right?

A. That's correct.

Q. Do you know whether the risks for these chemicals is different between ingestion, inhalation, and dermal exposure?

A. No, I don't.

Q. If Dr. Reynolds's ingestion overestimated Mr. Fancher's actual

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ingestion, is it possible that her charts would not underestimate his total exposure?

MS. SULPIZIO: Object to the form.

A. I don't understand the question. Say it -- say it again, please.

Q. Sure.

So, Dr. Reynolds calculates based on Mr. Fancher's deposition testimony and some field manuals that he ingested about 577,000 micrograms of TCE; is that right?

MS. SULPIZIO: Object to the form.

A. That's correct.

Q. Okay.

And that calculation is based, in part, on the ATSDR's water model; is that correct?

A. It's based in part on the ATSDR modeling, but it's also taking into account -- for the 577,000. It's also taking into account Mr. Fancher's

1  
2 deposition and testimony about how -- how  
3 often he was drinking, how often he was  
4 showering, et cetera.

5 Q. Okay.

6 Part of that calculation is  
7 determining the concentrations of TCE in  
8 Camp Lejeune water at the time Mr. Fancher  
9 was at Camp Lejeune; is that right?

10 A. That's correct.

11 Q. Okay.

12 Do you know where those  
13 concentrations come from?

14 A. They come from the ATSDR.

15 Q. Okay.

16 So I guess what I'm really  
17 trying to ask is if those concentrations  
18 are greater than what they actually were  
19 at Camp Lejeune, is it possible that the  
20 577,000 micrograms overestimates Mr.  
21 Fancher's total ingestion?

22 MS. SULPIZIO: Object to the  
23 form.

24 A. Yeah, I mean, it -- so what  
25 you're saying is if the ASDR -- ATSDR

1  
2 reported concentrations in the water  
3 supply were wrong and actually the  
4 concentration was much lower, then would  
5 that mean this is an overestimation of  
6 what Mr. Fancher was exposed to, then yes.

7 Q. Okay.

8 A. But we're relying on the ATSDR  
9 for the -- you know, for the  
10 concentrations that were present, or at  
11 least that's what Dr. Reynolds relied on.

12 Q. And if the 577,000 microgram  
13 ingestion isn't overestimate, it might not  
14 then underestimate the total exposure if  
15 the, I guess, the error is greater than  
16 the inhalation and dermal exposures, if  
17 that makes sense.

18 MS. SULPIZIO: Object to the  
19 form.

20 A. I mean, we don't know what the  
21 inhalation expose -- I mean, we -- we --  
22 the inhalation exposure and the dermal  
23 contact isn't factored into this equation.  
24 So we're getting into, kind of,  
25 hypotheticals that are really hard to come



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2 up with, but it would depend on how -- how  
3 far they were off on the ATSDR estimation  
4 of the concentrations versus how much more  
5 inhalation and dermal contact, and, you  
6 know, you'd have to compare those two.

7 Q. Right.

8 Are you aware of any  
9 calculations for Mr. Fancher's inhalation  
10 or dermal exposures?

11 A. I'm not.

12 Q. All right.

13 So, if there is some  
14 underestimation of the concentration at  
15 Camp Lejeune, we wouldn't be able to  
16 determine whether Dr. Reynolds's  
17 calculations overestimate or underestimate  
18 Mr. Fancher's total exposure?

19 MS. SULPIZIO: Object to the  
20 form.

21 A. I mean, if the -- now you're --  
22 if there's an underestimate of the  
23 concentration of chemicals by the ATSDR,  
24 then that would mean that all of these  
25 concentrations in Dr. Reynolds' actually

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should be -- should be higher.

Q. Okay.

Would -- if ATSDR overestimated the concentration levels, we wouldn't be able to determine whether Dr. Reynolds's calculations overestimate or underestimate Mr. Fancher's total exposure because we don't know the delta for the inhalation or dermal exposures.

Is that fair to say?

MS. SULPIZIO: Object to the form.

A. It wouldn't be that we couldn't calculate it because we don't know the dermal and inhalation. It would be because if -- if their estimates were wrong, then we wouldn't be able to rely on their estimates.

Q. Okay.

Did Mr. Fancher have a cumulative exposure to 27 to 44 milligrams of PCE?

A. How much, I'm sorry? Say that again?

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Q. Sure.

A. Did he have a cumulative exposure of?

Q. Of 27 to 44 milligrams of PCE.

MS. SULPIZIO: Object to the form.

A. 27 -- I'm not sure I'm reading you correctly. I'm not -- I don't think I'm understanding you correctly.

You're asking what was Mr. Fancher's cumulative exposure to PCE?

Q. Yes.

A. I mean, his cumulative exposure's, based upon Dr. Reynolds, was, you know, 247.

Q. 247 what?

A. Micrograms per liter month.

Q. Okay.

Does Dr. Reynolds use that to calculate possible consumption in milligrams or micrograms?

MS. SULPIZIO: Object to the form.

A. No.

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Q. Okay.

Did Mr. Fancher have an exposure to 1,580 ppb years of TCE?

MS. SULPIZIO: Object to the form.

A. I'm not sure -- I'm not sure I understand where that -- where you're getting that number from.

Q. Sure.

So, go back to page 8 of your report.

A. Yeah.

Q. And these are the levels you've listed that are found to be correlated with kidney cancer.

A. Okay.

Q. Right.

And the first level is cumulative exposure to 27 to 44 milligrams of PCE.

A. Okay.

Q. We can skip the second one.

The third one is cumulative exposure to greater than 1,580 ppb years

1  
2 of TCE, right?

3 A. Okay.

4 Q. So based on your understanding  
5 of Dr. Reynolds's report, did Mr. Fancher  
6 have an exposure to 1,580 ppb years of  
7 TCE?

8 MS. SULPIZIO: Object to the  
9 form.

10 A. Again, you're -- the problem  
11 with a lot of these studies is they --  
12 people interchangeably use "parts per  
13 billion" as a concentration as opposed to  
14 the "micrograms per liter months."

15 So, it -- it's very difficult  
16 for me to compare the two directly.

17 Q. Okay.

18 Did Mr. Fancher have a sustained  
19 exposure to zero to 25 parts per billion  
20 TCE?

21 A. I mean, I think his exposure --

22 MS. SULPIZIO: Object to the  
23 form.

24 A. I think his exposure was  
25 probably higher than that.

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Q. Higher in terms of the concentration of TCE?

A. It's probably higher in terms of total amount mass ingested.

Q. What do you think -- do you have an opinion about the total mass ingested in the Andrew 2022 study?

A. No.

Q. Okay.  
Do you recall if they described total mass ingested in that study?

A. I don't recall.

Q. All right.  
And earlier we discussed that the study was looking at 5-, 10-, and 15-year residential exposures, right?

A. Correct.

Q. Okay.  
Did Mr. Fancher have a five-year residential exposure?

A. No.

Q. Okay.  
His exposure was less than one year, right?

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MS. SULPIZIO: Object to the  
form.

A. His exposure was from, you know,  
October of -- you know, October of '79 to  
June of '81. So it was, like, 337 days.

Q. So less than one year?

MS. SULPIZIO: Object to the  
form.

A. Less than one year.

Q. Okay.

And for the Parker study, items  
5 and 6 on your report, you don't know how  
long the duration of those exposures in  
the -- the Parker study were, right?

A. That's correct.

Q. Okay.

A. 'Cause it's just reported as a  
total.

Q. Okay.

So we can -- we can't determine  
whether Mr. Fancher's exposure was similar  
in duration to those in the Parker study.

Is that fair to say?

MS. SULPIZIO: Object to the

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form.

A. I think that's fair to say.

Q. Okay.

And because Parker was looking at residential exposures in Woburn, is it fair to say that Mr. Fancher's one-year exposure at Camp Lejeune was shorter than those in the Parker study?

MS. SULPIZIO: Object to the form.

A. I think it's hard -- I think it's really it's hard to know because these were -- this is like a community retrospective study and you don't -- you don't have nearly as much information about those people and where they were living and for how long and what they were exposed to as you did -- as you do in Mr. Fancher's case where because he was in the military, you know what days he was living there and what days he was living off campus.

So I think you -- you have a lot more information about Mr. Fancher than



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you could possibly have in a  
retrospective, kind of, community  
observational study.

Q. Okay.

So do you think the durations of  
exposure in Parker are comparable to Mr.  
Fancher's one-year exposure?

MS. SULPIZIO: Object to the  
form.

A. I don't think I can say.

Q. Was Mr. Fancher exposed to PCE  
at 21 parts per billion?

MS. SULPIZIO: Object to the  
form.

A. I mean, again, the -- the  
calculations by Dr. Reynolds were not in  
parts per billion. So, you know, I think  
you would -- I think you would have to  
literally, you know, do a calculation  
to -- to convert, but I think it would be  
higher. I think it would be higher, to be  
honest with you.

Q. Okay.

And why do you think it would be

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higher than 21 parts per billion?

A. Because of the -- because of the level that was, you know, in the water, the concentration, and the period of time he was there. I think it would have -- it was a very high -- it was a high concentration of TCE, PCE, all of these, you know, compounds.

Q. Okay.

MR. BU: Sharon, can we pull tab 25, please?

(Weiss Exhibit 17, Appendix 7 David William Fancher (Kidney Cancer), was marked for identification, as of this date.)

BY MR. BU:

Q. I've handed you what's marked as Exhibit 17.

Have you seen this document before?

A. Boy, that is really small print. Yes.

Q. And what is it?

A. It's a -- it's a table that was

1  
2 calculated by Dr. Reynolds looking at  
3 specifically for Mr. Fancher and potential  
4 exposure to each of the organic compounds,  
5 and it's an estimate first based upon if  
6 you just assumed he had a one liter per  
7 day ingestion and then by utilizing what  
8 the ATSDR had said that a, you know, a  
9 marine staying on base would have been  
10 exposed to, and then lastly, it's a --  
11 it's a combination of the ATSDR -- ATSDR  
12 used for the concentration of exposure  
13 combined with Mr. Fancher's testimony as  
14 to how often he was, you know, felt to be  
15 ingesting --

16 Q. Okay.

17 A. -- the water.

18 Q. Can you turn I guess it's to the  
19 third page of this exhibit?

20 A. Okay.

21 Q. And there's a table with blue  
22 headers on the right-hand side for TCE,  
23 PCE, VC, and BZ.

24 Do you see that?

25 A. Is it Chart 1 "Days on Base and

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Cumulative"?

Q. Yes.

A. Okay. I see blue headers.

Q. All right.

And are these blue headers describing the concentration of contamination for the different VOCs at these different points in time?

A. Yes.

Q. Okay.

And for PCE, most of these concentration levels are less than 21 micrograms per liter month; is that right?

MS. SULPIZIO: Object to the form.

A. Yeah, it looks like all of them but two.

Q. Okay.

A. No, three.

Q. And you said there's some uncertainty about translating micrograms per liter month to ppbs; is that right?

MS. SULPIZIO: Object to the form.

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2           A.       I think that, unfortunately, at  
3       least based upon my review of these  
4       literature, these -- these concentrations  
5       are almost -- almost seemed seem to be  
6       used interchangeably. And so it's hard  
7       for me -- it's hard to compare.

8           Q.       Okay.

9                    Looking at item 13 of your  
10       Fancher report, would you agree that Mr.  
11       Fancher's cumulative exposure was not  
12       greater than 7,700 micrograms per liter  
13       month of TCE?

14                   MS. SULPIZIO: Object to the  
15       form.

16           A.       Mr. Fancher's exposure was  
17       believed to be a little over, you know,  
18       5,340 to TCE.

19                   So is the question is that less  
20       than 7,000? Yes.

21           Q.       Okay.

22                   And he was not exposed to  
23       greater than 380 micrograms per liter  
24       month of PCE, right?

25           A.       That's correct. He was exposed

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to 200 -- estimated to be 247.

Q. Okay.

And he was not exposed to more than 12,250 micrograms per liter month of all compounds at Camp Lejeune?

MS. SULPIZIO: Object to the form.

A. I mean, I think, again, these are all estimates. So I can't say that he was not exposed to that much.

The -- the estimates of total organic compounds exposed to, what -- what number did you -- did you say again?

Q. Sure.

This is item 15 in your report. Mr. Fancher was not exposed to greater than 12,250 micrograms per liter month to all compounds at Camp Lejeune?

MS. SULPIZIO: Object to the form.

A. I think based upon these un -- and, again, what I thought were underestimates by Dr. Reynolds, they would be less than -- his exposure would be less

1  
2 than 12,250, but, again, based upon what I  
3 think are underestimates by Dr. Reynolds.

4 Q. Okay.

5 Are you able to determine the  
6 magnitude of that underestimate?

7 A. I'm not.

8 MS. SULPIZIO: Object to the  
9 form.

10 BY MR. BU:

11 Q. Are you able to determine, based  
12 on that underestimate, how likely it would  
13 be for Mr. Fancher to have been exposed to  
14 more than 12,250 micrograms per liter  
15 month of all compounds?

16 MS. SULPIZIO: Object to the  
17 form.

18 A. I can't.

19 Q. Okay.

20 Looking at item 20, similarly,  
21 you would agree that Mr. Fancher was not  
22 exposed to greater than 11,030 ppb months  
23 of PCE?

24 MS. SULPIZIO: Object to the  
25 form.

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BY MR. BU:

Q. Right?

A. I -- I can't say.

Q. And why can't you say?

A. Well, again I think, you know, we're -- ask the question again? I'm sorry, I'm getting confused by your question.

Q. That's fine.

Would you agree that Mr. Fancher's cumulative exposure to TCE was not greater than 11,030 ppb months?

A. I don't think I can -- I don't think I can -- I don't think I can comment on that or calculate that.

Q. And why don't you think you can comment on that?

A. Because again we're looking at, you know, parts per billion months, and much of the analyses that I've used to look at Mr. Fancher's exposure have been based upon Dr. Reynolds, who's utilizing a, you know, micrograms per liter months, and -- and unfortunately they -- they seem



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to be using them interchangeably, but it's difficult to compare the two.

Q. Okay.

So you can't compare Mr. Fancher's exposures to item 20 either way?

MS. SULPIZIO: Object to the form.

A. I think -- I mean, I think you can. I think, you know, some of these studies, like the ATSDR, you know, that -- that's -- you know, just because they use that metric don't mean they're -- they can't be compared.

You know, I'm relying on Dr. Reynolds who's an expert on -- in putting these calculations together and then -- and then using those calculations that she's made to look at the existing literature, a/k/a the Bove paper, to say what's your -- you know, what's your -- a reliable, you know, exposure measures.

Q. Okay. Well, I think let's stay focused on this.

Item 20 comes from ATSDR's 2018

1  
2 morbidity study, right?

3 A. Yes.

4 Q. Okay.

5 Are you able to compare Mr.  
6 Fancher's exposures described in Dr.  
7 Reynolds's report to the ppb month  
8 exposures described in the morbidity  
9 study?

10 MS. SULPIZIO: Object to the  
11 form.

12 A. I mean, I think you can compare  
13 all of these things. They're just --  
14 they're just not an exact, you know,  
15 conversion, but you can compare. I  
16 believe that Mr. Fancher's exposures are  
17 in line with these exposures, which is  
18 what I delineated in this.

19 But I just -- I can't -- I can't  
20 give an exact number. I can't give an  
21 exact number. I can't calculate it.

22 Q. Okay.

23 So, is your opinion that Mr.  
24 Fancher's TCE exposures as described by  
25 Dr. Reynolds are consistent with item 20

1  
2 cumulative exposure greater than 11,030  
3 ppb months of TCE?

4 MS. SULPIZIO: Object to the  
5 form.

6 A. I think they can -- I think  
7 they're -- I think they can be compared.  
8 I think that these levels are, you know,  
9 based upon her calculations, are in the  
10 realm of -- of being similar values.

11 Q. And how do you determine that  
12 they're similar values?

13 A. Based upon -- you know, based  
14 upon review of the literature. Based upon  
15 reviewing the ATSDR. Based upon, you  
16 know, her statement in -- in formulating  
17 these. Putting all them together.

18 Q. Okay.

19 So, Dr. Reynolds describes Mr.  
20 Fancher's TCE exposure as 5,340 micrograms  
21 per liter month, right?

22 A. For TCE?

23 Q. For TCE, yes.

24 A. Yes.

25 Q. Can you walk me through how you

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compare that to the cumulative exposure greater than 11,030 ppb months of TCE in item 20 of your Fancher report?

MS. SULPIZIO: Object to the form.

A. I can't. I can't cal -- I mean, I can't do a calculation here. I don't -- I'm not sure there is a calculation that can be done to convert those.

What -- what I'm -- you know, what I'm utilizing is her calculation, which is believed to be a, you know, an underrepresentation of what the exposure level was and then trying to compare it to the existing literature on parts per billion.

Q. Okay.

But can you walk me through how you make that comparison to this 11,030 ppb month of TCE in item 20 of your Fancher report?

MS. SULPIZIO: Object to the form.

A. No, I can't.

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Q. Can you explain how you would compare Mr. Fancher's PCE exposure as calculated by Dr. Reynolds to item 21 cumulative exposure greater than 711 ppb months of PCE?

MS. SULPIZIO: Object to the form.

A. Ask the question again?

Q. Sure.  
Similar to TCE, Dr. Reynolds also calculates Mr. Fancher's PCE exposure, correct?

A. Correct.

Q. All right.  
And she expresses that PCE exposure in micrograms per liter month, correct?

A. Correct.

Q. And she determines that Mr. Fancher's cumulative exposure to PCE was 247 micrograms per liter month, correct?

A. Correct.

Q. All right.  
And 247 micrograms per liter

1  
2 month is the exposure that you rely on for  
3 your report.

4 Is that also correct?

5 A. That's correct.

6 Q. Okay.

7 Can you walk me through how you  
8 would compare that 247 micrograms per  
9 liter month PCE exposure to item 21 in  
10 your Fancher report, cumulative exposure  
11 greater than 711 ppb months of PCE?

12 A. I mean, really I'm relying on --  
13 MS. SULPIZIO: Object to the  
14 form.

15 A. Really I'm relying on the  
16 experts to develop these numbers, Dr.  
17 Reynolds, and then I'm comparing these  
18 numbers to the existing literature, but in  
19 particular for -- for TCE, PCE, VC, I'm  
20 really -- I'm really comparing it to the  
21 low, moderate, and high risk groups from  
22 the -- from the Bove paper because that  
23 allows me to compare apples to apples,  
24 because, you know.

25 Q. Okay.

1  
2           So would it be fair to say  
3       you're not really comparing Dr. Reynolds's  
4       calculations to the ATSDR morbidity study?

5           MS. SULPIZIO: Object to the  
6       form.

7           A.       I wouldn't say that's fair to  
8       say. I mean, I still -- I'm still able to  
9       compare it, or at least, you know, take it  
10      into -- into -- into thought, but -- but  
11      it's much more challenging to compare the  
12      two. I mean, you're not -- but I'm still  
13      using it to compare.

14          Q.       Okay.

15                 And, I'm sorry, I'm still a  
16      little unclear on this. How do you make  
17      that comparison between 247 micrograms per  
18      liter month to 711 ppb months?

19          A.       I rely on the experts that  
20      calculate, you know, this. I -- I rely on  
21      the -- the -- the expert causation  
22      statements as to exposure, but most of my  
23      comparison for these values that were --  
24      that were developed by Dr. Reynolds, I'm  
25      really utilizing to put into a low,

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moderate, high risk category to compare to the Bove paper.

Q. Okay.

Looking at item 16, you would agree that Mr. Fancher did not reside on base more than 18 months, right?

MS. SULPIZIO: Object to the form.

A. Correct.

Q. All right.

And you would also agree that he was not employed on base for more than 2.5 years?

A. Correct.

Q. And looking at item 23 on the next page, Mr. Fancher did not spend more than 21 quarters on base as a civilian worker, right?

A. Correct.

Q. Can you turn to page 9 of your report?

A. Okay.

Q. Do you see the paragraph starting "According to ATSDR" at the



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bottom of that page?

A. I do.

Q. Okay.

And do you see the third sentence after footnote 5?

A. Okay.

Q. And you wrote. According to ATSDR/Bove, it is likely that during training, the water supplied in the field came from the Hadnot Point Water System with both measured and estimated levels of TCE and PCE substantially higher than their MCLs.

Did I read that correctly?

A. Yes.

Q. Okay.

What is your understanding of what an MCL is?

A. It's been a while since I put this together, but -- I think -- I'll be honest, I don't recall. I think it's basically just in comparison to, you know, a water system that wasn't the Hadnot system, would be like an expected

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exposure.

Q. Okay.

So your recollection is this is not comparing to the levels at which we would expect to see health effects.

Is that fair to say?

MS. SULPIZIO: Object to the form.

A. I -- I'll be honest with you, I don't -- I don't recall. I don't recall.

Q. Okay.

And how do you define whether estimated levels are substantially higher?

A. I mean, substantially higher would mean that, you know, it's having a -- a significant, you know, effect or it's -- it's a subjective term "substantially."

Q. Are there any articles or guidelines you consulted to determine whether a level of contamination is substantially higher?

A. No.

MS. SULPIZIO: Nathan, when

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you're at a point, can we take maybe  
five minutes?

MR. BU: Sure. Give me just one  
more question.

MS. SULPIZIO: That's fine.

BY MR. BU:

Q. Are you able to express the  
probability of cancer risk for exposures  
at a given level?

MS. SULPIZIO: Object to the  
form.

A. No.

Q. And do you know what the MCLs  
for TCE and PCE are?

A. I mean, I'm sure I did, but I  
don't right now.

MR. BU: All right. We can stop  
there.

THE VIDEOGRAPHER: The time  
right now is 2:17 p.m., and we are off  
the record.

(Recess taken.)

THE VIDEOGRAPHER: The time  
right now is 2:33 p.m., and we're back

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on the record.

BY MR. BU:

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

A. I have not.

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

A. I don't think so.

Q. Okay.  
Do you know whether people are exposed to background levels of TCE in everyday life?

MS. SULPIZIO: Object to the form.

A. I mean, I don't know that. I would have no way of knowing it.

Q. Okay.  
Would you agree that people generally are exposed to background level of carcinogens?

A. Yes.

Q. And it's impossible to live a

1  
2 life completely free from all cancerogenic  
3 exposures.

4 Is that fair to say?

5 A. Yes.

6 Q. Did you consider whether Mr.  
7 Fancher was exposed to background levels  
8 of TCE in your report?

9 A. I mean, I -- I think in the  
10 differential I -- I thought about lots of  
11 things for Mr. Fancher and what he may or  
12 may not have been exposed to.

13 The reality is is that there's  
14 no way for me to know what he was exposed  
15 to other than the fact that I know he was  
16 exposed to TCE, PCE, vinyl chloride,  
17 benzene. That's all I can know.

18 Q. So you don't make any comparison  
19 to what you know about Mr. Fancher's TCE  
20 exposures to what might be reasonable  
21 background levels of TCE, do you?

22 MS. SULPIZIO: Object to the  
23 form.

24 A. I do not.

25 Q. Okay.

1  
2 Do you offer any opinions about  
3 why the exposure to TCE in Camp Lejeune  
4 water would be the cause of Mr. Fancher's  
5 cancer but not exposure to background  
6 levels of TCE?

7 MS. SULPIZIO: Object to the  
8 form.

9 A. I mean, I don't -- I would have  
10 no way of knowing what type of, you know,  
11 low level carcinogenic exposures anyone  
12 could have, let alone Mr. Fancher. All I  
13 have is known documented exposure to these  
14 volatile compounds. So that's all I could  
15 really pass judgment on or -- or measure.

16 You know, obviously in -- in  
17 developing a differential, I put forth all  
18 of the, you know, known causes of kidney  
19 cancers. For instance, one of those is  
20 exposure to heavy metals like cadmium, and  
21 we don't have any -- any data based upon  
22 his testimony or his deposition that he  
23 had had high exposure to -- to any of  
24 the -- you know, to that substance or any  
25 other carcinogen.

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Q. Okay.

Did you consider how frequently TCE is detected in water supplies or in groundwater?

MS. SULPIZIO: Object to the form.

A. I didn't. I -- I -- I think I recall reading somewhere about a very, very low level being in, you know, in other water supplies, but I -- and again, I don't remember the numbers, but I remember thinking they were much, much, much smaller than what was seen in the Hadnot Water Plant.

Q. Okay.

Did you consider whether TCE occurs frequently at -- in ambient air?

A. I did not.

Q. Okay. All right.

MR. BU: Sharon, can we grab the next exhibit, the TCE tox profile?

(Weiss Exhibit 18, ATSDR Toxicological Profile For Trichloroethylene June 2019, was

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

4                   THE WITNESS:   Okay.

5       BY MR. BU:

6           Q.       Have you seen -- I've just  
7       handed you what's been marked Exhibit 18.

8                   Have you seen this document  
9       before?

10          A.       I -- I don't remember reading  
11       this document. It doesn't mean I haven't  
12       seen it before. Again, I reviewed a lot  
13       of documents, in particularly in the  
14       beginning when I was, you know, first  
15       started reviewing this case, but I -- I  
16       don't recall this one in particular. It  
17       doesn't mean I didn't review -- that I  
18       didn't read it.

19          Q.       Okay.

20          A.       To be honest.

21          Q.       Okay.

22                   Does it appear to be a  
23       toxicological profile on TCE by the ATSDR?

24          A.       It does.

25          Q.       Okay.



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And you reviewed and relied upon other ATSDR publications for your report, right?

A. Yeah. I mean, I relied on multiple ATSDR, you know, documents.

Q. Can you turn to page 322 for me, please?

A. Okay.

Q. And do you see the section heading "6.4.1 Air"?

A. I do.

Q. All right.

The ATSDR reports that trichloroethylene is widely detected in ambient air, correct?

MS. SULPIZIO: Object to the form.

A. That's what it says here.

Q. All right.

Do you have any reason to disagree with that statement?

A. No.

Q. All right.

Do you know whether TCE is also

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detected in food?

A. I do not know.

Q. Okay.

Would it surprise you to find that TCE is detected in low levels in different food products?

MS. SULPIZIO: Object to the form.

A. I mean, it wouldn't surprise me that TCE is found in ambient air or in food products, but the reality is is that everyone is inhaling ambient air, and everyone is being exposed to food products. So -- so you would think in these comparison groups and in cohort studies, et cetera, that everyone would have that same level of exposure, at least at baseline.

And then in this case, you're looking at, you know, additional documented increased levels of exposure. But it wouldn't surprise me if there's some level of TCE in the air or in food.

Q. Okay.

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Can you turn to page 333, Table  
6-8?

ATSDR reports various ppb TCE  
contamination in different food products,  
right?

A. Yes.

Q. Okay.

And for your report, you  
didn't -- did not consider whether Mr.  
Fancher may have been exposed to TCE  
through these different types of food  
products, did you?

MS. SULPIZIO: Object to the  
form.

A. I mean, I did not -- I did -- I  
did not take into account him having any  
type of, you know, different exposures  
outside of the camp that the general  
population wouldn't have. So I didn't  
take into -- you know, how many slices of  
American cheese does he eat or how many,  
you know, nuts does he eat as compared to  
the general population.

I would assume that his

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low-lying ambient exposure would -- would be the same as the general population with the addition of his time on Camp Lejeune.

Q. And you don't try to quantify what that ambient exposure would be for the general population; is that correct?

A. I do not.

Q. Okay.  
Can you turn to page 335 for me, please?

A. Okay.

Q. Do you see the middle paragraph starting "Assuming a typical air concentration"?

A. Okay.

Q. And it reads: Assuming a typical air concentration range of 100 to 500 ppt and a breathing rate of 20 cubic meters air per day, the average daily air intake of trichloroethylene can be estimated at 11 to 33 micrograms per day.

Did I read that correctly?

A. You did.

Q. And a ppt is a part per

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trillion, right?

A. Yes.

Q. And ATSDR goes on to report that: Average daily water intake of trichloroethylene can be estimated at 2 to 20 micrograms per day assuming a typical concentration range of 2 to 7 ppb in consumption of two liters water per day.

Did I read that correctly?

MS. SULPIZIO: Object to the form.

A. You read it correctly, yes.

Q. Do you have any reason to disagree that the average daily air intake of trichloroethylene can be estimated at 11 to 33 micrograms per day?

MS. SULPIZIO: Object to the form.

A. I mean, I -- I truthfully have no way of knowing. I mean, it's -- it's in this report. I have no reason to believe it's wrong or -- or right. I mean, I -- I have no -- no basis for passing judgment on that.

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Q. Okay.

Do you have any reason to disagree that the average daily water intake of TCE can be estimated at 2 to 20 micrograms per day?

A. I have no reason to disagree, and I have no reason to agree.

Q. Do you know whether benzene is commonly found in ambient air?

A. I don't, but my guess is 'cause you're asking that it will be.

Q. And do you know whether benzene is also found in foods?

A. Yeah, benzene I -- I -- I do believe is found in some foods, yes.

Q. Okay.

And do you know if benzene is found in gasoline products?

A. I believe it is, but I can't say definitively.

Q. Okay.

MR. BU: Sharon, can we pull up the next exhibit, please?

(Weiss Exhibit 19, U.S.

1  
2 Department of Health and Human  
3 Services Toxicological Profile For  
4 Benzene August 2007, was marked for  
5 identification, as of this date.)

6 THE WITNESS: I will say that  
7 the more I'm seeing these, the more  
8 that I'm realizing that the estimate  
9 of Mr. Fancher's exposure is probably  
10 even more of an underestimate because  
11 he, in addition to having the time on  
12 the base, would have the same exposure  
13 as the general population or people  
14 walking around and eating or drinking.  
15 I don't think his exposure, you  
16 know...

17 MR. BU: Okay.

18 BY MR. BU:

19 Q. Do you know that general  
20 population exposures are comparable to the  
21 Camp Lejeune water exposures?

22 MS. SULPIZIO: Object to the  
23 form.

24 A. I would have no way of knowing.

25 Q. Okay.

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So you would have no way of knowing how much the Camp Lejeune water exposures underestimate Mr. Fancher's total exposure?

MS. SULPIZIO: Object to the form.

A. I -- I'm sorry, I don't -- explain the question again.

Q. Right.

A. Say the question again.

Q. Sure.

Earlier you had said that the estimated Camp Lejeune exposures are probably an underestimate given Mr. Fancher's, sort of, general population exposures, correct?

A. Yes.

Q. Okay.

What I'm asking you is do you know how significant that underestimate would be? Is it double? Is it triple? Is it 25 percent?

A. No, I don't know.

MS. SULPIZIO: Object to the



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form.

BY MR. BU:

Q. All right.

Can you turn to page 272 of  
Exhibit 19?

A. Okay.

Q. And, I'm sorry, I should have  
mentioned, Exhibit 19, have you seen this  
document before?

A. I have not.

Q. Okay. So, this is a  
toxicological profile for benzene by the  
ATSDR.

And on page 272, table 6-3 ATSDR  
reports different benzene concentrations  
in food.

Is that right?

A. Yes, that's what it appears.

Q. Okay.

And do you have any reason to  
disagree with the concentrations reported  
here in Table 6-3?

A. Truthfully, I would have no way  
of knowing whether this is true or not.

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2 But I have no reason to disagree with it,  
3 but I have no reason to agree with it  
4 either.

5 Q. Okay.

6 A. I -- and I just -- I just want  
7 to say that in particular for these  
8 documents, like the toxicology profile for  
9 benzene, I haven't had a chance -- I  
10 haven't seen this before. I haven't had a  
11 chance to review it in totality, so it's a  
12 little bit difficult for me to comment on  
13 it.

14 Q. Okay.

15 A. Like for this Table 6-3, I -- I  
16 haven't had a chance to analyze, like,  
17 what is this they're listing here, number  
18 of cases. Like did -- was there only two  
19 total cases with cheese that had benzene  
20 in it?

21 So I -- I really struggle with  
22 this.

23 Q. Okay.

24 For Mr. Fancher, you didn't  
25 review any chemical tests to determine

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whether his cancer was caused by a toxic exposure, did you?

A. I --

MS. SULPIZIO: Object to the form.

A. No, not that I'm -- no.

Q. Are you aware of any chemical tests or biomarkers for TCE-induced kidney cancer?

A. I'm not aware of any like clinically -- clinical test or any -- any lab test. I -- I would assume there's -- you know, when the exposure's occurring that you may be able to measure, like, you know, metabolites, but I'm unaware of any that being available or done in this case or any cases, to be honest.

Q. Are there clinical features that are more consistent with a chemically-induced kidney cancer?

A. Not that I'm aware of.

Q. Outside of litigation, have you ever diagnosed a patient with chemically-induced cancer?

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A. Yes, I believe I have.

Q. Okay.

In what context?

A. Currently in New York we're seeing a lot of patients that are developing malignancies that are related to 9/11, and some of those chemical exposures range from just aerosolization of, you know, asbestos, they range from heavy metals from when the towers came down, cadmium actually. So -- so I do see patients that frequently are part of that situation. I'm not involved in any way in that litigation or the lawsuit or if there even is a lawsuit related, but I see patients frequently through the 9/11 group that's being sent to me for cancers that are felt to be 9/11-related.

Q. Okay.

Are you referring to the World Trade Center Health Program?

A. Yes.

Q. Okay.

And so you treat patients that

1  
2 are referred to through the World Trade  
3 Center Health Program; is that right?

4 A. I -- I see patients that are  
5 part of that program because frequently as  
6 part of that program, we -- you know,  
7 they -- they require medical records and  
8 documentation about their cancer  
9 diagnosis. So -- so I see patients that  
10 are part of that. They're not being  
11 directly referred to me through that  
12 program.

13 Q. Okay.

14 When they come to you, has  
15 someone else already made a determination  
16 that they're eligible for medical care  
17 through the health program, or are you  
18 making that determination?

19 A. It depends. They're not --  
20 they're not always already part of that  
21 program, but frequently when they have the  
22 diagnosis, then -- and again, I don't -- I  
23 don't put them into the program, but --  
24 but they take the medical records to see  
25 if they're applicable for that program and

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they should be accepted, so.

I don't make that determination.

Q. Do you know whether that program applies any presumptions to determine whether an exposure caused cancer?

A. I don't know.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Do you know how the World Trade Center Health Program determines whether a cancer's caused by exposure to 9/11?

MS. SULPIZIO: Object to the form.

A. I do not.

Q. You mentioned earlier that Mr. Fancher's diagnosis was much younger than is typical.

Do you recall that?

A. I do.

Q. Okay. What is a -- what is the typical age range for a kidney cancer diagnosis?

A. I mean, the -- the vast majority

1  
2 of kidney cancers are diagnosed in people  
3 in their sixth and seventh decades of  
4 life, but, you know, when you look at the  
5 ultra extremes, you know, less than 40 is  
6 an extreme. I would say less than 5  
7 percent of kidney cancers are -- are  
8 diagnosed in people under the age of 40.

9 Q. On page 9 of your report, you  
10 also describe the latency period for Mr.  
11 Fancher's cancer. This is the third full  
12 sentence in Section 8.

13 A. I'm sorry, page 5?

14 Q. Page 9.

15 A. Page 9.

16 Q. Yeah.

17 A. Okay.

18 Q. And you write that: The latency  
19 period between exposure and the  
20 development of cancer is normal.

21 Do you see that?

22 A. I'm not going to find it here,  
23 but I -- but I -- yes. I mean, I wrote  
24 that and I know that, so.

25 It's very common to have a

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latency period from the exposure to when the cancer actually develops.

Q. And Mr. Fancher's latency period was 16 years, correct?

A. That's correct.

Q. What is a normal latency period between exposure and kidney cancer?

MS. SULPIZIO: Object to the form.

A. I mean, I think it -- I think it's dependent -- I don't think we know with a hundred percent certainty. It's probably dependent on what the exposure is to whether it be TCE, PCE or whether it's to a different carcinogen like smoking, but many cancers related to, you know, to -- to environmental exposures have a latency period of 10, 15, 20, sometimes even longer years. Probably that exposure risk probably continues for the life of the individual that was exposed. So -- so I don't think we -- you know, I don't think we can say the, quote, normal latency. It's frequently 10, 15, 20



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years.

Q. Okay.

So then would you say a cancer that is diagnosed less than 10 years or longer than 20 years after an exposure is less likely to be caused by that exposure?

A. No.

Q. Okay.

Is a cancer that is diagnosed between 10 and 20 years following exposure more likely to be caused by that exposure?

MS. SULPIZIO: Object to the form.

A. Not necessarily.

Q. And why is latency not necessarily indicative of causation?

A. I'm not sure I understand the question.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Right.

So -- so, in your report you make a comment, right, that Mr. Fancher's

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latency was normal, right?

A. Yeah, that it was normal to have a latency between an exposure and when the cancer develops. That's very common.

Q. Okay.  
Can you turn to page 12 of your report for me, please?

A. Okay.

Q. The last sentence of this section before Section 10 you write:  
After exposure to carcinogenic chemicals, there is well-established latency period before the actual development of cancer and 16 years is well within the normal range.

Do you see that?

A. I do.

Q. All right. And so I had asked you what is a normal range.

Do you recall that?

A. I do.

Q. All right.  
And so would a normal range be between 10 and 20 years?

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A. So, I think -- I think I misunderstood your question before because I thought you meant just what's normal range for a latency for cancer -- I thought you were specifically saying for kidney cancer.

This is referring to a latency period for cancerogenic exposure for a cancer development in general, and -- and we have a lot more experience in things like lung cancer, frequently, you know, 10, 15, 20 years. I don't think -- it's not like a confidence interval. We can't say 10 to 20 years. But 16 years is -- is within a, like, a normal range for how latency periods work for cancer development.

Q. Okay.

A. My point is it's not uncommon for it to take 16 years from the exposure to develop a -- to develop a cancer.

Q. All right.

Your understanding of a normal range for latency is based on what we know

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about smoking and latency.

Are there other latencies that we know about that you've considered?

MS. SULPIZIO: Object to the form.

A. I mean, there's lots of latency periods that we know about. We know about latency periods for skin cancer, you know, frequently 15, 20 years after a high-intensity skin -- skin damage or skin burning.

I'm just trying to think off the top of my head.

You know, chronic pancreatitis, people can have chronic pancreatitis for 10, 15 years and then they develop pancreas cancer. There's been a nidus, you know, 15 years after the development. So -- so many, many cancerous will develop 15, 20 years after the -- the exposure or the nidus or what we believe is the cause.

Q. And just so as there are latencies, sort of, within this normal range, there must be latencies outside of

1  
2 that normal range, right?

3 MS. SULPIZIO: Object to the  
4 form.

5 A. Yeah, I'm sure there are.

6 Q. Okay.

7 So to go back to what I was  
8 trying to get at earlier, if a latency is  
9 outside of that normal range, whatever it  
10 is, would you agree it's less likely to be  
11 caused by the exposure?

12 MS. SULPIZIO: Object to the  
13 form.

14 A. Not necessarily. Not  
15 necessarily. I think, you know, different  
16 people have, you know, different  
17 thresholds. Different people may have  
18 longer or shorter latencies based upon,  
19 you know, their own susceptibility to  
20 cancer. And so I can't say that if you're  
21 outside of the normal range that now it's  
22 less likely to be related to that  
23 exposure.

24 I -- my comment is just that  
25 it's pretty normal to have a -- a latency

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period of 16 years like -- like Mr.  
Fancher had.

Q. Okay.

Do you know whether the latency  
periods vary by different types of cancer?

A. Yeah, I mean, there's, yeah,  
different cancers, different environmental  
exposures may have a different, you know,  
latency period. Like, you know, when they  
dropped an atomic bomb in Japan, those  
patients developed cancer pretty quick  
when they were exposed to high intensity  
iodizing radiation and different types of  
cancer. So, yeah, I think there's  
probably a range.

Q. You also mentioned you don't  
think Mr. Fancher's cancer's related to a  
genetic predisposition.

Do you recall that?

A. I do recall it.

Q. Okay.

All else being equal, Mr.  
Fancher's young age of diagnosis would be  
considered suspicious for a genetic

1  
2 predisposition.

3 Is that fair to say?

4 A. I wouldn't say that it's  
5 suspicious. I mean, you know, when you  
6 have a young patient that develops a  
7 cancer, you're -- you're obviously trying  
8 to figure out why.

9 You know, a familial  
10 predisposition usually has a family  
11 history of such cancers, like kidney  
12 cancers. In a -- in a young patient,  
13 you're usually looking for did they  
14 develop a cancer in both kidneys or just  
15 one kidney, which wasn't him. You're  
16 looking for was it multiple tumors in  
17 the -- in the same kidney or was it one.  
18 He had a solitary lesion.

19 Just age in of itself I don't  
20 think would push you are towards saying  
21 this is a genetic predisposition for this  
22 individual. He really had no other risk  
23 factors.

24 Q. Okay.

25 In your report on page 11

1  
2 towards the bottom of that paragraph --  
3 or, bottom of that page, I'm sorry, you  
4 write: His history would not warrant  
5 genetic testing under the current standard  
6 of care due to the extreme low likelihood  
7 of revealing a genetic effect.

8 Do you see that?

9 A. I do.

10 Q. Okay.

11 Were there guidelines or  
12 resources you consulted for determining  
13 the current standard of care for genetic  
14 testing?

15 A. No. I mean, there -- there may  
16 be guidelines, but I didn't consult any of  
17 the guidelines. I looked at his -- I  
18 looked at his medical history, his family  
19 history, his presentation and what the  
20 disease looked like at presentation, and I  
21 based -- and -- and I based the opinion  
22 that -- that it -- you know, there was no  
23 clear indication that there -- there was  
24 no indication that this was genetically  
25 caused.



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Q. Okay.

Can you turn to page 13 of your report for me, please?

A. Okay.

Q. So, you opine that the treatment and care Mr. Fancher has received and is now receiving is fair, reasonable, and medically necessary, right?

A. I agree.

Q. Okay.

What treatment and care are you referring to?

A. Well, the original treatment of, obviously, undergoing the nephrectomy, the radical nephrectomy.

Can you rephrase the question? Do you want to know what he's undergoing currently or whether -- what he underwent?

Q. Is there treatment Mr. Fancher is currently receiving that's related to his kidney cancer?

A. I don't know if he's still being followed. You know, clearly he is now 20 -- more than 25 -- yeah, more than 25

1  
2 years out. So the chances of recurrence  
3 are very low, but they're still possible.  
4 So, many times these patients are followed  
5 for life for a potential recurrence.

6 I would assume that he's being  
7 followed for the fact that he has one  
8 kidney, so he's at least at risk for  
9 developing renal insufficiency, liver --  
10 you know, renal failure in the opposing  
11 kidney. So far at least, based upon the  
12 most recent records, his -- his remaining  
13 kidney is functioning, but I got to  
14 believe that he's being followed for that.

15 I'm unaware of any other, you  
16 know, treatment that he's receiving in  
17 terms of mental health and so forth.  
18 Obviously patients that are diagnosed with  
19 a cancer, it's a -- it's a dramatic event  
20 for them. They're told they have a  
21 cancer, then they're undergoing  
22 surveillance, and, you know, sometimes  
23 they have long-term sequelae related to  
24 that. And -- and I'm not aware of  
25 treatment that he's receiving for that,

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but it's certainly possible.

Q. Is there any other treatment or care that you are aware of that Mr. Fancher is receiving surveillance?

A. No.

Q. Okay.

A. I believe he has a hernia or at least a diastasis or an outpouching from where he had the incision 'cause I know he had a wound infection after surgery and frequently those patients get hernias. I don't think he's undergone any interventions or treatment for that, but they can sometimes be somewhat, you know, lifestyle-altering having that.

Q. Other than the hernia and the kidney removal, are there any other permanent effects that you attribute to Mr. Fancher's kidney cancer diagnosis?

A. I mean, the tough thing for these patients that have been treated for cancer, truthfully, is some of the, you know, lifelong anxiety of is it going to come back and the -- the mental health,

1  
2 you know. Some of these patients, you  
3 know, even can get, sort of like,  
4 post-traumatic distress disorder for being  
5 treated for their cancers.

6 But my understanding is -- is,  
7 you know, his tumor was removed. It was a  
8 T3N0 tumor. He has not had a recurrence  
9 of that cancer yet. Although, you know,  
10 again the likelihood is low, but it still  
11 could happen. I've seen recurrences of  
12 kidney cancers this far out. It happens.  
13 Pretty uncommon.

14 And then I would assume routine  
15 health as part of that related to the fact  
16 that he has one kidney and needs to be  
17 followed for that and managed  
18 appropriately, and it seems -- seems like  
19 he's, you know, in relatively decent  
20 shape.

21 Q. Okay.

22 Do you know whether the standard  
23 of care would be that Mr. Fancher requires  
24 cancer surveillance?

25 A. So, the standard of care would

1  
2 say that he could -- that he could have  
3 stopped surveillance, but I personally  
4 have experience with patients that have  
5 recurred long after the standard of care.

6 So, standard of care I would say  
7 at this point he probably does not need to  
8 undergo surveillance anymore.

9 Q. Okay.

10 A. But that doesn't mean that he  
11 can't recur at this point.

12 Q. When you were discussing Mr.  
13 Fancher's hernia, is that the same or  
14 different from his surgical scar?

15 A. It's different.

16 Q. Okay.

17 Do you know whether Mr.  
18 Fancher's surgical scar requires any  
19 interventions?

20 A. Currently I don't -- I don't  
21 believe it has.

22 Q. Okay.

23 On page 13 you also opine for  
24 item 4: The medical billing relating to  
25 Mr. Fancher's kidney cancer diagnosis, the

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surgery to remove his kidney, and the follow-up treatment related to his kidney cancer was reasonable and medically necessary.

Do you see that?

A. Yes.

Q. Okay.

How did you determine whether medical billing is reasonable?

A. I -- I didn't review actual billing for this, but the billing of performing a -- you know, a -- a radical right nephrectomy and the aftercare and everything else would be appropriate. You -- you would bill for those services in someone that has cancer. But I certainly didn't review, like, the actual bills and make sure that the bills were the proper amounts and so forth.

Q. Okay.

So you're not opining to the specific costs for medical services.

Is that fair to say?

MS. SULPIZIO: Object to the

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form.

A. I mean, I didn't review the actual costs of his procedures.

Q. Okay.

When did you first become aware of the Camp Lejeune water litigation?

A. I think the first I became aware of it was -- was -- was when I was contacted by Mr. Mandell about, you know, would I be willing to review a case to see if, you know, I wanted to be a -- a witness in the case.

In hindsight maybe I saw some -- you know, on TV maybe a commercial here or there, but to be honest, there's so many of these commercials that I don't remember the specifics. But I think the first I really became aware of the case was -- was last year when I was contacted by Mr. Mandell, you know.

Q. When you say "Mr. Mandell," do you know if that was Mark Mandell or Zach Mandell?

A. It was Zach Mandell.

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Q. When was this that Zach Mandell reached out to you?

A. Like the -- the middle of 2024, I want to say back in maybe September or August, around that time. I can't give you the exact date, but I think that's when he first reached out to me, I believe.

Q. At that initial meeting in -- whenever you had your first contact with Zach Mandell, do you recall how the Camp Lejeune water litigation was described?

A. I don't.

MS. SULPIZIO: Object to the form.

To the extent that it has any questions in regard to, you know, privilege, I instruct you not to answer.

BY MR. BU:

Q. Do you recall when you were retained by plaintiffs' counsel in this litigation?

A. When I was contacted, I was



1  
2 essentially retained. I don't do a lot  
3 of, you know, witness testimony and -- and  
4 basically when I do, I -- if I'm retained,  
5 that's to review the -- the materials. It  
6 was probably -- it -- it must have been  
7 around the same time, September.

8 Q. When you say "retained to review  
9 materials," do you mean as like a  
10 consulting expert or a medical consultant?

11 A. I'm not sure I understand the  
12 difference --

13 MS. SULPIZIO: Object to the  
14 form.

15 A. -- to be honest.

16 Q. Sure. Let me ask it this way.  
17 How much of your annual income  
18 would you say is related to assisting in  
19 legal proceedings?

20 A. Very little. I could calculate  
21 it, I guess, but it's probably far under  
22 5 percent. Maybe 5 percent.

23 Q. And of the legal proceedings  
24 that you've assisted in, I think you  
25 mentioned you testified in maybe six; is

1  
2 that right?

3 A. I've been deposed about five or  
4 six times. I maybe will do, you know,  
5 either a medical consulting or a legal  
6 case maybe -- maybe once or -- or twice a  
7 year, tops. It doesn't represent very  
8 much of my efforts in my income.

9 Q. Okay.  
10 When you were retained by  
11 plaintiffs' counsel, did you execute a  
12 retainer agreement or some other type of  
13 contract?

14 MS. SULPIZIO: Object to the  
15 form.

16 A. If I -- if I'm involved in a  
17 case like this, I have a standard retainer  
18 sheet just that describes what, you know,  
19 the hourly rate will be and so forth.

20 And so, I'll be honest, I don't  
21 understand the -- I should, I guess. I  
22 don't understand the legal back-and-forth  
23 of what I was, quote, retained versus I  
24 just -- you know, I give them a --  
25 basically a fee schedule so that they know

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what I charge and then that's it.

MR. BU: Okay.

Sharon, can we pull up tab 27,  
please?

(Weiss Exhibit 20, invoices and  
fee schedule of Matthew J. Weiss, MD,  
MBA, Bates  
CL\_PLG-EXPERT\_WEISS\_0000000001-006,  
was marked for identification, as of  
this date.)

BY MR. BU:

Q. I'm handing you what's been  
marked Exhibit 21.

A. Okay.

Q. Have you seen this document  
before?

A. I have.

Q. And what is this document?

A. This is a copy of an invoice  
that I sent to the Mandells on November of  
2024.

Q. Okay.

And if you flip to, I guess, the  
next few pages, there's also a fee

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schedule for deposition and for court appearances.

Do you see that?

A. Correct, yes.

Q. Okay.

Is this the retainer sheet that you were describing earlier?

A. This is.

Q. Okay.

Do you know whether there are any other agreements that you signed with plaintiffs' counsel?

MS. SULPIZIO: Object to the form.

A. Not that I'm aware of.

Q. Okay.

And you drafted this retainer agreement; is that correct?

A. This was drafted by me.

MS. SULPIZIO: Object to the form.

BY MR. BU:

Q. Okay.

Do you know whether you would

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have done any work on this case before  
September 25th, 2024?

A. I don't recall the exact dates,  
to be honest. I don't remember. I -- I  
don't think so.

Oh, wait now. Here. Here,  
'cause I just -- yeah, September 25th,  
2024, that would have been the first day  
that I worked on it, correct.

Q. Okay.  
Is there work in this litigation  
that you performed but did not bill for?

MS. SULPIZIO: Object to the  
form.

A. I don't think so.

Q. Okay.  
So you wouldn't have, like,  
written off any time?

MS. SULPIZIO: Object to form.

A. No, I don't think so.

Q. Okay.  
Is there any compensation you  
expect to receive other than your hourly  
rate for casework, testimony fees, and

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travel costs?

A. No.

Q. Is your fee schedule on this case the same as your fee schedule used in other cases?

A. Exactly the same, yes.

Q. The second invoice is from February 5th, 2025.

Do you see that?

A. I do.

Q. Okay.

Have you submitted any invoices to plaintiffs' counsel since February 5th, 2025?

A. I have not.

Q. Have you performed work on this case since January 25th, 2025?

A. I have.

Q. All right.

About how much time have you spent on this case since January 25th, 2025?

A. I would say maybe five or six hours re-reviewing, 'cause obviously

1  
2 things that I reviewed back in September  
3 of 2024, in preparation for the deposition  
4 I wanted to go back and re-review. I  
5 would guess, I don't know, five or six  
6 hours.

7 Q. Did you speak with any other  
8 plaintiffs' experts in the course of  
9 preparing your reports in this case?

10 A. I did not.

11 Q. You mentioned spending five to  
12 six hours on depo prep.

13 Can you tell me what you did to  
14 prepare for your deposition today?

15 MS. SULPIZIO: Objection only to  
16 the extent that it reveals  
17 communications between him and  
18 counsel.

19 Go ahead, you may answer.

20 A. I went back and reviewed what I  
21 remember being the most critical papers  
22 related to this case. I went back and  
23 reviewed depositions by Mr. Fancher. I  
24 reviewed his medical record again. I  
25 reviewed some of the -- the expert, I

1  
2 wouldn't call it testimony, but the  
3 experts of -- of Dr. Reynolds, Dr. Hatten,  
4 Dr. Bird. Basically it was just reading  
5 through a lot of documents that I had read  
6 before, but I wanted to refresh my memory.

7 Q. Okay.

8 Without telling me what you may  
9 have discussed, did you meet with any  
10 attorneys to prepare for your deposition?

11 MS. SULPIZIO: Object to the  
12 form.

13 A. I did.

14 Q. Okay.

15 How many times did you meet with  
16 attorneys?

17 A. Two short meetings.

18 Q. How long were those meetings?

19 How long was the first meeting,  
20 I'm sorry?

21 A. Maybe a half an hour.

22 Q. Do you remember which attorneys  
23 you met with?

24 A. I guess the question is can I --  
25 can I say that?



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MS. SULPIZIO: You can answer.

A. Yeah, Mr. Mandell and -- and Gabby.

Q. Okay. So, there are two Mandells.

Do you know if you met with Zach or Mark?

A. Sorry. I met with Zach.

Q. How long was the second meeting with the attorneys?

A. About the same, about a half an hour. It was a -- it was -- it was really a phone call.

Q. And who did you meet with for that second meeting?

A. The same.

Q. Zach Mandell and Gabby?

A. Zach Mandell and Gabby.

Q. Okay.

Was anyone other than a plaintiffs' attorney present for either of those meetings?

A. No.

Q. Have you had communications with

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anyone other than a plaintiffs' attorney  
to prepare for your deposition?

A. No.

Q. Have you had any communications  
with Mr. Fancher?

A. No.

Q. Have you had any communications  
with any of the plaintiffs in the Camp  
Lejeune water litigation, that you're  
aware of?

A. No.

Q. Have you had any communications  
with any of Mr. Fancher's treating  
physicians?

A. No.

Q. The cases in which you've served  
as an expert witness, how many of those  
cases, other than this one, have involved  
exposures to toxic substances?

A. None of them.

Q. Were the other cases medical  
malpractice cases?

A. Yes.

Q. Other than this litigation, is

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all of your work in litigation related to  
medical malpractice?

MS. SULPIZIO: Object to the  
form.

A. I would say up -- yes. Yes.  
Yes.

Q. There are no other types of  
cases that you've worked on?

A. No.

Q. Prior to this case, have you  
ever worked as an expert witness for the  
Mandell law firm?

A. No, never before.

Q. Okay.

Have you ever worked as an  
expert witness for The Bell Legal Group?

A. No.

Q. To the best of your knowledge,  
have you ever worked as an expert witness  
for any of the other plaintiffs' firms  
involved in the Camp Lejeune water  
litigation?

A. I don't know what all the other  
plaintiffs' firms are, but I -- I can't

1  
2       imagine that I have.

3           Q.       Okay.

4                    Have you ever worked as an  
5       expert witness in a case involving the  
6       United States?

7           A.       No.

8                    MR. BU:    Could we go off record?

9                    THE VIDEOGRAPHER:   The time  
10       right now is 3:21 p.m., and we're off  
11       the record.

12                   (Recess taken.)

13                   THE VIDEOGRAPHER:   The time  
14       right now is 3:30 p.m., and we're back  
15       on the record.

16       BY MR. BU:

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

20           A.       I did not.

21           Q.       Okay.

22                    Is there anything that you've  
23       testified to today that you would like to  
24       clarify or correct?

25           A.       No.

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Q. Can you turn to page 12 of your report, Exhibit 1, for me, please?

A. Okay.

Q. In that first full paragraph you write: Mr. Fancher was also exposed to PCE, benzene, and VC, which are known carcinogens and --

A. Hold on. I'm having trouble finding you.

You said page 12 of my -- of my report, correct.

Q. Yes, page 12 of your report.

A. Okay.

Q. The first full paragraph at the top, the third line of that paragraph beginning "Mr. Fancher was also exposed."

A. Okay. Yes.

Q. So, you write: Mr. Fancher was also exposed to PCE, benzene, and VC, which are known carcinogens and also associated with the development of kidney cancer.

Did I read that correctly?

A. Yes.

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2 Q. And you go on to write: This  
3 exposure likely and probably would have  
4 been additive or synergistic when combined  
5 with his TCE exposure.

6 Did I read that correctly?

7 A. Yes.

8 Q. Can you explain what you mean by  
9 "additive or synergistic"?

10 A. What I mean is that he had --  
11 you know, if he has -- obviously has the  
12 TCE exposure which has its own  
13 carcinogenic, you know, potential, and  
14 that these other substances, PCE, benzene,  
15 and VC, also have a carcinogenic  
16 potential, and then since he was exposed  
17 to all four, he's -- each of them would  
18 have its own cancer potential. It's not  
19 like one would be protective over the  
20 other. So I would say that they're at  
21 least additive.

22 And it's even possible that some  
23 carcinogens when they're combined can have  
24 a synergistic effect, meaning they can  
25 make them even more potent than just being

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additive.

Q. Did you review any literature about how these different chemicals interact with one another?

A. I did not.

Q. Okay.  
Did you review any literature about how they are metabolized together?

A. Not together.

Q. Okay. Okay.  
Are there any additional materials you feel would be relevant to your report that you did not have a chance to review?

A. No.

Q. Okay.  
And do you -- do you consider yourself an epidemiologist?

A. No.

Q. All right.  
Have you ever been a principal invest -- investigator for an epidemiological study?

A. No.

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Q. Do you consider yourself a toxicologist?

A. No.

Q. Do you hold yourself out as an expert in environmental health?

A. No.

Q. Do you hold yourself out as an expert in occupational medicine?

A. No.

Q. Do you hold yourself out as an expert in risk assessment?

A. Just general risk assessment? I wouldn't say an expert. I mean, we utilize risk assessment daily life in -- in medical care and in reviewing the literature all the time, but I wouldn't say I'm an expert in -- in risk assessment, per se.

Q. Okay.

Have you ever published peer-reviewed literature regarding the effects of TCE on cancer?

A. I have not.

Q. Have you ever published



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peer-reviewed literature regarding the effects of PCE, benzene, or vinyl chloride on cancer?

A. I have not.

Q. To the best of your knowledge, have you ever treated a patient with cancer related to exposure to water at Camp Lejeune?

A. Not that I'm aware of.

Q. Okay.

Have you ever been asked to write a letter to the Department of Veterans Affairs related to benefits for Camp Lejeune exposures?

A. No.

Q. Does your practice require training on --

MR. BU: Let me rephrase that.

Sorry.

Q. Does your practice require training on the potential health risks related to TCE exposure?

MS. SULPIZIO: Object to the form.

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A. I mean, not specifically for TCE exposure, no.

Q. What about specifically for PCE, vinyl chloride, or benzene?

MS. SULPIZIO: Object to the form.

A. No. I mean, my practice requires knowing about environmental exposures and their -- and their of developing cancer, but I wouldn't say specifically these.

Q. Other than in this case, have you ever offered an expert opinion in another case involving exposure to TCE, PCE, vinyl chloride, or benzene?

A. No.

Q. Okay.

Other than in this case, have you ever offered an opinion about the etiology of a kidney cancer?

A. No.

Q. Other than in this case, have you ever offered an expert opinion on the etiology of a -- of any type of cancer?

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MS. SULPIZIO: Object to the  
form.

A. No, I don't believe so.

Q. Do you feel that your testimony  
that you've given today is complete and  
accurate to the best of your ability?

A. I do.

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

A. No.

MR. BU: Okay.

No further questions.

MS. SULPIZIO: I just have one  
quick question for you, Doctor.

EXAMINATION BY

MS. SULPIZIO:

Q. You were asked some questions  
today about prospective randomized trials,  
and you talked about the connections  
between them and causation.

Do you recall that conversation?

A. I do.

Q. Would you agree that you are

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able to prove causation in cases like Mr. Fancher without prospective randomized control trials?

A. Absolutely.

Q. Okay.

A. A prospective randomized trial for a situation like this would -- would be unethical, and the only way you can draw conclusions is by using the best available retrospective data to -- to formulate an opinion and a conclusion.

Q. And that's exactly what you did in your report to make your opinions for Mr. Fancher, correct?

A. Correct.

MS. SULPIZIO: I don't have any further questions.

THE VIDEOGRAPHER: The time right now is 3:37 p.m., and we're off the record.

MS. SULPIZIO: Doctor, you're going to read and sign, right?

THE WITNESS: Yes.

(Time noted: 3:37 p.m. EDT)

INSTRUCTIONS TO WITNESS

Please read your deposition over carefully and make any necessary corrections. You should state the reason in the appropriate space on the errata sheet for any corrections that are made.

After doing so, please sign the errata sheet and date it. It will be attached to your deposition.

It is imperative that you return the original errata sheet to the deposing attorney within thirty (30) days of receipt of the deposition transcript by you. If you fail to do so, the deposition transcript may be deemed to be accurate and may be used in court.

A C K N O W L E D G M E N T

STATE OF )  
: ss  
COUNTY OF )

I, MATTHEW J. WEISS, M.D., MBA,  
hereby certify that I have read the  
transcript of my testimony taken under  
oath in my deposition of June 30, 2025;  
that the transcript is a true and complete  
record of my testimony, and that the  
answers on the record as given by me are  
true and correct.

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

Signed and subscribed to before me this  
----- day of -----, 20\_\_.

-----  
Notary Public, State of



C E R T I F I C A T E

I, MARIE FOLEY, Registered Merit  
Reporter, Certified Realtime Reporter, and  
Notary Public for the State of New York,  
do hereby certify that prior to the  
commencement of the examination, MATTHEW  
J. WEISS, M.D., MBA, was duly remotely  
sworn by me to testify to the truth, the  
whole truth and nothing but the truth.

I DO FURTHER CERTIFY that the foregoing  
is a verbatim transcript of the testimony  
as taken stenographically by me at the time,  
place and on the date hereinbefore set forth,  
to the best of my ability.

I DO FURTHER CERTIFY that I am neither  
a relative nor employee nor attorney nor  
counsel of any of the parties to this action,  
and that I am neither a relative nor employee  
of such attorney or counsel, and that I am  
not financially interested in the action.

-----  
COURT REPORTER

Registered Merit Reporter  
Certified Realtime Reporter

Notary Public

Dated: July 9, 2025



LAWYER ' S    N O T E S

PAGE / LINE

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<b>&amp;</b>	<b>10</b> 4:6 8:13	<b>11:43</b> 141:22	<b>157</b> 8:12
<b>&amp;</b> 2:13	53:25 54:11	<b>11:55</b> 142:2	<b>15th</b> 23:10
<b>0</b>	93:8 94:12	<b>12</b> 8:20 147:23	<b>16</b> 9:13 175:10
<b>0000000001-...</b>	157:9,14	166:7,13	188:19,24
10:10 282:9	159:21,25	169:11 177:10	239:5 263:5
<b>00897</b> 1:6	160:4,9 173:11	265:7 292:2,11	265:15 266:15
<b>02903</b> 2:16	188:17 203:9	292:13	266:21 269:2
<b>05</b> 162:13	204:22 221:16	<b>12,250</b> 169:20	<b>164</b> 8:15
180:12 198:12	263:19,25	229:5,18 230:2	<b>166</b> 8:19
<b>1</b>	264:5,11	230:14	<b>167</b> 8:22
<b>1</b> 2:15 6:7 22:2	265:11,25	<b>12:55</b> 200:24	<b>17</b> 9:16 107:4
22:2,3 28:23	266:13,15	<b>13</b> 4:9 7:23 8:23	179:16 225:13
39:12 57:21,25	267:17	130:12 132:22	225:19
87:15 143:8	<b>100</b> 70:19	133:17 157:8	<b>178</b> 9:6
152:21 153:11	251:18	167:20 168:4	<b>18</b> 9:19 175:10
169:2 194:25	<b>100,000</b> 111:22	170:2 171:20	175:17,24
199:10 200:4	<b>107</b> 7:13	173:11 228:9	176:6 177:8
200:15 204:17	<b>10:26</b> 77:23	272:3 276:23	184:15,19
226:25 292:3	<b>10:40</b> 78:3	<b>130</b> 7:18	185:15,22
<b>1,580</b> 156:11,16	<b>11</b> 8:16,22	<b>14</b> 9:7 164:8	239:7 246:23
219:4,25 220:6	87:14 139:7	173:22 178:23	247:7
<b>1,800</b> 111:22	164:11,17	179:4	<b>183</b> 9:9
<b>1.4</b> 72:7,9	167:21 173:22	<b>142</b> 8:6	<b>188</b> 9:12
<b>1.5</b> 161:7,7	251:22 252:17	<b>148</b> 8:9	<b>19</b> 9:23 178:4
<b>1.5.</b> 172:16	270:25	<b>15</b> 9:10 43:8	178:16 183:21
<b>1.8</b> 106:9,10	<b>11,030</b> 185:24	159:21 160:2,4	184:15,22
110:5,22	186:13 230:22	160:9 161:22	187:20 253:25
111:10 112:3,5	231:13 234:2	161:25 162:7	256:6,9
112:19,23	235:3,20	169:19 183:4	<b>19.5</b> 154:24
113:3,9,18	<b>110</b> 185:23	183:23 184:4	<b>1969-1978</b> 8:17
114:22 115:7	<b>1100</b> 3:10	221:17 229:16	164:13,23
<b>1.92</b> 180:8	<b>1111</b> 1:17 12:13	263:19,25	<b>1975</b> 175:11
	14:9,19	266:13 267:10	176:7 194:18
		267:17,19,21	

<b>1981</b> 8:17 164:13 <b>1985</b> 175:11 176:7 194:19 <b>1:32</b> 201:7 <hr/> <b>2</b> <hr/> <b>2</b> 4:5 6:10 8:22 24:16 25:2 28:17 39:15 40:2,6,8,10 110:13 149:3,7 159:18 160:15 160:19 161:11 167:21 252:6,8 253:5 <b>2,100</b> 207:19 <b>2.5</b> 239:13 <b>20</b> 10:7 152:2 184:15,19,22 185:15 186:11 196:6,24 197:3 197:10,22,24 198:24 230:20 232:6,25 233:25 235:4 235:21 251:19 252:7 253:5 263:19,25 264:6,11 265:25 266:13 266:15 267:10 267:21 272:25 282:6 301:21	<b>200</b> 229:2 <b>20005</b> 3:12 <b>2007</b> 9:25 254:4 <b>201</b> 4:11 <b>2010</b> 8:9 148:3 148:19 152:17 <b>2011</b> 138:15 <b>2013</b> 138:16 <b>2014</b> 8:19 9:6 138:16 166:8 166:18 168:14 169:21 175:18 175:23 178:24 179:10 <b>2017</b> 7:23 30:23 130:12 <b>2018</b> 9:9 183:24 186:7 187:4 195:10 232:25 <b>2019</b> 9:20 246:25 <b>202.616.4226</b> 3:13 <b>2022</b> 8:12 157:10 221:8 <b>2024</b> 9:12 23:10 188:20 189:7 279:4 282:22 284:3,9 286:3 <b>2025</b> 1:13 12:10 108:17 285:9 285:15,18,23 301:11 303:24	<b>21</b> 184:15 187:25 194:11 224:13 225:2 227:13 236:4 237:9 239:18 282:14 <b>219</b> 2:7 <b>22</b> 6:6 137:17 194:17 <b>225</b> 9:15 <b>23</b> 8:17 139:20 140:21 164:13 194:10,25 199:10 200:4 200:15 239:16 <b>24</b> 6:9 <b>246</b> 9:18 <b>247</b> 218:16,17 229:2 236:22 236:25 237:8 238:17 <b>25</b> 6:17,21 158:9 160:8 162:14 220:19 225:12 255:23 272:25,25 <b>253</b> 9:22 <b>25th</b> 153:24 284:3,8 285:18 285:22 <b>27</b> 143:13,24 144:13,20 150:13 217:22 218:5,8 219:20	282:4 <b>272</b> 256:5,15 <b>28</b> 180:20 <b>282</b> 10:6 <b>289</b> 144:2 <b>29</b> 167:14 <b>290</b> 144:24 <b>29440</b> 2:8 <b>298</b> 4:10 <b>2:17</b> 242:21 <b>2:33</b> 242:25 <b>2nd</b> 2:15 <hr/> <b>3</b> <hr/> <b>3</b> 4:5 6:18 25:18 26:4,21 27:15 27:21 28:18,22 39:15 40:2,6 149:3 156:13 190:24 191:3 191:10 193:23 194:16 206:9 <b>3,100</b> 207:13,17 <b>30</b> 1:13 23:13 51:23,24,25 94:25 183:3 300:15 301:11 <b>301</b> 4:12 <b>302</b> 4:13 <b>303</b> 4:14 <b>30th</b> 12:10 <b>31</b> 155:4 <b>322</b> 248:7 <b>33</b> 251:22 252:17
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<b>3314</b> 3:11	<b>5</b>	<b>6-8</b> 250:3	179:22 187:6
<b>333</b> 250:2	<b>5</b> 4:6 7:7 11:16 52:2,11 93:7 114:7 115:6 116:23 159:18 159:21,25 160:4,9 165:3,8 177:11,17 197:9,14,22 198:16 221:16 222:13 240:6 262:6,13 280:22,22	<b>6.4.1</b> 248:11	187:17,23
<b>335</b> 251:10		<b>60</b> 81:9	219:11 262:12
<b>337</b> 222:6		<b>60s</b> 113:15	<b>8,000</b> 106:7,7
<b>36</b> 187:21		<b>7</b>	106:21 108:25
<b>380</b> 228:23		<b>7</b> 7:19 9:15	109:8
<b>3:21</b> 291:10	<b>5,340</b> 228:18 234:20 <b>5.7</b> 144:8 <b>5.8</b> 155:4 <b>50</b> 104:12 152:2 153:25 183:8 <b>500</b> 251:19 <b>52</b> 7:6 <b>577,000</b> 213:13 213:24 214:20 215:12 <b>5th</b> 285:9,14	130:8,16	<b>80,000</b> 108:18
<b>3:30</b> 291:14		137:15 171:19	<b>81</b> 222:6
<b>3:37</b> 299:20,25		171:21 173:11	<b>842.546.2408</b>
<b>4</b>		185:3,5,10	2:9
<b>4</b> 6:22 11:16		186:17 190:24	<b>877.370.3377</b>
25:22 26:5	<b>6</b>	191:3 207:11	1:25
27:15,22 28:18		225:13 252:8	<b>9</b>
39:20 158:6		<b>7,000</b> 228:20	<b>9</b> 8:10 139:7
160:6 179:22		<b>7,700</b> 228:12	148:2,7 168:25
193:10,12		<b>70s</b> 113:15	173:16 178:22
194:5,9 276:24	<b>6</b> 7:14 107:4,5 108:2 130:7 165:3,8 222:13 <b>6,531</b> 152:21 <b>6-3</b> 256:15,23 257:15	<b>711</b> 187:21	193:9 239:21
<b>4,100</b> 207:18		188:2 236:5	262:9,14,15
<b>40</b> 113:21,23		237:11 238:18	303:24
114:5,12,17,25		<b>75th</b> 153:25	<b>9/11</b> 259:8,17
115:21,24		<b>76</b> 149:9 155:6	259:19 261:13
116:17,24	<b>8</b>	185:2	<b>90</b> 75:25
117:18 118:4		<b>78</b> 187:8,9	<b>90th</b> 144:20
262:5,8		<b>79</b> 187:7 222:5	<b>917.591.5672</b>
<b>401.273.8330</b>		<b>7:23</b> 1:6	1:25
2:17		<b>8</b>	<b>95</b> 114:23
<b>42</b> 170:21	<b>6</b> 7:14 107:4,5 108:2 130:7 165:3,8 222:13 <b>6,531</b> 152:21 <b>6-3</b> 256:15,23 257:15	<b>8</b> 8:7 140:7	188:10 195:16
172:12		142:13,18	196:2,8,23
<b>44</b> 143:13,24		143:9,12 144:3	198:11,15
144:13,20		149:3 156:10	<b>9:00</b> 1:19
150:14 217:22		158:2 165:3	<b>9:26</b> 12:3,11
218:5 219:20	<b>8</b>	166:6,25	
<b>4600</b> 169:2		173:22 177:11	

<b>a</b>	<b>academies</b> 7:6 49:19,22 50:8 50:18,23 52:3 53:5,18 55:3	175:7 212:25 265:14 277:11 277:18 278:4	<b>aerosolization</b> 259:9
<b>a.m.</b> 1:19 12:3 12:11 77:23 78:3 141:22 142:2	<b>academy</b> 49:23 50:15 51:17	<b>actually</b> 23:13 30:17,17 44:11 57:5 73:20	<b>affairs</b> 7:10 52:5 296:14
<b>abdominal</b> 73:15	<b>accepted</b> 20:10 20:21 21:6,7 49:25 261:2	80:17 99:15 108:4 120:14 120:20 141:19	<b>affect</b> 15:18 97:8 209:19
<b>ability</b> 15:10,18 42:19,24 43:11 43:16 63:3 68:23 298:7 303:15	<b>access</b> 124:25	204:7,8 214:18 215:3 216:25 259:12 263:3	<b>affected</b> 162:19
<b>able</b> 38:20 39:3 56:4 74:17 128:20 216:15 217:6,18 230:5 230:11 233:5 238:8 242:8 258:15 299:2	<b>account</b> 51:8 51:19 112:12 145:12,18 146:6,8,18 147:2,10,17 182:17,18,21 212:15 213:24 213:25 250:17	<b>addition</b> 251:4 254:11	<b>affecting</b> 76:3,6
<b>abnormal</b> 62:21 68:23	<b>accounting</b> 38:7	<b>additive</b> 293:4 293:9,21 294:2	<b>affirm</b> 13:9
<b>above</b> 1:19 29:3 46:7,11 139:12 154:17 170:20 181:19	<b>accurate</b> 15:14 15:19 298:7 300:19	<b>address</b> 14:8,9 14:17	<b>aftercare</b> 277:14
<b>absence</b> 34:19 35:7	<b>accurately</b> 19:3	<b>additional</b> 8:22 167:20 205:6 205:10 249:21 294:12	<b>afternoon</b> 4:11
<b>absent</b> 115:24 116:17	<b>acknowledge</b> 21:6	<b>adds</b> 128:22,23	<b>age</b> 113:21,22 114:5,12,17,25 115:21,23 116:16,24 117:18 118:4 261:24 262:8 269:24 270:19
<b>absolutely</b> 117:19 299:5	<b>acronyms</b> 129:17	<b>adjustments</b> 57:14	<b>agents</b> 158:23 163:5
<b>academic</b> 50:3 58:22,24	<b>act</b> 33:7,10	<b>admittedly</b> 156:5	<b>agree</b> 18:25 19:7,11,22 20:5 20:16 21:18 33:20 34:5,11 36:25 37:5,10 37:17 38:13,17 38:23,25 39:5,6 39:10 42:8,12 42:15 49:4,9,12 49:14 58:13,15 65:20 66:2 69:8 69:13 74:2 76:22 77:3 79:3
	<b>action</b> 303:18 303:21	<b>adults</b> 211:12	
	<b>actual</b> 26:10 123:12 124:25	<b>advances</b> 50:3	
		<b>advantages</b> 35:19	

86:19 87:9 96:19,24 97:6,7 97:12,19 112:10 113:2,7 116:19 125:19 150:18 162:18 174:15,21 175:2 180:24 181:7,23 196:5 209:18 228:10 230:21 231:11 239:6,12 243:21 253:8 257:3 268:10 272:10 298:25 <b>agreed</b> 5:5,11 5:15 122:13 <b>agreement</b> 55:10,14 125:11 126:15 281:12 283:19 <b>agreements</b> 283:12 <b>ahead</b> 286:19 <b>air</b> 155:7,12 156:2,6 157:2 246:18 248:11 248:16 249:11 249:13,24 251:14,18,20 251:20 252:15 253:10 <b>al</b> 138:15,16,16	<b>allow</b> 68:14 93:21 <b>allows</b> 237:23 <b>alter</b> 85:15 <b>alteration</b> 67:11 <b>altering</b> 274:16 <b>ambient</b> 246:18 248:16 249:11 249:13 251:2,6 253:10 <b>american</b> 73:21 250:22 <b>amount</b> 101:14 152:10 202:5,9 202:13,18 204:11 221:5 <b>amounts</b> 277:20 <b>analyses</b> 231:21 <b>analysis</b> 40:12 40:14,19 125:24 137:25 179:10 <b>analyze</b> 257:16 <b>andrew</b> 8:12 157:9 221:8 <b>anecdotal</b> 124:22 <b>anesthesia</b> 73:21 <b>aneurysms</b> 98:8	<b>annual</b> 280:17 <b>answer</b> 11:4 15:10 17:18 18:2 106:16 279:20 286:19 288:2 <b>answers</b> 16:21 18:16 301:14 <b>anxiety</b> 274:24 <b>anymore</b> 276:8 <b>aortic</b> 98:8 <b>apologize</b> 45:23 108:3 111:3 <b>apoptosis</b> 69:3 <b>appear</b> 88:9 247:22 <b>appearances</b> 4:5 283:3 <b>appears</b> 256:19 <b>appendix</b> 9:15 225:13 <b>apples</b> 237:23 237:23 <b>applicable</b> 260:25 <b>application</b> 59:23 <b>applied</b> 67:12 <b>applies</b> 43:19 261:5 <b>apply</b> 32:15 46:3 <b>applying</b> 30:9 30:12 32:20	<b>appropriate</b> 19:14,25 122:12 131:14 132:4 277:15 300:7 <b>appropriately</b> 20:8,18 275:18 <b>approximately</b> 106:25 111:10 111:21 112:19 <b>approximating</b> 161:2 <b>april</b> 9:9 183:24 <b>areas</b> 19:13,24 <b>argue</b> 72:3 <b>argument</b> 128:23 <b>arises</b> 81:17 <b>article</b> 7:8,14 52:4 107:6 131:4 140:13 140:14,17,25 141:14 143:2 144:24 148:18 149:2 157:24 159:11 161:20 165:2 166:24 167:5 179:9,15 184:14 <b>articles</b> 62:11 62:16 72:18 138:19 148:12 195:2 241:20
--	---	---	--

<b>asa</b> 73:22	<b>associated</b> 64:8	131:19,24	<b>attribute</b>
<b>asbestos</b> 89:24	64:23 75:12	132:8 133:2	274:19
259:10	76:8,16 85:21	183:23 184:8	<b>august</b> 9:25
<b>aschengrau</b> 8:6	124:3 157:21	186:7 187:4	23:10 153:2
141:9,13	163:21 199:22	195:7 203:19	254:4 279:6
142:13,22	292:22	204:5,9,15	<b>author</b> 44:25
143:17 145:9	<b>association</b>	213:22 214:14	<b>authoritative</b>
147:8,14,24	34:13 38:14,16	214:25 215:8	50:24 51:2,16
150:5,12	40:20 73:22	216:3,23 217:4	<b>authors</b> 145:10
<b>asdr</b> 214:25	136:5 137:7	226:8,11,11	148:19 159:20
<b>aside</b> 57:20	171:11 195:9	232:11 234:15	<b>available</b> 74:16
139:5 147:25	195:20 196:8,9	238:4 239:25	74:23 75:9
164:10 166:4	196:25 198:18	240:9 246:23	258:17 299:11
<b>asked</b> 17:20	198:25	247:23 248:3,6	<b>avenue</b> 1:18
19:18 45:24	<b>associations</b>	248:14 250:4	12:13 14:10,19
193:22 265:19	34:13,16 35:14	252:4 256:14	<b>average</b> 176:20
296:12 298:19	196:6,24	256:15	176:22 177:6
<b>asking</b> 20:25	198:24 199:12	<b>atsdr's</b> 133:8	251:20 252:5
53:13 218:11	<b>assume</b> 17:19	133:24 137:25	252:15 253:4
253:12 255:20	155:25 250:25	213:20 232:25	<b>aware</b> 33:2
<b>assessment</b>	258:13 273:6	<b>attached</b>	42:2,6 45:14
7:18 30:22,25	275:14	300:12	46:2,6 53:4,14
45:25 74:22	<b>assumed</b> 226:6	<b>attack's</b> 98:9	53:15,17,23
130:3,9,22	<b>assuming</b> 26:25	<b>attorney</b> 14:22	75:18 76:5
131:19 132:13	251:14,17	288:22 289:2	85:20 92:14
133:9,25 208:7	252:7	300:15 303:17	124:2 199:11
208:8 295:12	<b>atdsr</b> 30:22	303:20	199:21 200:12
295:13,15,19	<b>ate</b> 206:17	<b>attorneys</b>	200:17 207:16
<b>assist</b> 19:2,8	<b>atomic</b> 269:11	140:18 287:10	208:19,25
91:6	<b>atsdr</b> 7:18 9:9	287:16,22	216:8 258:8,11
<b>assisted</b> 280:24	9:18 30:24 33:4	288:11	258:22 273:24
<b>assisting</b> 280:18	41:7 45:25 46:6	<b>attributable</b>	274:4 278:6,8
<b>associate</b> 176:6	129:17 130:2,8	92:20 93:2,7	278:19 283:16
	130:22 131:13	94:13	289:11 296:10

<b>b</b>	176:7,23	271:21,21	169:14 192:17
<b>b</b> 6:3 7:3 8:3	179:11,13	273:11	208:11 211:23
9:3 10:3 56:24	184:12 189:11	<b>baseline</b> 249:19	233:16 252:23
57:15 61:19	194:11,18	<b>basically</b> 62:21	253:16,20
97:16	204:6,10	73:10 87:21	259:2 267:22
<b>back</b> 35:12	205:15,16,17	91:3 99:12	273:14 274:8
39:21 57:21	205:18,23	117:15 141:2	276:21 279:9
66:3 78:4 85:10	206:18 211:11	153:20 192:18	298:4
85:11 123:3	226:9,25 239:7	240:23 280:4	<b>believed</b> 21:14
129:25 132:21	239:13,18	281:25 287:4	120:10 228:17
139:6 142:3	254:12	<b>basing</b> 60:11,12	235:13
143:8 150:7	<b>based</b> 20:9,20	61:19	<b>believes</b> 56:24
197:19 199:2	21:5 29:25	<b>basis</b> 151:20	<b>bell</b> 2:5 290:17
201:7 206:8	30:24 41:19	252:24	<b>belllegalgrou...</b>
219:11 242:25	50:2 58:10,11	<b>bates</b> 10:8	2:10
268:7 274:25	58:16 60:8,8	282:8	<b>benefit</b> 36:7,9
279:5 281:22	74:22 88:7 92:3	<b>becoming</b>	<b>benefits</b> 47:9
286:2,4,20,22	92:4 123:2	95:13	296:14
291:14	125:11 134:21	<b>beginning</b>	<b>benjamin</b> 119:2
<b>background</b>	151:22 175:22	69:12 144:7	<b>benz</b> 120:9
25:14 112:13	181:14,16	148:13 247:14	<b>benzene</b> 9:25
112:18 113:2	202:2 204:2	292:17	36:20 120:9
113:16 114:15	205:3 206:16	<b>behalf</b> 2:4 3:4	127:4,10
120:8 121:7	207:6 208:21	<b>behave</b> 84:4	128:17 169:8
122:11 243:14	209:20 210:15	<b>believe</b> 24:10	211:19 244:17
243:22 244:7	210:22 211:14	28:7,19,20	253:9,13,15,18
244:21 245:5	213:11,19,22	30:12 55:17	254:4 256:13
<b>bacteria</b> 86:3	218:15 220:4	57:4 63:25	256:16 257:9
<b>bad</b> 63:24 64:2	226:5 228:3	68:20 86:8,15	257:19 292:7
136:21	229:22 230:2	99:14 100:13	292:20 293:14
<b>ballpark</b>	230:11 231:23	104:15 110:10	296:3 297:5,16
110:11	234:9,13,13,14	110:18 141:11	<b>best</b> 15:10
<b>base</b> 61:25	234:15 245:21	149:5 152:9	21:20 24:12
166:21 175:10	266:25 268:18	156:6 164:7	28:14 71:4,8



290:19 296:6 298:7 299:10 303:15 <b>better</b> 37:2,12 37:21 38:6 47:2 47:21 57:11,16 57:16 61:18 66:17 <b>bias</b> 37:3 38:21 42:5,14,19 43:12 146:19 147:4 <b>biases</b> 35:23 37:23 39:16 40:3,23 41:3,8 <b>bible</b> 51:3 <b>big</b> 73:4,15 83:16 <b>bile</b> 82:10,21 <b>biliary</b> 82:10 <b>bill</b> 277:16 284:13 <b>billing</b> 276:24 277:10,12,12 <b>billion</b> 149:12 149:14,17,22 149:24 150:11 155:6,11,13 162:14 163:16 163:21 164:5 220:13,19 224:13,18 225:2 231:20 235:17	<b>bills</b> 277:19,19 <b>biologically</b> 84:5 <b>biomarkers</b> 258:9 <b>bird</b> 31:3 119:5 119:20 120:2 122:5 125:7 287:4 <b>bird's</b> 31:12 123:7 125:16 126:21 139:25 <b>bit</b> 35:18 62:5 66:17 67:6 75:5 95:13 114:8 150:5 163:12 257:12 <b>blood</b> 57:8,9 75:4 210:18,21 <b>blue</b> 226:21 227:4,6 <b>bmi</b> 210:2 <b>bodies</b> 85:5 <b>body</b> 65:17 68:6 85:6,10 209:20,23 210:6,15,22 <b>boisclair</b> 2:13 <b>bomb</b> 269:11 <b>bottom</b> 54:11 57:24 240:2 271:2,3 <b>bove</b> 8:19 9:6 9:12 120:20	121:8 123:17 124:6,15 140:8 140:12,14,17 140:25 166:7 166:17 169:12 169:21 175:18 175:23 176:18 178:23 179:9 188:19 189:6 195:7 202:8 207:6,9 232:20 237:22 239:3 240:9 <b>bove's</b> 168:13 <b>bowel</b> 80:6 <b>boy</b> 225:22 <b>branch</b> 3:6 <b>break</b> 17:4,7,10 77:19 78:6 141:20 142:7 201:10 243:4 291:17 <b>breaking</b> 85:7 <b>breast</b> 106:12 <b>breathing</b> 251:19 <b>broadly</b> 63:22 <b>bu</b> 3:8 4:9 12:23,23 13:22 14:21 21:23 22:7 24:6,23 26:2 29:24 37:8 51:22,25 52:9 53:13 54:9	77:15 78:5 94:2 105:11 107:3 107:10 130:6 130:14 141:18 142:4,16 147:22 148:5 157:7,12 164:8 164:15 166:3 166:11 167:13 167:17 168:2 174:3 178:14 178:21 179:2 183:21 184:2 186:3 188:17 188:22 197:13 200:20 201:9 225:11,17 230:10 231:2 242:4,7,18 243:3 246:21 247:5 253:23 254:17,18 256:3 261:10 264:22 279:21 282:3,12 283:23 291:8 291:16 296:19 298:13 <b>burning</b> 267:12 <b>byproducts</b> 120:12 <b>bz</b> 226:23
--	--	--	--

<b>c</b>	<b>call</b> 29:18 47:5	63:2,6,7,12,12	109:13,15,21
<b>c</b> 2:2 3:2 129:18	99:13 103:15	63:15 64:9,24	110:6,17,22
301:2 303:2,2	105:20 287:2	65:9,21,23	111:12,23
<b>cadmium</b> 89:21	288:14	66:12,14,22	112:7 113:9,21
245:20 259:12	<b>called</b> 86:3	67:3,10,22	113:22 114:4,5
<b>cal</b> 235:7	<b>camp</b> 1:7 7:20	68:18 69:9,17	114:17 115:19
<b>calculate</b>	12:14 29:4,9	70:6 71:17,19	115:23 116:16
151:17 202:18	33:7,9 120:22	76:13,19 77:6	117:16,18,21
203:4 204:4	130:10,24	78:16,20,25	118:2,8,12
211:21,24	131:11,21	79:4,9,10,12,15	120:13,17
212:2 217:15	132:2,6,9,14	79:16,17,22,24	121:3 124:4,13
218:21 231:16	134:18 135:3	80:6,6,7,22,24	124:18 125:25
233:21 238:20	135:11 166:21	81:8,16,20,21	126:6,7 127:2,4
280:20	169:3 176:23	81:21,24,25	127:14 128:5
<b>calculated</b>	179:14 184:12	83:11 84:15,19	135:21,25
226:2 236:4	189:11 191:6,6	84:22 85:17,22	136:6 137:19
<b>calculates</b>	192:7 193:13	85:24 86:9,14	139:14 142:23
211:17 213:10	193:18,19,24	86:19,21,24	145:10,16,22
236:12	193:24 199:13	87:3,11,18,23	146:11,14
<b>calculation</b>	199:17 214:8,9	88:7,19 89:10	147:8,15
182:15 213:19	214:19 216:15	89:18 90:8,11	150:21 152:24
214:6 224:20	223:8 229:6,19	91:5,17,19,24	157:21 158:23
235:8,9,12	245:3 250:19	95:8,10,15,21	159:6 162:19
<b>calculations</b>	251:4 254:21	95:22 96:2,21	163:8,22 164:6
31:4 121:11	255:3,14 278:7	97:3,14,24 98:3	164:22 170:6
125:5 150:9	279:12 289:9	98:7,10,16,20	170:18,22
202:4,23	290:22 296:9	99:2 102:23	172:8 174:7
204:17 212:5,9	296:15	103:3,19,24	175:8,23 176:9
212:15 216:9	<b>campus</b> 223:23	104:14 105:2	177:14,25
216:17 217:7	<b>cancer</b> 6:12	105:16,23,24	180:3,7 185:18
224:17 232:17	7:13,14,16 9:16	106:11 107:6,6	186:20,24
232:18 234:9	24:19 35:25	107:8,12,17,18	187:16 188:9
238:4	36:22 62:12,16	107:18 108:7	189:7,14,18
	62:18,20,25	108:13 109:5,9	190:3,13 191:5

191:11,18 194:5 199:14 199:18,23 200:6,14 202:15 219:16 225:14 242:9 245:5 258:2,10 258:21,25 260:8 261:6,24 262:11,20 263:3,8 264:4 264:10 265:5 265:14 266:5,7 266:10,12,17 266:22 267:9 267:18 268:20 269:6,12,15 270:7,14 272:22 273:19 273:21 274:20 274:23 275:9 275:24 276:25 277:4,17 292:23 293:18 295:23 296:4,8 297:11,21,25 <b>cancer's</b> 76:12 76:15 106:13 106:13 261:13 269:18 <b>cancerogenic</b> 210:9 244:2 266:9	<b>cancerous</b> 267:20 <b>cancers</b> 7:22 31:9 63:10,21 64:25 65:13,14 66:2,9,24 67:14 67:23 68:10,12 69:13 80:2,3,8 80:10,17,20 81:4,12,19 82:5 83:20 84:8 85:25 86:2,6,15 92:8,19,25 93:11 94:4,11 95:18 96:8 100:10 106:12 106:19 109:10 109:20,24 113:3,13 114:9 115:13 116:22 116:23 130:11 130:24 131:12 189:25 245:19 259:18 262:2,7 263:17 269:8 270:11,12 275:5,12 <b>candidates</b> 73:12 <b>capable</b> 164:5,7 <b>car</b> 128:10 <b>carcinogen</b> 36:19 69:6 70:4 98:4,13 152:13	245:25 263:16 <b>carcinogenesis</b> 120:11 122:10 141:6 <b>carcinogenic</b> 100:14 104:7 117:25 128:11 128:18,24 146:10 158:22 163:4 245:11 265:12 293:13 293:15 <b>carcinogens</b> 31:7 99:19,22 100:2 243:23 292:8,21 293:23 <b>carcinoma</b> 82:7 82:9,25 83:3,13 83:14 <b>carcinomas</b> 92:7 <b>cardiovascular</b> 73:4 <b>care</b> 71:5,15 75:17 80:13,18 260:16 271:6 271:13 272:7 272:12 274:4 275:23,25 276:5,6 295:16 <b>carefully</b> 300:5 <b>carolina</b> 1:3 2:8	<b>case</b> 1:5 15:3 22:15 24:9 25:9 29:3,9 30:17 35:15 36:16 44:11 45:20 104:4,9 112:23 115:16 117:14 117:23 128:6 131:15 223:20 247:15 249:20 258:17 278:11 278:13,19 281:6,17 284:2 285:5,18,22 286:9,22 290:11 291:5 297:13,15,19 297:23 <b>cases</b> 1:10 96:2 106:7,7,21 108:16,25 124:23,24 145:11,16 147:9,15 152:16 154:5 154:22 170:22 175:5 257:18 257:19 258:18 285:6 289:17 289:19,22,23 290:9 299:2 <b>casework</b> 284:25
---	--	--	--

<b>categorical</b> 8:23 167:21	124:18 125:25 128:22 138:3	69:7 80:17 85:16,16 86:20	<b>certify</b> 301:9 303:6,11,16
<b>categories</b> 169:25 185:11 185:17	143:7 150:21 152:23 167:23 176:3,16 198:7	86:24 87:3,11 87:12 92:7,8 93:11 124:12 126:6,7 192:2,7	<b>cetera</b> 75:23 100:15 214:4 249:17
<b>categorizing</b> 99:6	204:8 222:18 245:4 253:11	<b>cells</b> 63:12 67:25 68:2,24 69:2 82:6,13,17 83:5 87:10	<b>challenge</b> 95:13 149:20 171:16 181:20 182:24
<b>category</b> 73:23 105:19 186:7 186:17 239:2	267:22 274:10 284:8 285:25	<b>center</b> 8:15 164:11 259:22 260:3 261:12	<b>challenging</b> 71:21 238:11
<b>causal</b> 29:12,14 30:3,14 31:14 31:23 32:6,12 34:18 36:4 38:14	<b>caused</b> 59:8 61:5 65:15 66:11 67:10,11 67:16,17 69:25 88:15 95:21 103:17 258:2 261:6,13 264:7 264:12 268:11 271:25	<b>certain</b> 35:5,6 35:14,15 46:20 56:15 63:19 98:19 122:18 151:3	<b>chance</b> 39:16 40:3,22 41:3,8 42:4,10,25 43:16 114:7 115:3 118:7 171:10,17 173:7 174:18 197:3,8 199:3 257:9,11,16 294:14
<b>causation</b> 6:6 22:4 29:11 30:2 31:2 32:16,21 33:3 34:21,23 70:13 119:2,21 123:10 134:7 134:14 238:21 264:17 298:22 299:2	<b>causes</b> 63:12 69:8,15,17 84:16 86:4 87:22 88:2 89:18 91:5,19 92:2 96:21 99:10,15 103:12,20 105:2,8,9,16 112:6 117:24 128:5 245:18	<b>certainly</b> 51:19 200:18 274:2 277:18	<b>chances</b> 72:19 117:21 118:5 273:2
<b>causative</b> 34:15 125:6,14	92:2 96:21 99:10,15 103:12,20 105:2,8,9,16 112:6 117:24 128:5 245:18	<b>certainty</b> 36:3 58:4,9,17,22 59:11,16,25 60:9,13,18 61:8 61:23 62:3 70:12 125:13 263:13	<b>change</b> 61:16 182:20,23 302:3
<b>cause</b> 8:25 67:3 67:10 74:16,16 76:25 84:13,21 93:10 96:10 99:2,7,13 102:11 104:2 104:14,20,22 105:18 112:11	<b>causing</b> 33:23 34:8 65:9 164:5	<b>certificate</b> 4:14 <b>certification</b> 5:7 <b>certified</b> 1:21 303:4,23	<b>characteristics</b> 64:8,13 83:24 87:7 <b>characterizes</b> 20:8,19 <b>charge</b> 282:2 <b>chart</b> 169:15 204:4 206:17

226:25 <b>charts</b> 202:3,24 202:25 203:4 213:2 <b>check</b> 169:15 <b>checklists</b> 92:11 <b>cheese</b> 250:22 257:19 <b>chemical</b> 257:25 258:8 259:8 <b>chemically</b> 258:21,25 <b>chemicals</b> 129:3 207:25 208:6,22 210:4 210:5 212:20 216:23 265:12 294:4 <b>chemistry</b> 155:20 <b>children</b> 211:9 211:9 <b>chloride</b> 36:20 120:10 127:4,9 128:18 169:8 211:18 244:16 296:3 297:5,16 <b>cholangiole</b> 82:8 83:3,13 <b>chronic</b> 73:5 84:12,13,25 86:4,10,11,16 88:24 90:6	267:15,16 <b>circumvent</b> 68:15 <b>cite</b> 123:3 149:7 156:10 157:25 158:8 165:2 166:24 175:10 179:16 184:14 185:23 <b>cited</b> 140:5 143:3 149:3 158:5 <b>citing</b> 140:9 <b>civil</b> 3:6 <b>civilian</b> 179:12 189:9 193:13 193:17,18 194:12 239:18 <b>cl</b> 10:9 282:9 <b>clarify</b> 17:23 19:19 78:11 142:11 201:16 243:10 291:24 298:11 <b>classically</b> 74:14 <b>classifications</b> 207:9 <b>classified</b> 103:25 <b>clear</b> 70:3,3 73:18 87:9,12 92:7 96:9 126:6 191:25 192:6	271:23 <b>clearly</b> 32:25 61:18 64:23 95:17 97:22 98:18 171:5 210:5 272:24 <b>clinical</b> 59:3 79:20,23 81:3 83:24 87:6 151:19 210:13 258:12,19 <b>clinically</b> 171:3 172:15,17 258:12 <b>club</b> 50:6 <b>cohort</b> 166:22 179:10 189:11 249:16 <b>college</b> 94:22 94:24 155:20 <b>colon</b> 80:6 <b>column</b> 144:6 204:17 <b>combination</b> 91:15 206:14 226:11 <b>combined</b> 126:5 226:13 293:4,23 <b>come</b> 50:14 56:4 59:18 82:6 82:7,14 102:16 129:25 132:20 140:14 177:4	199:2 214:13 214:14 215:25 260:14 274:25 <b>comers</b> 113:16 <b>comes</b> 46:22 82:9 102:8 137:12 141:8 143:17 232:25 <b>coming</b> 83:4 102:18 175:18 176:17 <b>commencem...</b> 303:7 <b>commencing</b> 1:19 <b>comment</b> 19:18 231:15,18 257:12 264:25 268:24 <b>commenting</b> 60:6 <b>commercial</b> 278:15 <b>commercials</b> 278:17 <b>committed</b> 71:6 <b>committee</b> 54:16 <b>common</b> 81:20 81:23 82:3 87:10,12 103:20,23 105:23 106:4 106:12,13,14
--	--	---	---

106:18,20 108:6,10,13 114:11 211:9 211:12 262:25 265:5 <b>commonly</b> 209:8 253:10 <b>communicati...</b> 286:17 288:25 289:5,8,13 <b>community</b> 49:6 51:15 53:7 54:19 56:4,22 56:24 91:2 223:14 224:3 <b>community's</b> 56:14 <b>comorbid</b> 73:2 <b>comorbidities</b> 71:18 73:19 <b>comparable</b> 224:7 254:20 <b>compare</b> 109:23 110:3 125:2 216:6 220:16 228:7 232:3,5 233:5 233:12,15 235:2,15 236:3 237:8,23 238:9 238:11,13 239:2 <b>compared</b> 105:23 106:4	123:15 232:14 234:7 250:23 <b>comparing</b> 91:21 92:8 193:17 237:17 237:20 238:3 241:5 <b>comparison</b> 191:5 193:23 202:6 235:20 238:17,23 240:23 244:18 249:16 <b>compensation</b> 284:23 <b>complete</b> 15:15 15:19 89:16 298:6 301:12 <b>completely</b> 85:18 171:16 173:7 244:2 <b>complication</b> 71:24,25 72:5 73:8 74:19 76:25 <b>complications</b> 72:15 75:12,24 <b>component</b> 83:15 <b>compound</b> 128:5 <b>compounds</b> 89:8 96:17 118:2 124:21	126:5 127:12 135:24 169:3 225:9 226:4 229:6,13,19 230:15 245:14 <b>concentration</b> 149:8,15,18,25 150:16,20 151:23 155:7 156:24 206:11 206:13 209:9 215:4 216:14 216:23 217:5 220:13 221:3 225:5,8 226:12 227:7,13 251:15,18 252:8 <b>concentrations</b> 158:13,16 162:15,20 165:9 205:5 206:24 214:7 214:13,17 215:2,10 216:4 216:25 228:4 256:16,22 <b>conclude</b> 29:12 29:13 30:3,13 31:14,23 32:11 <b>concluded</b> 53:5 53:18 145:10 <b>concludes</b> 54:17	<b>conclusion</b> 55:2 56:16 133:8,24 134:10 299:12 <b>conclusions</b> 134:17 299:10 <b>condition</b> 46:21 47:19 66:14 84:16 91:25 92:3 103:13 <b>conditions</b> 73:3 73:10 90:7 <b>conduct</b> 198:10 <b>conducted</b> 40:19 135:15 <b>conducting</b> 196:22 <b>confidence</b> 38:22 39:17 40:4,24 41:9,12 41:14,17,19 42:4 61:9 161:12,15 172:13 174:16 174:22 177:24 178:7 180:9,15 180:25 181:11 181:18 186:19 188:7,10,13,14 191:20 194:4 195:16,17,21 196:2,8 198:14 266:14 <b>confident</b> 98:24
--	---	---	---

<b>confirm</b> 34:14 <b>confounders</b> 37:23 <b>confounding</b> 37:13 39:4 42:13 146:19 147:3 <b>confused</b> 117:12 198:8 231:8 <b>connections</b> 298:21 <b>consensus</b> 49:5 49:16 52:24 53:7 54:18 55:19,20,25 56:5,6 <b>consider</b> 28:15 32:20 42:18,24 43:11,15 190:14 203:23 205:11 206:25 244:6 246:3,17 250:10 294:18 295:2 <b>considered</b> 6:13,19,23 24:19 25:11,20 25:24 27:21 28:17 40:17 50:18,23 57:16 87:18 89:13 107:22 109:13 124:16 145:11	145:16 147:9 147:15 202:7 206:4 267:4 269:25 <b>considering</b> 146:17,25 <b>consistent</b> 129:2 141:5 233:25 258:20 <b>consult</b> 271:16 <b>consultant</b> 280:10 <b>consulted</b> 92:11 241:21 271:12 <b>consulting</b> 280:10 281:5 <b>consumption</b> 218:21 252:9 <b>cont</b> 3:2 <b>contact</b> 215:23 216:5 279:11 <b>contacted</b> 278:10,20 279:25 <b>contain</b> 24:7 <b>contaminants</b> 7:20 130:10,23 131:10,20,25 132:14 134:18 <b>contaminated</b> 142:23 157:2,3 166:20 179:13 184:11	<b>contamination</b> 135:10,11 157:23 227:8 241:22 250:5 <b>context</b> 49:2 55:11 133:19 134:23 259:4 <b>contexts</b> 33:18 46:11 <b>continues</b> 263:21 <b>contract</b> 281:13 <b>control</b> 70:11 71:15 299:4 <b>controlling</b> 37:2,12,21 38:6 42:13 <b>controls</b> 144:21 153:24 <b>convention</b> 198:10 <b>conversation</b> 298:23 <b>conversations</b> 59:6 62:24 <b>conversely</b> 182:6 <b>conversion</b> 233:15 <b>conversions</b> 150:8 <b>convert</b> 224:21 235:10	<b>convey</b> 101:7 <b>convincing</b> 40:13 <b>copy</b> 282:20 <b>corps</b> 166:21 184:12 189:11 <b>correct</b> 14:7 17:25 18:7,10 22:20 41:5 57:3 57:4,7 62:13,17 74:9 76:7 78:11 79:2,18 86:25 87:19 90:16 96:21 105:5 108:12 109:7 111:13 112:9 115:25 116:3 117:5 119:21 119:22 123:23 123:24 139:22 141:10 142:11 143:15 149:11 149:15 150:3 156:21 158:11 161:18 165:10 167:11 169:5 169:10,23 170:4 171:25 173:13,20,24 177:22 179:20 180:7 182:10 184:17,21,24 188:5,16 189:14 193:20
--	---	--	--



194:2,7 198:13 198:22 199:6 200:10 201:16 207:15 211:6 211:20 212:18 213:17,21 214:10 221:18 222:16 228:25 236:13,14,18 236:19,22,23 237:4,5 239:10 239:15,20 243:10 248:16 251:7 255:17 263:5,6 283:5 283:19 284:10 291:24 292:12 298:11 299:15 299:16 301:15 <b>corrections</b> 18:12 300:6,8 <b>correctly</b> 176:24 218:9 218:10 240:15 251:23 252:10 252:13 292:24 293:6 <b>correlated</b> 120:16 139:14 200:6,14 219:15 <b>correlating</b> 119:15 124:10	<b>correlation</b> 33:21 34:6 36:14 101:13 161:6 <b>corresponds</b> 27:2 187:22 188:3 <b>cost</b> 47:16 <b>costs</b> 47:9 277:23 278:4 285:2 <b>counsel</b> 5:6 12:18 26:15 279:23 281:11 283:13 285:14 286:18 303:18 303:20 <b>country</b> 72:4 <b>county</b> 301:6 <b>couple</b> 120:6 182:17 <b>course</b> 286:8 <b>court</b> 1:2 5:18 13:3 93:24 283:2 300:20 303:22 <b>courtroom</b> 18:22 <b>covers</b> 37:25 <b>creating</b> 85:8 <b>critical</b> 125:9 286:21 <b>cross</b> 171:5,8 171:15 172:14	172:18 174:17 177:25 194:6 195:21 <b>crossed</b> 195:17 <b>crosses</b> 172:20 173:2 178:8 180:7,9,16 186:21,24 188:9,11 191:21 <b>cubic</b> 251:19 <b>cumulative</b> 8:23 140:6,7 150:6,16 156:11,21,25 167:22 168:25 169:19 171:23 173:18 174:6 185:6,23 186:12 187:12 187:25 207:12 217:22 218:3 218:12,14 219:20,24 227:2 228:11 231:12 234:2 235:2 236:5,21 237:10 <b>cured</b> 109:11 190:8,9 <b>current</b> 23:9 48:21 53:20 54:22 271:5,13	<b>currently</b> 259:5 272:19,21 276:20 <b>curriculum</b> 22:16,22 23:3,7 23:22 <b>curve</b> 151:2 <b>curves</b> 102:4,7 <b>cutoff</b> 207:17 <b>cutoffs</b> 207:22 <b>cv</b> 1:6 <b>d</b> <b>d</b> 301:2 <b>d.c.</b> 3:12 <b>daily</b> 151:20 251:20 252:5 252:15 253:4 295:15 <b>damage</b> 267:11 <b>dame</b> 121:18 <b>data</b> 25:11 41:19 61:20 75:18 88:8 107:12,22 127:10 135:4 151:16 192:22 203:19,19,23 204:9,13,15,16 245:21 299:11 <b>data's</b> 127:8 <b>database</b> 107:17 <b>date</b> 1:20 12:10 22:6 23:9 24:9
--	---	---	---



24:22 25:21,25 52:8 107:9 130:13 142:15 148:4 157:11 164:14 166:9 167:24 178:25 183:25 188:21 225:16 247:3 254:5 279:7 282:11 300:11 303:14 <b>dated</b> 303:24 <b>dates</b> 284:4 <b>daughters</b> 28:9 <b>david</b> 6:7,15 9:15 22:4 24:21 225:14 <b>day</b> 100:9 226:7 251:20,22 252:7,9,17 253:6 284:9 301:21 <b>days</b> 75:25 95:15 109:11 150:8 206:20 222:6 223:21 223:22 226:25 300:16 <b>dealt</b> 123:10 131:9 <b>death</b> 8:25 68:17,20 69:7 167:23	<b>deaths</b> 109:2,8 <b>decades</b> 114:10 115:5 262:3 <b>decedent</b> 275:19 <b>decide</b> 136:20 <b>decision</b> 7:11 52:6 54:21 60:8 <b>deemed</b> 300:19 <b>defect</b> 70:2 <b>defects</b> 63:15 68:7 <b>defendant</b> 3:4 <b>defense</b> 44:13 45:5 <b>define</b> 58:7 63:9 103:8 208:13 241:13 <b>defined</b> 29:11 30:2 159:10 <b>definitely</b> 121:24 157:18 198:7 <b>definition</b> 30:22 31:22 32:10 <b>definitively</b> 36:8 253:21 <b>degree</b> 58:3,8 58:18,21 59:10 59:15,24 60:9 60:13,17 61:6,7 61:22 62:2 125:12	<b>delineated</b> 233:18 <b>delta</b> 182:20 217:9 <b>denote</b> 10:13 <b>denotes</b> 49:5 53:6 54:18 55:19 <b>densities</b> 156:3 <b>density</b> 156:7 <b>department</b> 3:5 7:9 9:22 14:22 52:5 254:2 296:13 <b>depend</b> 78:25 158:14 216:2 <b>dependent</b> 210:19 263:12 263:14 <b>dependents</b> 184:10 <b>depending</b> 78:20 81:15 <b>depends</b> 19:17 57:7,18 76:11 76:19 150:25 190:19 260:19 <b>depo</b> 286:12 <b>deponent</b> 12:16 <b>deposed</b> 15:21 15:25 16:8 281:3 <b>deposing</b> 300:15	<b>deposition</b> 1:16 11:2 12:12 15:2 16:19 18:11 27:7,14,19 28:6 28:8 142:6 201:11 205:4 205:24 206:2 213:11 214:2 243:5 245:22 283:2 286:3,14 287:10 289:3 291:18 300:4 300:12,16,18 301:11 <b>depositions</b> 28:5 205:13 286:23 <b>dermal</b> 212:17 212:21 215:16 215:22 216:5 216:10 217:10 217:16 <b>describe</b> 42:3 62:18 144:12 153:14 201:18 262:10 <b>described</b> 29:20 122:9 159:10 194:24 221:11 233:6,8,24 279:13 <b>describes</b> 185:5 187:11 234:19 281:18
---	---	--	---

<b>describing</b> 41:7 103:6 121:6 149:14 156:20 173:17 177:17 227:7 283:8	<b>determining</b> 38:13 39:2,8 112:11 124:17 150:18 161:4 205:11 206:4 214:7 271:12	117:20 118:8 121:3 202:24 245:17 259:7 273:9 297:11	<b>difference</b> 38:12 62:25 81:11 189:17 192:16 280:12
<b>description</b> 6:5 7:5 8:5 9:5 10:5	<b>detoxifies</b> 85:6	<b>development</b> 31:8 79:9,11 88:18 98:20 120:17 158:23 159:6 170:17 262:20 265:14 266:10,18 267:19 292:22	<b>different</b> 33:2 60:16,20 64:24 68:3 69:6 78:16 79:4,15 80:2 81:18 82:4,17 82:17 83:4,7,19 83:20,23 84:3,7 84:8 86:20,23 86:24 87:2,3,6 92:20 97:8 135:10 138:8 150:5 151:25 153:9,14 155:12 156:2,3 156:6 163:2,3 167:6,18 180:2 190:6,22 191:17 212:20 227:8,9 249:7 250:5,12,18 256:16 263:16 268:15,16,17 269:6,8,8,9,14 276:14,15 294:4
<b>designates</b> 25:8	<b>detoxify</b> 85:5	<b>develops</b> 62:22 117:18 263:3 265:5 270:6	
<b>designation</b> 6:9 24:17 26:20	<b>develop</b> 36:22 87:21 91:13,25 97:2,24 98:2 100:7 111:23 114:3,9,11,17 140:20 146:11 146:15 163:7 202:15 237:16 266:22,22 267:17,20 270:14	<b>diagnosed</b> 111:11 113:14 114:4,24 115:13 116:23 258:24 262:2,8 264:5,10 273:18	
<b>desired</b> 47:18 48:3	<b>developed</b> 68:14 98:16 127:16,18 189:23 190:7 190:11 238:24 269:12	<b>diagnosis</b> 71:19 103:11 260:9 260:22 261:18 261:24 269:24 274:20 276:25	
<b>desk</b> 153:4		<b>dialysis</b> 88:24	
<b>detail</b> 205:19		<b>diastasis</b> 274:9	
<b>detected</b> 145:22 246:4 248:15 249:2,6	<b>developing</b> 63:21 68:10 84:15,18 86:13 88:7 89:10 106:10 110:6 110:17 113:9 113:17,20,22 114:3 115:19 115:23 116:16	<b>dichotomy</b> 64:14	<b>differential</b> 87:22 89:17 91:3,14 92:2 93:3 96:12,13 105:7 244:10
<b>determination</b> 39:2,7 125:10 260:15,18 261:3		<b>die</b> 69:2,3 98:7 98:7 189:22 190:6	
<b>determinations</b> 34:17			
<b>determine</b> 35:5 203:24 216:16 217:6 222:21 230:5,11 234:11 241:21 257:25 261:5 277:9			
<b>determined</b> 207:9			
<b>determines</b> 236:20 261:12			

245:17 <b>differentiate</b> 95:14,20 <b>differently</b> 84:2 84:5 97:9,15 209:19 <b>difficult</b> 94:7 94:14,18 95:3,4 95:19 170:15 170:19 192:15 220:15 232:3 257:12 <b>difficulty</b> 174:25 <b>digestive</b> 80:5 <b>digit</b> 114:6 <b>direct</b> 36:4 101:13 <b>direction</b> 11:4 <b>directly</b> 220:16 260:11 <b>dis</b> 172:22 <b>disagree</b> 55:2 111:19 126:18 147:7,13,21 172:25 248:22 252:15 253:4,7 256:22 257:2 <b>disagreeing</b> 126:12,20 <b>disappointed</b> 155:21 <b>disclosure</b> 6:10 24:17	<b>discuss</b> 142:5 201:11 243:5 291:18 <b>discussed</b> 41:2 83:12 199:9 221:15 287:9 <b>discussing</b> 78:15 276:12 <b>discussion</b> 144:25 145:6 145:15 146:3 <b>disease</b> 33:21 33:23 34:6,8 40:21 61:6 73:4 73:10 76:11 88:24 98:9 112:12 115:16 189:22,23 190:7,8,9,10,11 193:3 271:20 <b>diseases</b> 7:22 75:16 130:12 130:25 131:12 180:2 <b>disjunctive</b> 29:18 <b>disorder</b> 275:4 <b>disregulation</b> 63:13 <b>disrupt</b> 67:9 <b>distinguishes</b> 167:6 <b>distress</b> 275:4	<b>distributed</b> 210:6 <b>distribution</b> 210:20 <b>district</b> 1:2,3 <b>divide</b> 153:21 153:21 154:2 <b>divided</b> 56:16 160:4 177:19 185:10 <b>divides</b> 68:5 <b>dividing</b> 68:2 124:7 <b>division</b> 1:24 3:6 68:3,8 85:16 <b>dna</b> 67:21 68:5 <b>doctor</b> 243:4 298:16 299:22 <b>document</b> 1:9 10:14 22:8,13 22:15 25:4,8,13 26:10 52:12,14 54:6,7 55:7,9 56:9 130:18 131:14 133:15 133:20 134:24 138:4 142:19 144:18 148:8 157:16,20 164:19 166:14 179:6 184:5 188:25 225:20 247:8,11	256:10 282:16 282:19 <b>documentation</b> 260:8 <b>documented</b> 89:9 245:13 249:22 <b>documents</b> 11:9 22:12 25:15 26:8,12 27:18 28:2,11 32:9 45:7 119:14 205:6 205:10 206:3 247:13 248:6 257:8 287:5 <b>doing</b> 16:25 91:18 136:10 182:15 300:10 <b>dose</b> 57:14 100:19 101:4,8 101:11,14,16 101:20,21,22 102:4,7,10,22 151:2,5 152:5 158:22 210:14 211:2,3 <b>dosed</b> 210:22 211:14 <b>doses</b> 151:3 <b>double</b> 255:22 <b>doubting</b> 126:12
--	---	--	--

<p><b>dozen</b> 16:2  <b>dr</b> 6:17,21  12:16 13:23  25:18,22 31:3,5  31:11,12 78:6  119:2,5,8,10,20  119:20,25  120:2 121:9,17  121:23,24  122:4,5 123:6,7  123:12 125:5  125:15,16  126:10,21  139:25 142:5  201:10 202:2  202:22 203:4  204:3 207:24  211:17,21  212:4,8,14,24  213:10 215:11  216:16,25  217:6 218:15  218:20 220:5  224:17 226:2  229:24 230:3  231:23 232:15  233:6,25  234:19 236:4  236:11 237:16  238:3,24 287:3  287:3,4 291:17  <b>drafted</b> 91:10  283:18,20</p>	<p><b>drafting</b> 25:11  91:7  <b>dramatic</b>  273:19  <b>drank</b> 206:19  <b>draw</b> 299:10  <b>drinking</b> 7:19  130:10,23  132:2 142:23  166:20 179:14  184:11 189:10  214:3 254:14  <b>dropped</b> 269:11  <b>drs</b> 31:2 125:7  <b>drug</b> 47:16,17  56:22,24 57:11  57:15 102:8,12  <b>drugs</b> 102:17  210:25  <b>ducts</b> 82:10,21  <b>due</b> 170:23  271:6  <b>duly</b> 13:17  303:8  <b>duration</b>  150:19 151:4,5  152:16 154:7  154:18 158:15  209:10 222:14  222:23  <b>durations</b>  224:6</p>	<p><b>e</b>  <b>e</b> 2:2,2 3:2,2 6:3  7:3 8:3 9:3 10:3  14:4 201:4,4  301:2,2 302:2  303:2,2  <b>earlier</b> 22:19,23  52:22 70:9  78:14 90:10  103:6 110:4  118:20 123:9  151:2 221:15  255:13 261:17  268:8 283:8  <b>early</b> 102:9  115:12 124:20  <b>easier</b> 40:7  183:4  <b>eastern</b> 1:3  <b>easy</b> 122:12  <b>eat</b> 250:22,23  <b>eating</b> 205:18  254:14  <b>edt</b> 1:19 12:3  299:25  <b>effect</b> 5:17  18:21 35:6  47:18 151:4  181:2,24 182:6  210:8 211:4  241:17 271:7  293:24  <b>effects</b> 47:17  241:6 274:19</p>	<p>295:23 296:3  <b>efforts</b> 281:8  <b>eight</b> 153:6  <b>either</b> 49:12  50:2 63:4,16  65:21 210:11  232:6 257:4  281:5 288:22  <b>elevated</b> 171:5  172:7 174:10  174:13 205:4  <b>eligible</b> 260:16  <b>eliminate</b> 38:21  <b>email</b> 2:10 3:14  <b>employed</b>  239:13  <b>employee</b>  303:17,19  <b>employees</b>  179:12 184:10  <b>endpoint</b> 57:7,9  57:12,19  <b>ends</b> 39:15  <b>engineering</b> 7:7  52:3  <b>enroll</b> 182:22  183:9  <b>ensure</b> 20:7,18  <b>entire</b> 55:7 56:9  82:20 85:2,3  98:6 113:17  133:20 134:23  154:2 170:22</p>
---	---	--	--

<b>entities</b> 60:25	<b>equivalent</b> 214:4 249:17	13:21 298:17
<b>environmental</b> 49:11,13 57:2	<b>etiology</b> 297:21	303:7
3:7 8:16 63:17	297:25	<b>examined</b> 13:19
64:3,14,17,21	<b>errata</b> 4:13	<b>example</b> 77:5
67:18 89:4,19	300:8,11,14	79:14 97:13
97:21 98:23	<b>error</b> 38:3,4,7	98:22 207:11
118:6 132:6	39:9 146:19	<b>examples</b>
151:14 159:5	171:10 178:9	210:17
164:12,23	178:18 180:17	<b>except</b> 5:11
263:18 269:8	180:22 215:15	<b>exclusion</b>
295:6 297:9	<b>errors</b> 37:19	103:11
<b>epics</b> 159:21	<b>esquire</b> 2:6,14	<b>exclusive</b> 55:21
<b>epidemiologi...</b> 3:8,9	<b>essentially</b>	<b>execute</b> 281:11
40:17 208:20	46:16 49:24	<b>exhibit</b> 6:7,10
294:24	50:5 68:13 85:4	6:18,22 7:7,14
<b>epidemiologist</b> 85:14 107:16	<b>established</b>	7:19 8:7,10,13
294:19	265:13	8:16,20,23 9:7
<b>epidemiologi...</b> 161:3 280:2	<b>estimate</b> 226:5	9:10,13,16,19
151:13	254:8	9:23 10:7 21:25
<b>equal</b> 162:22	<b>estimated</b>	22:2,3 24:16
269:23	204:6 229:2	25:2,18,22 26:4
<b>equate</b> 49:10	240:12 241:14	26:5,21 27:21
<b>equation</b> 215:23	251:22 252:6	27:22 28:17,18
<b>equations</b> 101:18	252:16 253:5	28:18,23 39:12
<b>equipoise</b> 46:7	255:14	52:2,11 57:21
46:10,13 48:25	<b>estimates</b> 181:2	87:15 107:4,5
49:5,10,17	217:17,19	110:14 130:8
52:23 53:6,19	229:10,12	130:16 137:21
54:17 55:18,20	<b>estimation</b>	142:13,18
55:24 56:6,7,17	216:3	143:8,9 144:3
134:7	<b>et</b> 75:23 100:15	148:2,7 157:9
	138:15,15,16	157:14 164:11
		164:17 166:7
		166:13 167:16

167:18,20 168:4 170:2 171:20 177:10 178:23 179:4 183:23 184:4 188:19,24 225:13,19 226:19 246:22 246:23 247:7 253:24,25 256:6,9 282:6 282:14 292:3 <b>exhibits</b> 4:6,16 10:12 27:15 <b>existing</b> 31:5 92:6 134:25 232:19 235:16 237:18 <b>exists</b> 29:13 30:4 <b>expect</b> 196:6,14 196:25 198:15 198:25 241:6 284:24 <b>expected</b> 192:8 240:25 <b>expensive</b> 46:23 47:20 57:15 <b>experience</b> 19:15 20:2 58:11 91:15 103:18 125:12 266:11 276:4	<b>experiential</b> 92:4 <b>experiment</b> 295:9 <b>expert</b> 6:6,11 10:9 15:22 16:4 19:3,12,23 20:6 20:17 22:4 24:18 25:9 27:10,14 44:12 45:5 232:16 238:21 280:10 282:9 286:25 289:18 290:12 290:17,20 291:5 295:6,12 295:14,18 297:14,24 <b>expert's</b> 118:21 <b>expertise</b> 50:4 151:16 158:21 <b>experts</b> 121:5 151:14 237:16 238:19 286:8 287:3 <b>explain</b> 17:14 35:18 66:17 101:10 120:7 127:20 189:16 236:2 255:9 293:8 <b>explaining</b> 90:15,18 171:11	<b>explanation</b> 100:12 <b>expose</b> 36:18,21 215:21 <b>exposed</b> 69:5 89:20,23 99:21 100:8 120:24 123:23 144:21 145:11,17 146:9 147:9,16 166:19 179:13 184:11 189:9 202:5,9 204:7 210:4 215:6 223:19 224:12 226:10 228:22 228:25 229:4 229:11,13,17 230:13,22 243:14,22 244:7,12,14,16 249:14 250:11 263:22 269:13 292:6,17,20 293:16 <b>exposure</b> 31:4,6 31:21 33:21,23 34:6,7 35:16 40:15,20 61:5 64:17,21 67:18 70:3 86:6 89:7 96:16 97:24 98:2,4,13 100:14 104:7	104:18 117:25 120:15,23 121:7,12 123:8 124:3,7,8,9,10 124:17 128:11 139:12 140:6,7 141:3 143:23 146:12 149:8 150:6,15,15,19 150:21 151:10 151:25 152:2,2 152:10,12,16 154:6,8,18,23 156:11,21 157:2 158:9,15 159:2,3,10,22 159:25 160:2,7 160:8 161:22 162:8 163:7,11 163:13,20 164:4 165:25 167:7 169:2,3,7 169:12,20,21 171:23 173:18 174:6 177:18 177:20 185:6 185:11,17,23 186:7,12,16,23 187:12,21,23 187:25 188:3 192:7 195:10 199:13,22 200:5 201:19 201:24 202:4,7
--	--	--	---

202:13 203:13 203:18,25 205:3,12,20 206:5,9,12,14 206:15 207:6,8 207:12,12 208:7,21 209:2 209:8,10,19 211:22,25 212:10,17,22 213:4 215:14 215:22 216:18 217:8,22 218:4 218:12 219:3 219:20,25 220:6,19,21,24 221:21,24 222:4,22 223:8 224:7,8 226:4 226:12 228:11 228:16 229:25 231:12,22 232:22 234:2 234:20 235:2 235:14 236:3,5 236:13,17,21 237:2,9,10 238:22 241:2 245:3,5,13,20 245:23 249:18 249:22 251:2,6 254:9,12,15 255:5 258:3 261:6,13	262:19 263:2,8 263:14,20 264:6,7,11,12 265:4,12 266:9 266:21 267:21 268:11,23 293:3,5,12 296:8,23 297:3 297:15 <b>exposure's</b> 218:15 258:14 <b>exposures</b> 8:24 29:4,10 63:17 64:4 89:4,19,23 90:3 95:2 97:21 98:19,22 99:15 99:17 118:6 132:6 156:17 159:6 160:10 165:12,17 167:10,22 169:25 177:3 184:20,23 186:20 188:8 188:12 202:17 202:19 203:5 211:18 215:16 216:10 217:10 221:17 222:14 223:6 232:6 233:6,8,16,17 233:24 242:9 244:3,20 245:11 250:18	254:20,21 255:4,14,17 259:9 263:18 269:9 289:20 296:15 297:10 <b>express</b> 242:8 <b>expressed</b> 58:3 <b>expresses</b> 236:16 <b>extent</b> 63:7 84:11 279:17 286:16 <b>extraordinarily</b> 118:9,13 <b>extreme</b> 210:7 262:6 271:6 <b>extremely</b> 97:23 <b>extremes</b> 209:23 262:5 <b>f</b> <b>f</b> 201:4 303:2 <b>fact</b> 16:9,11,13 21:15 146:9 244:15 273:7 275:15 <b>factor</b> 70:6 84:15 94:4 95:8 95:9 96:25 97:8 <b>factored</b> 206:21 215:23 <b>factors</b> 72:14 72:24 74:8,12 74:15,23 75:10	76:23 79:4,8,14 79:16,17 84:8 86:24 87:18 88:18,23,25 89:13 90:19 91:4,16 95:14 96:3 103:3 270:23 <b>facts</b> 7:14 25:10 107:7 <b>fail</b> 300:17 <b>failure</b> 73:6 273:10 <b>fair</b> 17:19 60:15 61:3 104:24 105:12,22 158:17 159:24 161:17 171:9 178:10,19 180:17,19,22 180:23 192:24 193:4 195:22 198:19 207:20 211:5 217:11 222:24 223:3,7 238:2,7 241:7 244:4 270:3 272:8 277:24 <b>fairly</b> 105:23 106:4 <b>falling</b> 162:12 <b>familial</b> 270:9 <b>familiar</b> 49:18 100:18 107:11
--	---	---	---



129:5 <b>families</b> 71:3 <b>family</b> 88:11,15 120:22 270:10 271:18 <b>fancher</b> 6:7,15 9:16 22:5,14 24:21 28:3,6 30:9 32:16 115:17 120:15 120:24 123:14 123:23 125:4 128:6 149:4 156:9 158:2 165:4 168:22 173:11,23 175:9 179:16 184:16 185:16 185:22 186:11 194:10 199:11 205:2,12,14 206:5 210:2 212:10 214:8 215:6 217:21 219:3 220:5,18 221:20 223:25 224:12 225:14 226:3 228:10 229:17 230:13 230:21 235:4 235:22 237:10 239:6,17 244:7 244:11 245:12 250:11 257:24	269:3 272:7,20 274:5 275:23 286:23 289:6 292:6,17,19 299:3,15 <b>fancher's</b> 24:8 26:24 28:7 30:17 36:16 87:23 90:10 91:20 104:4 117:14 121:11 139:11 201:19 202:17 203:5 203:13 205:3,6 212:25 213:11 213:25 214:21 216:9,18 217:8 218:12 222:22 223:7,20 224:8 226:13 228:11 228:16 231:12 231:22 232:6 233:6,16,24 234:20 236:3 236:12,21 244:19 245:4 254:9 255:4,16 261:18 262:11 263:4 264:25 269:18,24 274:20 276:13 276:18,25 289:14	<b>fancher</b> 000 26:22 <b>fantry</b> 165:3 <b>far</b> 17:2 78:10 117:22 118:17 118:17,17 211:13 216:3 273:11 275:12 280:21 <b>fashioned</b> 63:24 64:2 <b>fat</b> 210:19 <b>fax</b> 1:25 <b>features</b> 258:19 <b>february</b> 285:9 285:14 <b>federal</b> 5:2 <b>fee</b> 10:6 281:25 282:7,25 285:4 285:5 <b>feel</b> 294:13 298:5 <b>fees</b> 284:25 <b>felt</b> 176:23 202:8 226:14 259:19 <b>fewer</b> 192:6 211:13 <b>field</b> 34:12 50:4 50:19,24 100:13 107:23 213:12 240:10 <b>figure</b> 99:12 104:21 111:19	134:23 270:8 <b>file</b> 8:22 167:21 205:7 <b>filing</b> 5:7 <b>financially</b> 303:21 <b>find</b> 57:3 104:16 181:25 182:8 191:12 192:17 196:7 249:5 262:22 <b>finding</b> 292:10 <b>findings</b> 177:13 <b>fine</b> 28:13 93:23 111:4 129:24 144:17 167:19 231:10 242:6 <b>firm</b> 290:13 <b>firms</b> 290:21,25 <b>first</b> 13:17 21:25 22:13 29:6 115:4 120:7 128:16 139:10,17 141:8 143:3,13 144:7 145:5 170:2 219:19 226:5 247:14 278:6,8,18 279:8,11 284:9 287:19 292:5 292:15
---	--	--	---



<b>five</b> 16:3 23:12 23:14 77:19 110:2 116:21 117:3,7 197:8 197:21,24 221:20 242:3 281:3 285:24 286:5,11	<b>foregoing</b> 303:11 <b>form</b> 5:12 20:12,23 21:10 29:23 30:11 31:18 32:2,23 33:25 35:2 37:7 37:16 48:8,15 49:8 53:9,22 55:5,16 56:19 60:3,22 61:11 66:7,16 67:5 69:11,20 70:16 70:22 77:12 78:23 79:7 87:10,13 90:23 91:9 92:13 93:5 93:17 94:6 96:5 97:5,11,18 99:4 100:5,21 103:22 105:4 105:14 106:2 109:17 111:15 112:2,15,21 113:6 114:19 115:9 116:2,10 117:2,10 118:15 122:7 127:6 129:11 131:7,23 132:17 133:5 133:12 134:5 134:12,20 136:8 140:16	140:23 143:20 144:15 145:14 145:25 146:21 147:19 150:23 152:7 154:10 155:15,18 157:5 158:4,19 159:13 160:12 161:16,24 162:24 163:24 165:6,19 170:12 171:13 172:24 174:2 174:20 175:14 176:11 178:13 179:19 181:5 186:2 190:17 192:10 194:14 194:21 195:4 195:13,24 196:11,17 197:5,12,16 198:3,21 199:5 199:16,25 200:9 203:7 207:3 208:10 213:6,16 214:23 215:19 216:20 217:13 218:7,24 219:6 220:9,23 222:3 222:9 223:2,11 224:10,15 227:16,25	228:15 229:8 229:21 230:9 230:17,25 232:8 233:11 234:5 235:6,24 236:8 237:14 238:6 239:9 241:9 242:12 243:17 244:23 245:8 246:7 248:18 249:9 250:15 252:12 252:19 254:23 255:7 256:2 258:6 261:9,15 263:10 264:14 264:21 267:6 268:4,13 278:2 279:16 280:14 281:15 283:15 283:22 284:15 284:20 287:12 290:5 296:25 297:7 298:3 <b>formation</b> 67:22 68:18 <b>formed</b> 24:8 204:25 <b>former</b> 184:9 <b>formulate</b> 33:5 122:25 299:12 <b>formulating</b> 25:16 234:16
---	---	---	---

<b>forth</b> 47:17 50:13 73:17 110:2 143:8 151:18 245:17 273:17 277:20 281:19,22 303:14 <b>found</b> 40:22 136:4,10,12 139:13 143:22 159:7 199:13 200:6,13 219:15 249:11 253:10,14,16 253:19 <b>four</b> 23:17 28:4 94:23 115:4 125:17,20 203:15 293:17 <b>fours</b> 154:3 <b>fourth</b> 157:25 <b>free</b> 244:2 <b>freeman</b> 121:17 <b>frequently</b> 36:11 63:14 73:14 84:2,3 94:15 102:9 181:12 182:12 190:3 246:3,18 259:13,17 260:5,21 263:25 266:12 267:10 274:12	<b>fresh</b> 198:5 <b>full</b> 54:7 139:10 139:17 144:7 204:21 262:11 292:5,15 <b>functioning</b> 273:13 <b>further</b> 5:10,14 30:18 298:14 299:18 303:11 303:16  <b>g</b>  <b>g</b> 301:2 <b>gabby</b> 93:21 288:4,18,19 <b>gabriele</b> 2:6 12:20 <b>gasoline</b> 253:19 <b>gastric</b> 80:6 85:25 <b>gastritis</b> 86:4 <b>general</b> 17:9 37:3 79:25 93:6 93:13,15,18 95:16 104:10 114:22 117:13 118:25 119:21 155:20 250:19 250:24 251:3,7 254:13,19 255:16 266:10 295:13 <b>generally</b> 37:12 62:12,16 96:19	136:22,24 181:23 243:22 <b>genetic</b> 63:8,10 63:14,20 65:21 66:9,11,25 67:11,15,16,19 68:7 70:2 88:4 88:10 115:20 115:24 116:17 117:19 118:4 269:19,25 270:21 271:5,7 271:13 <b>genetically</b> 88:14 115:14 271:24 <b>genetics</b> 63:8 63:11,19 64:4 64:15 65:8,11 65:15 67:9 <b>geographically</b> 82:24 <b>georgetown</b> 2:8 <b>germline</b> 65:10 65:11,16,22 66:5 69:24 88:5 <b>getting</b> 115:18 117:12 139:24 150:7 198:8 215:24 219:9 231:8 <b>gilbert</b> 121:23 121:25	<b>give</b> 13:10 15:14 21:13 94:9,21 101:21 167:17 233:20 233:20 242:4 279:6 281:24 <b>given</b> 18:16 150:19 242:10 255:15 298:6 301:14 <b>giving</b> 21:8,20 101:14 <b>go</b> 57:21 68:12 109:10 139:6 139:19 206:8 219:11 268:7 286:4,19 291:8 293:2 <b>goes</b> 30:18 39:21 205:18 252:4 <b>going</b> 17:6 21:24 26:4 36:17 53:10 54:5,6 71:3,4,6 71:7 73:24 77:15 93:9 94:9 102:14 105:6 114:24 115:4 127:24 128:8,8 128:9 143:7 158:24 169:14 182:19,19,22 183:3,9 262:22
---	---	---	---

274:24 299:23 <b>gold</b> 35:4 46:18 48:21 <b>golkow</b> 1:24 12:9 <b>good</b> 16:25 17:12 47:4 48:3 63:23 64:2 72:3 77:16,21 100:12 104:12 136:21 200:22 205:19 <b>government</b> 131:9 <b>grab</b> 246:21 <b>grain</b> 51:10 <b>graphs</b> 202:3 <b>great</b> 93:25 <b>greater</b> 76:9 81:9 116:18 149:9 156:11 164:4 169:20 180:11 186:12 188:2,13,15 214:18 215:15 219:25 228:12 228:23 229:17 230:22 231:13 234:2 235:3 236:5 237:11 <b>grossly</b> 23:20 <b>groundwater</b> 157:22 246:5	<b>group</b> 2:5 49:24 57:10,17 63:23 104:11,12 154:2 170:23 188:3 195:10 259:17 290:17 <b>groups</b> 57:2 177:20 237:21 249:16 <b>grow</b> 68:12 85:11 <b>growing</b> 211:10 <b>grows</b> 85:10 <b>growth</b> 62:21 <b>gsulpizio</b> 2:10 <b>guarantee</b> 70:25 71:9,10 71:12 72:11 <b>guarantees</b> 70:19,24 72:8 <b>guess</b> 19:16 35:19 51:4 82:16 94:10 96:6 98:21 99:25 104:24 110:3 114:13 114:16 129:18 149:17 151:22 166:25 168:23 169:11 204:16 208:14 209:13 214:16 215:15 226:18 253:11 280:21 281:21	282:24 286:5 287:24 <b>guessing</b> 156:5 176:16 <b>guidance</b> 46:2 <b>guidelines</b> 42:2 92:10 241:21 271:11,16,17	<b>h</b>	<b>happening</b> 117:21 <b>happens</b> 85:11 95:23 275:12 <b>happy</b> 24:4 129:23 <b>hard</b> 57:3 59:9 129:21 170:24 192:17 215:25 223:12,13 228:6,7 <b>harder</b> 173:6 174:17 178:8 178:17 180:16 180:21 183:19 <b>harm</b> 63:4,5 <b>hatten</b> 31:3,12 119:2,20 120:2 122:4 123:7 125:7,16 287:3 <b>hatten's</b> 126:11 <b>hazard</b> 169:24 170:9,14,20 171:4,22 172:4 172:6,15 173:9 173:18,21 174:5,9,12,16 175:22 177:17 179:25 180:6,8 181:15 191:10 191:16,21,25 192:13,25 193:12 195:15 196:3
		<b>h</b>	<b>h</b> 6:3 7:3 8:3 9:3 10:3 86:4 <b>hadnot</b> 240:11 240:24 246:15 <b>half</b> 16:2 36:18 36:21 56:21,23 58:14 96:8 172:16 287:21 288:12 <b>hand</b> 13:7 26:5 144:6 160:16 226:22 <b>handed</b> 166:12 184:3 188:23 225:18 247:7 <b>handing</b> 24:25 26:3 52:10 130:15 142:17 148:6 157:13 164:16 168:3 179:3 282:13 <b>happen</b> 74:13 275:11 <b>happened</b> 161:9	

<b>hazards</b> 8:16 164:12,23 <b>head</b> 200:19 267:14 <b>headers</b> 226:22 227:4,6 <b>heading</b> 248:11 <b>health</b> 9:22 14:6,16 64:7,13 146:7 151:14 151:24 241:6 254:2 259:22 260:3,17 261:12 273:17 274:25 275:15 295:6 296:22 <b>heard</b> 100:24 <b>heart</b> 98:9 <b>heavily</b> 125:4 <b>heavy</b> 89:20 245:20 259:11 <b>held</b> 1:17 <b>help</b> 192:21 <b>helped</b> 192:21 <b>hepatocellular</b> 82:7,25 83:13 <b>hepatocytes</b> 82:8,22 <b>hereinbefore</b> 303:14 <b>hereto</b> 5:5 <b>hernia</b> 274:8,17 276:13	<b>hernias</b> 274:12 <b>high</b> 40:18 73:7 84:14 98:2 120:25 125:12 137:2 151:3 162:21 167:9 167:11 169:21 177:19 182:3 185:11,17 186:16,23 188:3,12 195:9 202:7 207:6 225:7,7 237:21 239:2 245:23 267:11 269:13 <b>higher</b> 71:22 86:13 102:14 152:14 163:9 163:13,13 183:11 217:2 220:25 221:2,4 224:22,22 225:2 240:13 241:14,15,23 <b>highly</b> 113:24 <b>hindsight</b> 278:14 <b>hippel</b> 88:16 115:15 <b>histologies</b> 83:8 83:10 <b>historic</b> 157:22 <b>histories</b> 94:15	<b>history</b> 88:11 88:15 90:5 94:17,20 97:16 209:17 270:11 271:4,18,19 <b>hm</b> 132:10 <b>hold</b> 21:18 108:10 162:2 292:9 295:5,8 295:11 <b>holes</b> 136:19 <b>home</b> 73:13 <b>honest</b> 26:18,19 27:2 28:11 32:24 44:10,17 45:2,9 52:15 65:7 100:8 106:17 108:25 109:20,24 117:12,14 119:13 121:15 122:20 126:14 128:15 129:12 129:15 136:9 137:8 141:17 148:10,15 155:9 158:21 158:24 165:24 168:9 176:12 204:14 208:24 209:4 210:10 224:23 240:22 241:10 247:20 258:18 278:16	280:15 281:20 284:5 <b>honestly</b> 19:9 122:12 159:3 <b>hour</b> 17:10 77:16 287:21 288:13 <b>hourly</b> 281:19 284:24 <b>hours</b> 154:15 285:25 286:6 286:12 <b>huge</b> 133:15 <b>human</b> 9:23 254:2 <b>hundred</b> 36:3 36:18 70:12,23 72:9 94:16 263:13 <b>hyde</b> 1:18 12:4 12:13 14:9,10 <b>hypotheticals</b> 215:25
			<b>i</b>
			<b>iarc</b> 129:5,8,18 129:19 <b>idea</b> 47:23 101:7 123:4 <b>identifiable</b> 96:3,9,14,15,20 104:14 105:9 <b>identification</b> 22:6 24:22 25:21,25 52:7

107:8 130:13 142:14 148:3 157:10 164:14 166:8 167:24 178:24 183:25 188:21 225:15 247:2 254:5 282:10 <b>identified</b> 96:15 99:16,18 100:3 128:16 136:4 193:5 <b>identifies</b> 138:8 <b>identifful</b> 96:9 <b>identify</b> 12:18 68:7 90:8 93:3 105:17 128:21 140:12 183:13 202:12 <b>identifying</b> 90:19 <b>idiopath</b> 103:25 <b>idiopathic</b> 99:10,13,14 103:7,8,10,16 103:25 104:8 104:20,21 105:20 <b>idiot</b> 99:11 <b>iii</b> 6:11 24:18 <b>imagine</b> 128:4 291:2 <b>imaging</b> 84:4	<b>immune</b> 68:22 68:22 <b>impact</b> 68:21 75:21 76:14 137:2 152:14 182:4 183:11 183:16 <b>impact's</b> 182:19 <b>imperative</b> 300:13 <b>important</b> 76:13 189:24 <b>impossible</b> 243:25 <b>inches</b> 153:6 <b>incidence</b> 8:15 106:8 109:9 164:12,22 172:12 176:8 189:7,15,18,22 190:10 193:2,7 <b>incidences</b> 100:10 107:18 190:4 192:6 <b>incision</b> 274:10 <b>include</b> 27:13 135:9 <b>included</b> 157:25 168:14 <b>includes</b> 38:14 39:7 112:6 163:16	<b>including</b> 19:2 127:13 180:2 <b>income</b> 280:17 281:8 <b>incomplete</b> 24:13 <b>inconsistent</b> 53:19 54:22 <b>incorrect</b> 24:14 <b>increase</b> 75:19 89:9 97:14 127:12 <b>increased</b> 40:21 64:24,25 75:3 98:19 100:10 138:14 170:14 173:6 176:8 181:14 193:2,6 195:15 196:3 249:22 <b>increases</b> 84:18 85:12 121:2 145:21 <b>independent</b> 33:6,11,15 <b>independently</b> 33:16 202:18 <b>index</b> 4:3,6 11:2 <b>indicates</b> 52:24 <b>indication</b> 181:13 271:23 271:24 <b>indicative</b> 264:17	<b>individual</b> 117:16 124:23 165:25 202:14 263:22 270:22 <b>individuals</b> 36:18 97:9 98:12 163:3 <b>induced</b> 258:9 258:21,25 <b>industrial</b> 189:9 <b>infection</b> 74:20 183:3,6,17 274:11 <b>infections</b> 86:3 <b>inference</b> 36:5 <b>inflammation</b> 84:12,13,17,21 84:25 85:8,14 85:20 86:7,10 86:12,16 <b>influence</b> 59:22 <b>inform</b> 31:12 31:21 32:10 <b>information</b> 74:16 181:8 205:24 223:16 223:25 <b>ingested</b> 204:7 204:12 206:16 207:25 208:6 208:12,17,21 213:13 221:5,7 221:12
--	--	---	--

<b>ingesting</b> 226:15 <b>ingestion</b> 150:14 212:16 212:21,24 213:2 214:21 215:13 226:7 <b>inhalation</b> 212:16,21 215:16,21,22 216:5,9 217:9 217:16 <b>inhaling</b> 249:13 <b>inherent</b> 68:6 <b>inherited</b> 88:6 <b>initial</b> 279:10 <b>inputs</b> 202:23 <b>insignificant</b> 172:5,21 174:8 174:12 <b>insinuates</b> 173:5 <b>instance</b> 36:15 59:7 68:25 69:4 69:23 72:6 73:3 75:3 93:7 102:7 151:19 153:23 182:11 209:25 245:19 <b>instances</b> 59:14 <b>institute</b> 7:13 107:6 <b>institute's</b> 107:12	<b>instruct</b> 279:19 <b>instructions</b> 300:2 <b>insufficiency</b> 73:5 273:9 <b>intake</b> 251:21 252:5,15 253:5 <b>intense</b> 206:25 207:7 <b>intensity</b> 203:18,24 206:9,12 267:11 269:13 <b>interact</b> 294:5 <b>interchangea...</b> 149:23 220:12 228:6 232:2 <b>interest</b> 40:21 <b>interested</b> 303:21 <b>interpret</b> 32:4 33:3 170:19 192:13,16 <b>interpreted</b> 51:9 <b>interquartile</b> 153:14,19,23 154:7 155:2 <b>interrelated</b> 151:7 <b>interval</b> 161:15 172:14 178:7 180:9,16 181:18 186:19	188:7,11,13,14 195:21 266:14 <b>intervals</b> 161:12 174:17 174:22 177:24 180:25 181:11 191:20 194:4 195:16,17 <b>intervention</b> 35:6,15 36:6,7 47:14 48:6,13 56:15 61:18,19 183:18 <b>interventional</b> 47:25 <b>interventions</b> 55:23,25 274:14 276:19 <b>introduce</b> 21:24 <b>intuitively</b> 163:10 <b>invest</b> 294:23 <b>investigator</b> 294:23 <b>invoice</b> 282:20 285:8 <b>invoices</b> 10:6 282:6 285:13 <b>involved</b> 80:10 259:14 281:16 289:19 290:22 <b>involving</b> 291:5 297:15	<b>iodizing</b> 269:14 <b>ionizing</b> 67:8 67:12 <b>island</b> 2:16 <b>issued</b> 119:21 121:6 132:8 <b>issues</b> 37:20 56:10 60:5,11 <b>italicized</b> 29:3 54:10 <b>item</b> 160:6 169:11,19 175:9 179:16 185:22 186:11 187:20,25 194:10,17 203:17 206:9 207:11 228:9 229:16 230:20 232:6,25 233:25 235:4 235:21 236:4 237:9 239:5,16 276:24 <b>items</b> 165:3 173:10,22 184:15,19 185:15 194:25 203:15 222:12  <b>j</b> <b>j</b> 1:17 3:8 4:8 6:14 10:7 13:16 24:20 282:7 301:8,18 303:8
---	--	---	--

<b>january</b> 7:23 8:17 130:12 164:13 285:18 285:22 <b>japan</b> 269:11 <b>job</b> 16:25 <b>jobs</b> 85:4 <b>john</b> 13:25 <b>joke</b> 99:11 <b>jonathan</b> 3:18 12:7 <b>journal</b> 51:21 153:5,9 <b>journals</b> 51:12 137:2 <b>juarez</b> 3:18 12:8 <b>judge</b> 18:22 <b>judged</b> 137:12 <b>judgment</b> 245:15 252:25 <b>july</b> 303:24 <b>jumping</b> 143:8 148:14 <b>june</b> 1:13 9:20 12:10 222:6 246:25 301:11 <b>jury</b> 18:22 <b>justice</b> 3:5 14:23 33:7,9	<b>kid</b> 31:8 <b>kidney</b> 6:12 7:15 9:16 24:19 31:9 80:8,10,19 80:22,23 81:3 87:23 88:7,18 88:24 89:10,18 90:8,11 91:17 91:19 92:7,19 92:25 94:3,11 95:8,10 96:2,8 96:21 97:3 103:19,24 104:14 105:2 105:16,23 106:11 107:7 109:5,9,10,13 109:21 110:21 111:11,23 112:7 113:9,13 114:3,4,9 115:12 117:16 117:18,20 118:2,8,12 120:17 121:3 124:3,18 125:25 126:7 127:2,4,13 135:25 136:5 137:19 139:14 145:10,16,21 147:8,15 157:21 162:19 163:8,22 164:5	170:6,22 172:8 174:6 175:7,23 176:8 177:14 177:25 180:3,6 185:17 186:20 186:24 187:16 188:8 190:13 191:11,17 194:5 199:14 199:18,23 200:6,14 202:15 219:16 225:14 245:18 258:9,21 261:24 262:2,7 263:8 266:7 270:11,15,17 272:22 273:8 273:11,13 274:18,20 275:12,16 276:25 277:2,3 292:22 297:21 <b>kidneys</b> 270:14 <b>kill</b> 63:5 <b>kind</b> 60:10,25 68:9 81:23 85:6 85:10,12 120:7 122:19 124:8 124:22 136:16 156:5 206:21 209:14 215:24 224:3	<b>know</b> 17:11 21:14 23:3,6,13 23:15,21 26:11 27:3 31:6 34:14 35:5 36:3,4,22 45:7,7,10 46:19 46:23 47:12,15 49:25 51:2,6,9 51:9,10,12,21 58:13,15 59:4,6 59:7,8,8,9 61:14,15,21,21 62:6 63:4,6,9 63:18,18,19 64:5,23 65:3,6 65:13 67:7,8 68:12,21 69:5 69:24 70:3,10 71:6,16,23,25 72:2,8,19 73:7 73:9,12,15,20 74:17,19,20,25 75:2,17 80:14 84:11,24 85:16 86:2,3,5,9,10 88:3,5,13,22 89:3,5,5,7,17 89:19,23,25 90:6,7,24 91:17 91:22,22 92:5 93:6,24 94:8,11 94:19,23 95:7 95:17 96:16 98:6,14,14,15
<b>k</b>			
<b>k</b> 232:20 301:2 <b>kathleen</b> 121:23			



99:19,19,20,21	151:10,13,15	232:10,11,12	275:2,3,7,9,19
100:6,9,15,17	152:12,15,21	232:15,21,22	275:22 276:17
101:17,18	153:6 155:8,9	233:14 234:8	277:13 278:10
102:9,12	155:11 156:2	234:13,16	278:12,15,21
103:16 104:3	158:25 159:5,7	235:11,13	278:23 279:18
104:11,25	159:9 161:4,4,8	237:24 238:9	280:3 281:4,18
106:6,8,11,16	163:4,5 170:19	238:20 240:23	281:24,25
107:17 109:11	172:11,16,25	241:16,17	283:11,25
109:18,19,23	173:5 174:25	242:14 243:13	286:5 288:7
109:25 114:9	176:20 181:8	243:18 244:14	290:24 293:11
115:20 117:14	181:14,15,19	244:15,17,19	293:13
117:17 120:7,8	181:20 182:4	245:10,16,18	<b>knowing</b> 91:16
120:9,14,16,24	182:16 189:20	245:24 246:10	243:19 245:10
122:8,9,11,17	189:21,25	247:14 248:6	252:21 254:24
122:18,22	190:5 192:17	248:25 249:3	255:3 256:25
123:4,11,16	196:3 204:13	249:21 250:18	297:9
124:7,20 125:5	204:15,19	250:21,23	<b>knowledge</b>
125:6,7 126:2,4	205:14,18,19	253:9,13,18	19:15 20:2
126:7,15 127:8	205:22 206:17	254:16,19	24:12 28:14
127:10,14,24	206:19 207:5,5	255:21,24	58:11 92:4
128:4,9,14,15	207:8 208:5,13	258:14,16	290:19 296:6
128:16,17,23	208:13 209:17	259:10 260:6	<b>known</b> 31:7
128:25 129:4	210:3,4,10,11	261:4,7,11	36:19 71:23
129:14,20,22	211:12 212:19	262:4,5,24	72:5 85:18 89:9
131:9 133:16	214:12 215:9	263:12,17,23	89:17 91:4
133:18 135:24	215:20 216:6	266:12,25	103:12 104:6
136:14,17,20	217:9,15	267:4,8,8,9,15	105:15,17
136:20,22,24	218:16 222:4,5	267:19 268:15	112:7 117:25
137:6,8 140:6	222:13 223:13	268:16,19	131:11 204:17
141:3,5 143:23	223:21 224:18	269:5,9,10	245:13,18
144:21 146:12	224:20 225:4,9	270:5,9 271:22	292:7,21
146:14 149:17	226:8,14	272:18,23,24	
149:19 150:8	228:17 231:6	273:10,16,22	
150:15 151:3,8	231:20,24	274:10,15,24	



<b>l</b>	<b>lawyer's</b> 304:2	251:4 254:21	132:14 134:18
<b>l</b> 3:10 301:2	<b>lawyers</b> 29:17	255:3,14 278:7	135:9,11
<b>lab</b> 258:13	<b>layperson</b>	279:13 289:10	139:13,20,24
<b>labeled</b> 168:11	62:19	290:22 296:9	140:4,8,9,21
<b>lack</b> 49:5 52:24	<b>lead</b> 85:17	296:15	141:4 149:3
53:6 54:18	120:12	<b>length</b> 176:19	165:25 167:6
55:19	<b>leads</b> 67:18	176:20,22	168:23 177:18
<b>large</b> 172:11	70:6 101:23	206:14	194:24 199:22
181:24 190:2	<b>leak</b> 74:19	<b>lesion</b> 270:18	200:5,13 205:4
<b>larger</b> 76:16	<b>leastly</b> 31:15	<b>letter</b> 296:13	217:5 219:14
182:7 211:2,2,3	<b>left</b> 82:13 144:6	<b>level</b> 61:9 71:4	227:13 234:8
211:4	151:12	97:23 98:2	240:12 241:5
<b>lastly</b> 226:10	<b>legal</b> 2:5 12:8	102:14 124:9	241:14 243:14
<b>latencies</b> 267:3	19:2,8 280:19	124:10 126:24	244:7,21 245:6
267:24,25	280:23 281:5	141:8 143:3,13	249:6,22
268:18	281:22 290:17	143:16 149:7	<b>life</b> 113:14
<b>latency</b> 128:7	<b>lejeune</b> 1:7	152:9 157:25	114:10 115:5
145:11,17,22	7:21 12:15 29:4	158:8 163:6,9	243:15 244:2
146:6,13,17,25	29:10 33:7,9	168:25 175:21	262:4 263:21
147:9,16	120:22 130:11	196:9,23	273:5 295:15
262:10,18	130:24 131:11	198:12,15	<b>lifelong</b> 88:20
263:2,4,7,19,25	131:21 132:2,6	202:4 207:6	274:24
264:16 265:2,4	132:9,15	209:3 219:19	<b>lifespan</b> 113:17
265:13 266:5,8	134:18 135:3	225:4 235:15	<b>lifestyle</b> 274:16
266:17,25	135:11 166:21	241:22 242:10	<b>lifetime</b> 106:10
267:2,7,9 268:8	169:4 176:23	243:22 245:11	110:5,17,21
268:25 269:5	179:14 184:12	246:10 249:18	111:13,24
269:10	189:11 191:6	249:24	112:5 113:8,12
<b>law</b> 54:20 173:3	192:8 193:14	<b>levels</b> 31:6	114:23
290:13	193:18,24	102:12 120:15	<b>lightheaded</b>
<b>lawsuit</b> 14:24	199:13,17	121:7 123:8,12	57:13
259:15,16	214:8,9,19	123:16,21,22	<b>likelihood</b> 61:5
<b>lawyer</b> 32:25	216:15 223:8	124:2,7,8,17,25	61:9 76:6
	229:6,19 245:3	131:10,21	113:20 115:22

116:8,15 117:8 271:6 275:10 <b>likely</b> 29:14 30:14,19 31:15 31:24 32:12 33:12,17 43:20 43:25 45:16,21 46:3 59:22 60:7 60:11,18,19 61:4,15 100:13 161:8,8 182:7 202:14 230:12 240:9 264:7,12 268:10,22 293:3 <b>limited</b> 181:2 181:16 <b>limiting</b> 37:23 <b>lindau</b> 88:16 115:15 <b>line</b> 11:5,10,15 11:20 55:8 56:10 156:13 170:5 209:5 233:17 292:16 302:3 304:3 <b>linear</b> 101:17 <b>lipworth</b> 138:15 <b>list</b> 6:13,19,23 24:20 25:10,20 25:24 27:21 28:17 73:18 91:13 108:6	139:21 140:21 141:3 143:13 200:4 <b>listed</b> 28:2,16 74:8 135:25 139:13 199:10 200:15 203:15 219:15 <b>listing</b> 257:17 <b>lists</b> 25:13 <b>liter</b> 169:2 204:18 207:13 207:17,18,19 218:18 220:14 226:6 227:14 227:23 228:12 228:23 229:5 229:18 230:14 231:24 234:21 236:17,22,25 237:9 238:18 <b>literally</b> 152:24 224:20 <b>literature</b> 31:6 35:10 42:18,23 43:4,7,10,15,19 43:22,24 44:5 44:18 45:15 50:12 72:13 92:6,6 94:19 102:17 128:22 128:25 129:16 135:2,6,8 137:25 140:24	141:4 228:4 232:20 234:14 235:16 237:18 294:3,8 295:17 295:22 296:2 <b>liters</b> 252:9 <b>litigation</b> 1:7 3:7 12:15 15:22 44:20,22 45:14 59:17,19 258:23 259:15 278:7 279:13 279:24 284:12 289:10,25 290:2,23 <b>little</b> 23:19 28:3 35:18 62:5 66:17 67:6 75:5 127:8 137:3 141:16 150:5 163:12 176:14 228:17 238:16 257:12 280:20 <b>live</b> 243:25 <b>liver</b> 72:25 73:9 79:17,21,24 81:8,12,16,19 81:19,20,20,23 81:24 82:2,3,5 82:11,18,21,22 82:23 83:2,11 83:18,19 84:8 84:10,15,17,18 84:21,25 85:4,9	86:18 273:9 <b>liver's</b> 85:2 <b>lives</b> 98:6 <b>living</b> 205:15 205:16,22 223:18,21,22 <b>local</b> 76:14,17 80:23 <b>localized</b> 109:21 <b>located</b> 14:12 82:17,20 <b>location</b> 82:12 <b>lodge</b> 93:21 <b>long</b> 38:9 43:6 75:21 146:13 151:5 165:16 165:23 177:6 222:14 223:18 273:23 276:5 287:18,19 288:10 <b>longer</b> 152:11 152:12 263:20 264:6 268:18 <b>look</b> 23:2 27:23 38:20 54:7 84:3 88:14 89:15 91:3,4 103:12 109:25 114:2 114:20,21 117:23 128:10 129:13,23 132:5 136:17
---	---	--	---

136:18,19 154:3 159:15 159:21 176:12 190:3 231:22 232:19 262:4 <b>looked</b> 32:10 88:17,22 89:2,3 101:24 120:14 141:4 153:4 195:20 271:17 271:18,20 <b>looking</b> 31:11 32:3,5 35:12 39:23 41:21 48:4,11 57:19 61:14 75:9 90:6 91:21 96:13 102:12 105:7,9 105:15 108:11 111:2 129:16 131:20 133:14 134:22 135:3 139:15 153:22 153:25 160:20 165:11 175:8 177:2,3 190:19 221:16 223:5 226:2 228:9 230:20 231:19 239:5,16 249:21 270:13 270:16 <b>looks</b> 120:20,21 227:17	<b>loss</b> 75:4 <b>lot</b> 14:15 28:10 71:14 80:19 84:16 95:2 99:8 99:14 100:24 106:18 107:19 109:9 115:12 119:13 124:20 124:23 129:14 129:17 149:21 172:11 183:4 220:11 223:24 247:12 259:6 266:11 280:2 287:5 <b>lots</b> 51:11 62:2 64:24 71:17 80:2 88:13 100:8 136:15 244:10 267:7 <b>lovenox</b> 210:19 <b>low</b> 97:23 98:12 124:9 162:20 163:6 167:9,11 172:12 177:19 185:10 188:14 202:6 207:12 237:21 238:25 245:11 246:10 249:6 251:2 271:6 273:3 275:10 <b>lower</b> 76:17 115:10 124:9	151:5 163:11 163:11,12 183:11 215:4 <b>lowers</b> 57:9 <b>luck</b> 63:24 64:3 <b>lump</b> 75:25 <b>lumps</b> 105:18 105:18 <b>luncheon</b> 201:2 <b>lung</b> 79:16 98:7 106:13 266:12 <b>lying</b> 251:2 <b>m</b> <b>m</b> 2:14 301:2 <b>m.d.</b> 1:17 4:8 13:16 301:8,18 303:8 <b>made</b> 88:14 125:10 207:21 232:19 260:15 300:9 <b>magnitude</b> 230:6 <b>main</b> 85:3 <b>majority</b> 261:25 <b>make</b> 32:5 34:16 71:20,20 73:11 133:16 133:18 170:19 170:24 235:20 238:16 244:18 261:3 264:25 277:19 293:25	299:14 300:5 <b>makes</b> 21:11 70:5 100:19 170:23,24 171:15,16 174:17 178:8 178:17 180:16 180:21 181:20 215:17 <b>making</b> 58:16 60:7 193:23 260:18 <b>malignancies</b> 80:15 127:13 259:7 <b>mallon</b> 119:9 119:11 <b>malpractice</b> 289:23 290:3 <b>managed</b> 275:17 <b>mandel</b> 2:12 <b>mandell</b> 2:13 2:13,14 12:21 278:10,21,22 278:23,24,25 279:2,12 288:3 288:18,19 290:13 <b>mandells</b> 282:21 288:6 <b>manner</b> 10:12 <b>manuals</b> 213:12
---	--	---	---

<b>marcus</b> 1:18 12:13 14:10,19	254:4 282:10 282:14	<b>maximal</b> 102:13	114:20 117:11 118:16 120:4
<b>margin</b> 75:11 76:2,5,15,16 77:9 78:15	<b>market</b> 102:8 102:19	<b>mba</b> 1:17 4:8 10:7 13:16 282:8 301:8,18 303:8	121:9,14 122:8 124:19 126:14 127:20 131:24 133:13 135:22
<b>margins</b> 75:18 76:8,10,12,13 77:7	<b>mass</b> 150:13,14 207:25 208:5 208:12,17,21 209:4 221:5,7 221:12	<b>mcl</b> 240:19 <b>mcls</b> 240:14 242:14	138:25 140:3 143:21 146:8 149:16 150:7 150:10,24 152:3,8,10 155:19,25
<b>marie</b> 1:20 13:4 303:3	<b>massachusetts</b> 141:11 142:24	<b>md</b> 10:7 282:7 <b>meals</b> 205:17 <b>mean</b> 16:11 17:21 19:16,17 19:18 20:24 21:11 23:8 27:3 32:24 41:13 46:12 47:11 49:11,23 50:11 50:12,20,25 51:5 55:6 56:3 56:20,25 57:2 60:23 61:12 62:20 63:11 66:10,24 67:25 69:21 70:23 74:14 77:13 82:19 83:25 85:25 87:20 89:14 92:21 94:7 96:25 99:18 100:6,22 102:6 104:20 106:3,17 109:18,21	158:20 160:3 162:9,11,25 163:10 165:20 165:23 170:13 171:3 172:6,15 174:9 175:5 181:6 183:15 189:19 190:18 192:5 195:14 196:12 207:4 208:11,25 209:3,21 214:24 215:5 215:20,21 216:21,24 218:14 220:21 224:16 229:9 232:9,13 233:12 235:7 237:12 238:8 238:12 241:15 241:16 242:16
<b>marine</b> 166:20 184:12 189:10 204:6,10 226:9	<b>material</b> 122:24 123:2		
<b>marines</b> 166:19 184:9 189:8 191:7 193:24 193:24	<b>materials</b> 6:13 6:19,22 24:19 25:19,23 27:21 28:15,16 89:6 100:15 280:5,9 294:13		
<b>mark</b> 167:16 278:23 288:8	<b>math</b> 114:25		
<b>marked</b> 11:19 22:5 24:21 25:2 25:20,24 26:4,5 52:7,11 107:8 130:12,16 142:14,18 148:3,7 157:10 157:14 164:13 164:17 166:8 166:13 167:23 168:4 178:24 179:4 183:24 184:4 188:20 188:24 225:15 225:18 247:2,7	<b>mathematical</b> 101:18,22,25 102:3,22 103:2 <b>matter</b> 12:14 72:10 84:12 208:16 <b>matthew</b> 1:17 4:8 6:14,17,21 10:7 12:16 13:16,25 24:20 25:18,22 282:7 301:8,18 303:7 <b>mature</b> 127:8 127:16,17,21		

243:18 244:9 245:9 247:11 247:17 248:5 249:10 250:16 252:20,21,24 261:25 262:23 263:11 267:7 269:7 270:5 271:15 274:21 276:10 278:3 280:9 293:8,10 295:14 297:2,8 <b>meaning</b> 16:12 16:13 35:11 51:2 65:16 75:22 88:4 103:11,16 162:12 293:24 <b>meaningful</b> 145:21 172:15 <b>means</b> 16:11,21 31:16,24 32:13 41:15,18 49:10 90:11 103:9 104:21 111:21 173:3 <b>meant</b> 90:25 266:4 <b>measure</b> 154:14,17 177:5 196:24 208:20 245:15 258:15	<b>measured</b> 196:6 209:9 240:12 <b>measurements</b> 190:6 <b>measures</b> 232:22 <b>measuring</b> 47:9 60:20 150:4 151:10 209:14 <b>mechanism</b> 68:6 120:10 <b>mechanisms</b> 68:16 121:7 123:11 <b>mechanistically</b> 65:3 <b>media</b> 156:8 <b>median</b> 152:15 154:6,23,24 155:16 161:25 <b>medians</b> 153:15 <b>medical</b> 26:24 32:5 33:17 34:12 35:10 42:17,23 43:4,7 43:10,14 44:18 49:6 51:14 53:7 54:19 56:3 58:4 58:8,17,22 59:10,16,24 60:9,13,18 61:7 61:8,22 62:3 71:18 73:10,19	90:5 91:2,20 104:5 125:13 260:7,16,24 271:18 276:24 277:10,23 280:10 281:5 286:24 289:22 290:3 295:16 <b>medically</b> 272:9 277:4 <b>medication</b> 15:18 57:8 <b>medications</b> 210:14 211:11 211:13 <b>medicine</b> 7:8 34:20 36:11 52:4 91:23 99:9 126:4 295:9 <b>medium</b> 167:9 167:11 169:12 177:19 185:11 185:16 186:6 186:20 187:22 188:8 202:6 <b>meet</b> 287:9,15 288:15 <b>meeting</b> 279:10 287:19 288:10 288:16 <b>meetings</b> 287:17,18 288:23	<b>member</b> 194:18 <b>members</b> 50:2 120:22 <b>memory</b> 119:17 287:6 <b>men</b> 111:10 113:8 <b>mental</b> 273:17 274:25 <b>mentioned</b> 118:20 135:15 212:8 256:9 261:17 269:17 280:25 286:11 <b>merit</b> 1:21 137:13 303:3 303:23 <b>met</b> 287:23 288:7,9 <b>meta</b> 40:12,14 40:19 <b>metabolites</b> 258:16 <b>metabolized</b> 120:11 210:25 294:9 <b>metabolizing</b> 210:5 <b>metals</b> 89:20 245:20 259:11 <b>metastasize</b> 62:23 63:3 68:13
--	--	--	---

<b>metastasizes</b> 81:22 <b>meters</b> 251:20 <b>methodology</b> 112:11 <b>methods</b> 35:9 <b>metric</b> 208:7 232:13 <b>metrics</b> 149:22 190:22 <b>michael</b> 121:17 <b>microgram</b> 204:18 207:19 215:12 <b>micrograms</b> 169:2 207:13 207:17,18 213:13 214:20 218:18,22 220:14 227:14 227:22 228:12 228:23 229:5 229:18 230:14 231:24 234:20 236:17,22,25 237:8 238:17 251:22 252:7 252:17 253:6 <b>middle</b> 29:2 106:20 138:12 153:25 251:13 279:4 <b>military</b> 223:21	<b>milligram</b> 144:13 <b>milligrams</b> 143:14,24 150:14 151:11 217:22 218:5 218:22 219:20 <b>mind</b> 122:19 125:9 192:22 <b>minute</b> 77:19 <b>minutes</b> 242:3 <b>misnomer</b> 81:24 <b>misunderstood</b> 196:19 266:3 <b>mixed</b> 83:14 <b>mm</b> 132:10 <b>mode</b> 68:19 69:6 <b>model</b> 101:22 213:20 <b>modeling</b> 203:19 213:23 <b>models</b> 101:25 102:22 103:3 <b>moderate</b> 124:9 207:5 237:21 239:2 <b>modifiable</b> 75:8 <b>monograph</b> 129:9 <b>monotonic</b> 40:15	<b>month</b> 23:15,17 169:3 175:17 207:13,19,19 209:11 218:18 227:14,23 228:13,24 229:5,18 230:15 233:7 234:21 235:21 236:17,22 237:2,9 238:18 <b>months</b> 23:25 175:10,24 176:6 177:8 185:24 186:13 187:22 188:2 204:18 220:14 230:22 231:13 231:20,24 234:3 235:3 236:6 237:11 238:18 239:7 <b>moore</b> 8:9 148:2,18 152:17 154:12 156:10 158:13 <b>morbidity</b> 184:9 195:11 233:2,8 238:4 <b>morning</b> 199:9 <b>mortalities</b> 175:7 <b>mortality</b> 72:7 72:9,20,24 73:8	74:4,6,12,21 75:13,14,19,24 109:4 124:12 166:18 168:14 170:17 189:12 189:17,19 <b>multifactorial</b> 69:9,18,23,25 70:5,8 <b>multiple</b> 22:12 89:7 127:13 248:6 270:16 <b>multiplied</b> 209:9 <b>mutation</b> 65:17 65:22 66:4,11 66:13,23 67:2 67:17,19 69:24 88:6 <b>mutations</b> 63:10 65:11,13 65:16 66:9,25 67:15 68:4,14  <b>n</b>  <b>n</b> 2:2 3:2 172:12 174:23 175:4 181:11 182:2,8 201:4,4 201:4 301:2,2 <b>name</b> 12:7 13:24 14:3,21 119:19 121:19 <b>names</b> 119:15
--	---	--	--

<b>narrow</b> 77:6	<b>need</b> 17:25	<b>nonsmoker</b>	181:10 183:5
<b>narrower</b> 76:10	89:15 111:5	88:19,20,21	183:12 190:2
<b>nathan</b> 3:8	129:13 133:18	<b>normal</b> 57:10	219:9 229:14
12:23 14:21	144:16 181:25	210:3 262:20	233:20,21
24:3 241:25	182:7,22	263:7,24 265:2	257:17
<b>nathan.j.bu</b>	183:12,13	265:3,15,20,24	<b>numbered</b>
3:14	210:14,22,25	266:4,16,24	108:4 152:22
<b>national</b> 7:6,13	211:3 276:7	267:24 268:2,9	152:23
49:19,21,23	<b>needed</b> 42:4	268:21,25	<b>numbers</b>
50:8,14,17,22	<b>needs</b> 54:8	<b>north</b> 1:3	170:24 173:4
51:17 52:2 53:4	128:12 137:12	<b>northwell</b> 14:6	181:17 237:16
53:18 55:3	275:16	14:16	237:18 246:12
107:5,12	<b>neither</b> 303:16	<b>northwell's</b>	<b>nuts</b> 250:23
<b>nationwide</b>	303:19	14:9	<b>nw</b> 3:10
107:16	<b>nephrectomy</b>	<b>notary</b> 1:22	<b>o</b>
<b>natural</b> 69:2	272:15,16	5:16 13:18	<b>o</b> 201:4,4,4
<b>navy</b> 166:19	277:14	301:24 303:5	301:2
189:8 191:7	<b>never</b> 34:21	303:24	<b>oath</b> 18:16
<b>nearly</b> 223:16	98:2 290:14	<b>note</b> 10:12 34:9	301:11
<b>necessarily</b>	<b>new</b> 1:18,18	45:10	<b>obese</b> 65:2,6
10:13 33:22	12:4,4,13,14	<b>noted</b> 299:25	95:23
34:7,14,22	13:19 14:9,10	<b>notes</b> 304:2	<b>obesity</b> 64:8,13
46:25 64:22	14:11 46:21	<b>november</b>	64:16,19,20,23
76:24 77:8,9	47:2,10 102:8	282:21	65:4 95:7,9,14
81:18 82:12	102:17 108:16	<b>noxious</b> 69:4	95:15,19,20
84:23 97:2	259:5 303:5	85:15	<b>object</b> 20:11,22
98:25 123:3	<b>nidus</b> 267:18,22	<b>number</b> 25:2	21:9 29:22
146:11 151:10	<b>nine</b> 165:22	52:11 108:21	30:10 31:17,25
151:12 181:6	<b>non</b> 40:15	114:21 118:18	32:22 33:24
264:15,17	194:12	127:19,22	34:25 37:6,15
268:14,15	<b>noninferiority</b>	128:19 140:7	48:7,14 49:7
<b>necessary</b>	46:17 47:6,8,24	149:7 158:6	53:8,21 55:4,15
66:13 272:9	48:4,11,24	167:18 172:9	56:18 60:2,21
277:5 300:5		175:5,6,6,7	61:10 66:6,15



67:4 69:10,19 70:15,21 77:11 78:22 79:6 90:22 91:8 92:12 93:4,16 94:5 96:4 97:4 97:10,17 99:3 100:4,20 103:21 105:3 105:10,14,25 109:16 110:8 111:14,25 112:14,20 113:5 114:18 115:8 116:2,9 116:25 117:9 118:14 122:6 127:5 129:10 131:6,22 132:16 133:4 133:11 134:4 134:11,19 136:7 140:15 140:22 143:19 144:14 145:13 145:24 146:20 147:18 150:22 152:6 154:9 155:14,18 157:4 158:3,18 159:12 160:11 161:23 162:23 163:23 165:5 165:18 170:11	171:12 172:23 173:25 174:19 175:13 176:10 178:12 179:18 181:4 185:25 190:16 192:9 194:13,20 195:3,12,23 196:10,16 197:4,11,15,23 198:3,20 199:4 199:15,24 200:8 203:6 207:2 208:9 213:5,15 214:22 215:18 216:19 217:12 218:6,23 219:5 220:8,22 222:2 222:8,25 223:10 224:9 224:14 227:15 227:24 228:14 229:7,20 230:8 230:16,24 232:7 233:10 234:4 235:5,23 236:7 237:13 238:5 239:8 241:8 242:11 243:16 244:22 245:7 246:6 248:17 249:8 250:14 252:11	252:18 254:22 255:6,25 258:5 261:8,14 263:9 264:13,20 267:5 268:3,12 277:25 279:15 280:13 281:14 283:14,21 284:14,20 287:11 290:4 296:24 297:6 298:2 <b>objection</b> 34:10 69:16 93:21 286:15 <b>objections</b> 5:11 <b>observational</b> 224:4 <b>observed</b> 138:14 <b>observes</b> 40:15 <b>obviously</b> 14:15 73:3 89:3 90:4 96:12 106:11 120:18 136:14 182:25 245:16 270:7 272:15 273:18 285:25 293:11 <b>occasionally</b> 102:6 <b>occupational</b> 295:9	<b>occur</b> 68:7 83:2 115:4 <b>occurring</b> 40:18 75:6 95:22 258:14 <b>occurs</b> 68:4 83:3 246:18 <b>october</b> 9:12 188:20 189:7 222:5,5 <b>odds</b> 160:16,21 161:2,3,6,21 162:6,9 185:5 187:11 <b>offer</b> 15:19 70:19 245:2 <b>offered</b> 45:19 297:14,20,24 <b>offering</b> 15:3 92:18 120:2 <b>oh</b> 123:3 284:7 <b>okay</b> 14:14,20 16:7,13,17 17:8 17:17,24 18:4 18:14 19:6 20:4 21:2,17,23 22:10,17,21,25 23:16 24:2,11 24:24 25:3,17 26:6,13 27:5,12 27:17 28:13,21 28:24 29:16 30:20 31:10 32:8 35:17 37:9
--	---	--	--



37:18 38:2,18	109:3 110:15	156:14,15	197:18 198:9
38:24 40:25	110:19 111:7,8	157:15 158:7	198:23 199:7
41:6,25 42:7,21	111:17 112:4	159:17,19	199:20 200:11
44:8,15,23 45:3	112:17 115:11	160:5,14,21,23	200:20 201:22
45:18 46:9 47:7	116:4,20	161:10,14	202:11,21
48:23 49:15	118:10,19,24	164:2,18,25	203:2,11,22
51:22 52:18	119:7,12,23	165:15 166:3	204:20 205:25
53:3,16,24 54:3	121:4,21	166:10,23	206:7,23
54:13,24 55:12	123:19,25	167:12 168:5	207:23 208:4
56:12 57:20,23	124:14 125:22	168:10,17	208:15 209:7
58:6,19,25	126:19,23	169:6,16 170:8	210:12 211:7
59:12,20 60:14	129:7,24 130:5	171:7,18 172:2	212:3,7,13
61:2,24 62:4,10	130:17,20	172:19 173:8	213:18 214:5
62:14 64:6,11	131:16 132:7	173:14 174:4	214:11,15
64:18 65:19	132:11,24	174:14 175:3	215:7 217:3,20
66:21 67:20	133:7 134:8	175:16,20	218:19 219:2
71:11 72:12,17	135:7,14 136:2	176:25 177:9	219:17,22
72:22 73:25	137:10,14,16	177:12,16,23	220:3,17
74:10,24 75:20	137:20,22,23	178:6,15 179:5	221:10,19,23
76:4,21 77:4,15	138:6,11,18,22	179:21,24	222:11,17,20
78:13,18 79:19	139:3,8,23	180:5,14	223:4 224:5,24
81:14 82:15	140:11,19	181:22 182:5	225:10 226:16
83:6 84:6 85:19	141:7,12,18	184:13,18,25	226:20 227:4
86:17 87:24	142:25 143:10	185:4,9,14	227:11,19
89:11 90:9,14	143:25 144:4	186:10 187:2,9	228:8,21 229:3
91:12 92:9,15	144:11,22	187:15,19	230:4,19 232:4
93:12,19 95:6	145:19 146:4	188:6 189:3	232:23 233:4
96:18,23 99:24	146:16 148:17	190:12,23	233:22 234:18
101:9 102:20	148:24 149:6	191:4,15,24	235:18 237:6
102:25 103:5	150:2,17	192:23 193:8	237:25 238:14
104:23 105:21	151:21 152:19	193:11,16,21	239:4,23 240:4
106:5,15,24	153:8,18	194:3,8,23	240:7,17 241:3
107:14,21	154:16,20	195:6,18	241:12 243:12
108:5,8,19	155:5,10,24	196:13 197:7	243:20 244:25

246:2,16,20 247:4,19,21,25 248:9 249:4,25 250:8 251:9,12 251:16 253:2 253:17,22 254:17,25 255:19 256:7 256:12,20 257:5,14,23 259:3,20,24 260:13 261:22 262:17 264:3,9 265:6,9 266:19 268:6 269:4,22 270:24 271:10 272:2,5,11 274:7 275:21 276:9,16,22 277:8,21 278:5 281:9 282:3,15 282:23 283:6 283:10,17,24 284:11,17,22 285:12 287:7 287:14 288:5 288:20 290:15 291:3,21 292:4 292:14,18 294:7,11,11,17 295:20 296:11 297:18 298:13 299:6	<b>old</b> 47:3,4 63:24 64:2 <b>once</b> 94:22 281:6 <b>oncologic</b> 75:22 <b>oncologist</b> 79:25 <b>ones</b> 36:21 83:16 122:14 122:19 200:15 <b>operate</b> 80:12 <b>operation</b> 72:7 72:10 73:15 74:5 75:4 <b>operations</b> 71:23 <b>operative</b> 73:11 73:24 74:3,6 <b>opine</b> 272:6 276:23 <b>opining</b> 277:22 <b>opinion</b> 25:16 29:25 32:6 33:5 60:12 61:20 63:16 116:6 117:6 123:2 159:7 163:20 164:3 205:2 221:7 233:23 271:21 297:14 297:20,24 299:12 <b>opinions</b> 15:3,4 24:8,13 45:19	58:2,15 92:18 92:24 113:19 114:14 119:25 125:16,20 245:2 299:14 <b>opportunity</b> 18:6 <b>opposed</b> 150:12 170:17 190:4 220:13 <b>opposing</b> 273:10 <b>optimize</b> 71:7 <b>order</b> 36:13 67:9 152:25 182:22 <b>ordinary</b> 114:14 <b>organic</b> 89:8 96:17 127:11 135:24 226:4 229:13 <b>organs</b> 85:9 <b>oriented</b> 82:24 <b>original</b> 4:16 272:14 300:14 <b>outcome</b> 35:16 47:13 48:3 55:24 56:2 71:8 75:5,22 101:15 <b>outcomes</b> 107:18,20 191:6	<b>outdated</b> 23:19 <b>outlandish</b> 41:22 <b>outlier</b> 114:8 <b>outpouching</b> 274:9 <b>outside</b> 59:16 250:19 258:23 267:25 268:9 268:21 <b>overall</b> 76:15 133:2 175:22 210:9 <b>overestimate</b> 215:13 216:17 217:7 <b>overestimated</b> 212:25 217:4 <b>overestimates</b> 214:20 <b>overestimation</b> 215:5 <b>own</b> 68:15 91:11 125:23 126:8 268:19 293:12,18 <b>oxygen</b> 73:13
			<b>p</b>
			<b>p</b> 2:2,2 3:2,2 161:25 162:13 178:4,16 180:11,20 198:12

<p><b>p.m.</b> 200:24 201:7 242:21 242:25 291:10 291:14 299:20 299:25</p> <p><b>pack</b> 94:21 97:14,15 209:13,17</p> <p><b>page</b> 4:4,12 6:5 7:5 8:5 9:5 10:5 11:5,10,15,20 28:22 39:11,18 39:20,25 53:25 87:14 108:2,12 110:13 111:6 132:22 133:14 133:17 137:17 137:19,21 139:7,7,18 143:12 144:2 144:24,24 149:3 152:21 156:10 158:2 159:18 165:3 166:25 170:2 171:19,21 173:16 177:11 179:22 185:2 187:4,7,8,9 190:24 191:3 193:9 203:9 204:22 219:11 226:19 239:17 239:21 240:2</p>	<p>248:7 250:2 251:10 256:5 256:15 262:9 262:13,14,15 265:7 270:25 271:3 272:3 276:23 292:2 292:11,13 302:3 304:3</p> <p><b>pages</b> 187:5 282:25</p> <p><b>pancreas</b> 79:24 80:18,21 86:13 86:14 267:18</p> <p><b>pancreatic</b> 79:21 81:8 86:8</p> <p><b>pancreatitis</b> 86:12 267:15 267:16</p> <p><b>paper</b> 44:9,19 120:20 124:11 137:6,9,11 142:22 143:22 154:11 158:5 166:2 170:16 189:6 232:20 237:22 239:3</p> <p><b>papers</b> 23:14 23:14,17 26:10 50:14 107:19 120:19 123:18 124:6 128:20 141:2 151:15 152:25 153:3</p>	<p>202:8 286:21</p> <p><b>paragraph</b> 29:2 138:7 139:10 139:17 144:7 144:12 145:5 203:12 204:22 239:24 251:13 271:2 292:5,15 292:16</p> <p><b>park</b> 1:18 2:15 12:4,13 14:9,10</p> <p><b>parker</b> 222:12 222:15,23 223:5,9 224:7</p> <p><b>part</b> 38:19,25 39:7 44:20,21 44:22 45:13 50:9 62:17 69:12 82:22,23 83:2 101:25 149:12,14,16 149:22 167:16 213:20,22 214:6 251:25 259:13 260:5,6 260:10,20 275:15</p> <p><b>particular</b> 52:14 68:21 80:11 97:20 102:18 122:3 123:13 148:12 237:19 247:16 257:7</p>	<p><b>particularly</b> 247:13</p> <p><b>parties</b> 5:5 303:18</p> <p><b>parts</b> 82:18 149:23 150:11 155:6,11,12 162:14 163:16 163:21 164:4 220:12,19 224:13,18 225:2 231:20 235:16</p> <p><b>pass</b> 245:15</p> <p><b>passing</b> 252:25</p> <p><b>patient</b> 46:24 47:21,22 71:16 74:7 76:24 77:5 91:24 95:22 97:2,14,16 98:22 118:3,7 118:12 211:2,4 258:24 270:6 270:12 296:7</p> <p><b>patient's</b> 91:5</p> <p><b>patients</b> 59:6 63:20,25 64:2 65:2,5 70:20,25 71:2,16 73:21 74:18 80:14,19 80:23 84:14 86:11 94:15 95:17 97:22,25 98:18 100:11</p>
---	--	--	---

103:19 104:12 104:12 114:3,8 115:14,14 120:21 128:13 128:21 163:6,8 172:10 182:21 183:5,10,12 189:20,21 190:2 259:6,13 259:17,25 260:4,9 269:12 273:4,18 274:12,22 275:2 276:4 <b>pause</b> 93:20 <b>pce</b> 36:19 120:9 127:3,9 129:9 129:17 133:9 133:25 134:2,6 134:6 135:21 136:5 137:25 138:7 140:7 143:14,24 169:8 173:19 174:6 184:22 187:12,22 188:2,8 195:10 211:18 217:23 218:5,12 219:21 224:12 225:8 226:23 227:12 228:24 230:23 236:3,6 236:12,16,21	237:9,11,19 240:13 242:15 244:16 263:15 292:7,20 293:14 296:3 297:4,16 <b>peer</b> 43:3,7,9 43:14,18,21 50:21 51:20 62:7,15 123:17 136:23,25 137:3 295:22 296:2 <b>pelvis</b> 7:15 107:7 110:21 111:12 <b>penalty</b> 18:17 <b>pending</b> 17:5,5 <b>pendleton</b> 191:7 193:19 193:25 <b>people</b> 49:25 73:11,23 98:5 99:20 111:22 113:14,15 149:21 159:4 209:19 220:12 223:17 243:13 243:21 254:13 262:2,8 267:16 268:16,17 <b>percent</b> 36:3 70:12,19,24 72:7 81:2,6,7	81:10 93:7,8,10 94:12,16 104:13 106:9 106:10 110:5 110:22 111:10 112:3,5,19,23 113:4,10,18 114:7,22,23 115:3,6,7,19,25 116:7,23 117:22 118:17 144:8 154:2 183:3,4,7,8,17 188:10 195:16 196:2,8,23 197:9,14,22,24 198:11,15,16 255:23 262:7 263:13 280:22 280:22 <b>percentage</b> 92:19,25 94:3 95:12 96:2 114:6 <b>percentages</b> 93:9,14 94:10 <b>percentile</b> 144:21 <b>perfect</b> 17:24 <b>perform</b> 36:12 71:21 <b>performed</b> 284:13 285:17	<b>performing</b> 277:13 <b>period</b> 127:23 127:25 128:7 128:20 152:11 208:16 225:5 262:10,19 263:2,4,7,19 265:13 266:9 269:2,10 <b>periods</b> 266:17 267:8,9 269:6 <b>perioperative</b> 74:12 <b>peripheral</b> 98:8 <b>perjury</b> 18:17 <b>permanent</b> 274:19 <b>permanently</b> 63:4 <b>personal</b> 64:7 64:12 <b>personally</b> 55:9 104:15 276:3 <b>personnel</b> 120:21 166:19 179:11 189:8 <b>perspective</b> 122:11 <b>pertains</b> 104:4 <b>pertinent</b> 209:22 <b>ph</b> 1:25
--	--	---	---

<b>phase</b> 6:10 24:17 102:9 <b>phone</b> 2:9,17 3:13 288:14 <b>phrase</b> 58:20 59:2,5,15 100:18 101:2,3 101:6 <b>physically</b> 82:16 <b>physician</b> 14:5 19:11,22 20:6 20:16 21:6 42:18,23 50:10 70:18 <b>physician's</b> 21:4 <b>physicians</b> 18:25 19:7 58:12,14 289:15 <b>place</b> 12:12 68:8 77:17 125:9 200:22 303:14 <b>places</b> 80:16 <b>plaintiff</b> 2:4 6:15 12:22 24:21 <b>plaintiffs</b> 6:9 24:16 140:18 279:23 281:11 283:13 285:14 286:8 288:22	289:2,9 290:21 290:25 <b>plant</b> 246:15 <b>play</b> 67:21 68:17 <b>please</b> 12:18 13:8,23 28:23 29:7 39:12 51:23 54:2,15 57:22 87:15 108:3 110:14 130:7 132:23 137:18 147:23 157:7 164:9 178:21 179:23 183:22 188:18 190:25 193:10 203:10 213:8 225:12 248:8 251:11 253:24 265:8 272:4 282:5 292:3 300:4,10 <b>plenty</b> 208:25 <b>plg</b> 10:9 282:9 <b>point</b> 39:15,15 40:6,6,8,10 111:12,23 158:6 240:11 242:2 266:20 276:7,11 <b>points</b> 40:2 227:9	<b>poison</b> 100:19 <b>poor</b> 73:11 <b>population</b> 114:22 117:13 250:20,24 251:3,7 254:13 254:20 255:16 <b>possibility</b> 147:3 <b>possible</b> 71:8 92:2 196:12 213:2 214:19 218:21 273:3 274:2 293:22 <b>possibly</b> 71:5 224:2 <b>post</b> 275:4 <b>postoperative</b> 73:7 <b>postsurgery</b> 72:20 <b>postsurgical</b> 72:14 76:25 <b>potent</b> 293:25 <b>potential</b> 35:16 62:22 71:8 87:22,25 88:17 89:18 90:6 91:5 91:18 105:2,8 112:6 117:24 128:4,22 210:9 226:3 273:5 293:13,16,18 296:22	<b>potentially</b> 146:13 184:10 <b>power</b> 182:15 <b>powered</b> 38:11 38:11 <b>ppb</b> 149:9,12 156:12,17,20 156:25 158:9 160:8 185:24 186:13 187:21 188:2 209:11 219:4,25 220:6 230:22 231:13 233:7 234:3 235:3,21 236:5 237:11 238:18 250:4 252:8 <b>ppbs</b> 227:23 <b>ppm</b> 209:10 <b>ppt</b> 251:19,25 <b>practice</b> 14:18 17:9 44:21 50:9 59:3 70:18 79:21,24 81:3 102:2 151:20 182:12 210:13 296:17,21 297:8 <b>precision</b> 181:3 <b>predict</b> 74:17 <b>predispose</b> 90:7 <b>predisposed</b> 115:14
--	--	---	--

<b>predisposes</b> 84:14	<b>prestigious</b> 50:5	254:9 255:15 263:14,20,21 269:16 276:7 280:6,21 293:3	<b>prognostic</b> 83:20 87:3
<b>predisposition</b> 63:20 88:4,6,10 115:21,24 116:17 117:20 118:5 269:19 270:2,10,21	<b>presume</b> 156:4 <b>presumption</b> 7:10 52:6 54:21 <b>presumptions</b> 261:5	<b>problem</b> 181:9 220:10	<b>program</b> 259:22 260:3,5 260:6,12,17,21 260:23,25 261:4,12
<b>preoperative</b> 75:9	<b>pretty</b> 23:9 37:24 73:18 114:5,12 126:8 205:19 210:2 268:25 269:12 275:13	<b>procedures</b> 278:4	<b>proliferation</b> 63:14 85:16
<b>preoperatively</b> 74:15,22	<b>primary</b> 14:18 83:18	<b>proceedings</b> 19:2,8 280:19 280:23	<b>prone</b> 163:7
<b>prep</b> 286:12	<b>principal</b> 294:22	<b>process</b> 7:11 52:6 54:21 65:5 65:23 69:2 137:3	<b>proof</b> 99:11
<b>preparation</b> 286:3	<b>print</b> 225:22	<b>produced</b> 114:2	<b>proper</b> 277:20
<b>prepare</b> 286:14 287:10 289:3	<b>prior</b> 290:11 303:6	<b>production</b> 11:9	<b>properly</b> 38:11
<b>preparing</b> 33:8 131:4 135:16 286:9	<b>privilege</b> 279:19	<b>products</b> 100:15 249:7 249:12,15 250:5,13 253:19	<b>prospective</b> 34:20,23 35:3,7 35:20,22 36:9 36:12,25 37:11 37:20 38:5,10 182:12,14 298:20 299:3,7
<b>presence</b> 96:25	<b>probability</b> 48:5,12 242:9	<b>profession</b> 20:10,21	<b>protect</b> 68:9
<b>present</b> 18:22 71:17 76:24 84:2 115:17 215:10 288:22	<b>probably</b> 16:2 21:12 23:12,24 43:8 64:3 81:5 81:9 90:25 94:11,12 98:9 115:10 116:21 150:24 151:7 152:12 162:25 163:2 209:22 220:25 221:4	<b>professional</b> 44:21	<b>protective</b> 68:15 192:18 293:19
<b>presentation</b> 271:19,20		<b>professor</b> 155:20	<b>prove</b> 182:23 299:2
<b>presented</b> 31:4		<b>profile</b> 9:18,24 246:22,24 247:23 254:3 256:13 257:8	<b>provide</b> 17:6 40:13,13 71:4
<b>presenting</b> 95:18			<b>provided</b> 26:15 45:13
<b>presents</b> 91:24 91:24			<b>providence</b> 2:16
<b>pressure</b> 57:8 57:10			<b>provides</b> 207:24

<b>public</b> 1:22 5:16 13:18 301:24 303:5 303:24 <b>publication</b> 51:7,8,20 <b>publications</b> 23:11,20 50:8 51:11 58:23,24 62:6 75:2 123:17 132:9 136:23,25 248:3 <b>publish</b> 23:11 23:14 <b>published</b> 23:13 30:23 43:23 44:4,6,7 44:13 46:2 62:11 72:2,13 72:18 135:5 140:10 148:19 295:21,25 <b>publishes</b> 152:24 <b>publishing</b> 107:19 <b>pubmed</b> 91:18 126:3,9 135:16 135:20,23 136:11,15 138:24 <b>pull</b> 51:23 107:3 130:6	147:23 157:7 164:8 166:5 167:14 178:21 183:21 188:17 225:11 253:23 282:4 <b>pulled</b> 136:16 137:9 <b>pulmonary</b> 73:12 <b>pure</b> 197:3 <b>purely</b> 170:16 181:16 <b>purpose</b> 14:25 85:2,3 <b>push</b> 270:20 <b>put</b> 22:14 36:24 64:12 91:2 96:12 116:4 238:25 240:20 245:17 260:23 <b>puts</b> 73:23 <b>putting</b> 141:2 232:16 234:17 <b>pylori</b> 86:4	<b>quantifying</b> 118:11 <b>quarters</b> 194:11,17 239:18 <b>quartiles</b> 153:22 154:4 <b>question</b> 5:12 17:4,5,14,16,18 17:20,22 18:11 44:3 94:8 133:21 162:4 176:3 196:18 196:19 198:4 208:24 213:7 228:19 231:7,9 236:9 242:5 255:9,11 264:19 266:3 272:17 287:24 298:16 <b>questioning</b> 209:6 <b>questions</b> 11:19 15:9 53:11 54:5 279:18 298:14 298:19 299:18 <b>quick</b> 115:2 269:12 298:16 <b>quit</b> 94:25 <b>quite</b> 181:12 <b>quotations</b> 10:12	<b>quote</b> 10:14 99:8 263:24 281:23 <b>quoting</b> 108:25
	<b>q</b>  <b>qualifications</b> 19:4 <b>quality</b> 38:15 127:19 <b>quantify</b> 94:20 95:4 201:23,25 202:10 251:5		<b>r</b>
			<b>r</b> 2:2 3:2 129:18 201:4 302:2,2 303:2 <b>radiation</b> 67:8 67:12 269:14 <b>radical</b> 272:16 277:13 <b>radiographic</b> 84:4 <b>raise</b> 13:7 <b>random</b> 38:3,4 38:7 39:9 146:19 171:10 178:9,18 180:17,21 <b>randomized</b> 34:20,23 35:3,8 35:20,22,24 36:10,12 37:2 37:11,20 38:5 38:10 70:11 182:13,14 298:20 299:3,7 <b>range</b> 57:10 144:13 153:19 154:7 155:3 162:15,21,21 163:15 251:18 252:8 259:9,10



261:24 265:16 265:20,24 266:5,16,25 267:25 268:2,9 268:21 269:16 <b>ranges</b> 153:15 <b>rapidly</b> 211:10 <b>rare</b> 83:17 114:6,12,16 <b>rate</b> 71:24 72:5 72:8 74:6 76:17 106:8 183:3,7 183:17 251:19 281:19 284:25 <b>rates</b> 68:3 71:25 73:8,9 74:19,20,20,21 75:23 76:14 110:2 124:12 <b>rather</b> 207:18 <b>ratio</b> 161:3,6,21 162:6,10 172:16 175:22 180:6,8 181:15 192:14,25 <b>ratio's</b> 161:2 <b>ratios</b> 160:16 160:22 169:24 170:9,14,20 171:4,22 172:4 172:7 173:9,18 173:21 174:5,9 174:12 177:18 180:2 185:6	187:12 191:11 191:17,21,25 193:13 195:15 196:4 <b>reached</b> 279:3 279:8 <b>read</b> 10:13 29:6 29:7 31:2 40:10 54:14 94:18 119:16 121:6 122:13,17 125:7,8 126:14 138:4 144:16 147:7,14 151:15 240:15 247:18 251:23 252:10,13 287:5 292:24 293:6 299:23 300:4 301:9 <b>reading</b> 29:9 40:12 54:16 56:9 218:8 246:9 247:10 287:4 <b>reads</b> 138:14 144:18 203:17 204:25 251:17 <b>ready</b> 23:4 <b>real</b> 114:25 182:24 <b>reality</b> 36:10 244:13 249:12	<b>realizing</b> 254:8 <b>really</b> 34:21 35:11,13 57:15 65:6 75:7,9 76:14,18 99:10 123:12,14 124:24 126:13 192:20 206:15 214:16 215:25 223:13 225:22 237:12,15,20 237:20 238:3 238:25 245:15 257:21 270:22 278:19 288:13 <b>realm</b> 41:23,23 234:10 <b>realtime</b> 1:21 303:4,23 <b>reason</b> 15:13 41:15,24 54:25 68:11 111:18 122:3 127:15 128:19 147:6 147:12,20 248:21 252:14 252:22 253:3,7 253:8 256:21 257:2,3 300:7 302:3 <b>reasonable</b> 38:22 39:17 40:4,23 41:9,12 41:14,17,18,20	58:3,7,18,21 59:10,15,23 60:9,13,17 61:6 61:7,21,21 62:2 77:14 244:20 272:8 277:4,10 <b>reasons</b> 88:13 <b>recall</b> 26:9,20 52:16,25 62:8 70:14,17 78:14 110:7 118:22 119:10 121:13 121:16,22 126:12,13,20 129:22 136:3 136:10 137:5 137:24 138:2 138:23,25 143:16,21 152:18 155:22 165:16,24 168:13,18 175:21 176:24 201:20 207:10 221:11,13 240:22 241:11 241:11 246:9 247:16 261:20 265:21 269:20 269:21 279:12 279:22 284:4 298:23 <b>receipt</b> 300:16
--	---	---	--



<b>receive</b> 284:24	<b>records</b> 26:12	<b>reflects</b> 143:18	296:8,14,23
<b>received</b> 272:7	26:14,24 91:20	143:23 162:15	<b>relates</b> 1:9
<b>receiving</b> 272:8	104:5 260:7,24	<b>refresh</b> 119:17	170:16
272:21 273:16	273:12	287:6	<b>relating</b> 276:24
273:25 274:5	<b>recur</b> 80:17	<b>regard</b> 279:18	<b>relation</b> 30:4
<b>recent</b> 23:7,22	276:11	<b>regarding</b>	112:22
273:12	<b>recurred</b> 276:5	123:7 125:16	<b>relationship</b>
<b>recess</b> 77:25	<b>recurrence</b>	125:20 295:22	29:12,14 30:14
141:24 201:2	75:22 76:6,9,14	296:2	31:8,14,23
242:23 291:12	76:17 77:2,6,8	<b>regenerates</b>	32:12 40:16
<b>recognize</b> 22:8	78:16,19,24	85:10	101:4,19
25:4 26:7,11,22	79:10 273:2,5	<b>registered</b> 1:20	126:25 152:5
68:23 121:18	275:8	303:3,23	<b>relative</b> 303:17
130:18 142:19	<b>recurrences</b>	<b>regularly</b> 75:17	303:19
164:19 166:14	80:20 275:11	<b>relate</b> 104:8	<b>relatively</b> 50:5
168:6,7,12	<b>recurs</b> 82:2	109:19 173:22	98:12 275:19
179:6 184:5	<b>reduce</b> 183:18	184:19,22	<b>relevant</b> 83:11
188:25	<b>reduced</b> 183:20	<b>related</b> 30:25	128:2 150:20
<b>recollection</b>	<b>reduction</b>	35:23 44:11	294:13
241:4	183:5,9	60:24 63:16	<b>reliable</b> 50:18
<b>record</b> 10:13	<b>refer</b> 155:7	80:11,12 82:5	107:22 112:10
12:7,19 13:24	173:10 185:16	86:2,6,7,10,15	232:22
29:8 40:11	194:16	92:6 98:10	<b>reliably</b> 171:9
54:15 77:24	<b>referred</b> 194:9	100:14 102:3	<b>relied</b> 123:12
78:4 89:22	260:2,11	109:5,8 118:2	123:14 125:4
141:23 142:3	<b>referring</b> 65:9	121:10 124:12	126:9 195:2
200:25 201:8	65:10 123:22	129:19 132:13	212:4 215:11
242:22 243:2	125:15 127:18	134:17 177:14	248:2,5
286:24 291:8	156:17,25	259:7,16,19	<b>rely</b> 151:16
291:11,15	160:9 206:10	263:17 268:22	159:4 217:18
299:21 301:13	259:21 266:8	269:18 272:21	237:2 238:19
301:14	272:13	273:23 275:15	238:20
<b>recorded</b> 16:19	<b>reflected</b> 10:12	277:3 280:18	<b>relying</b> 31:21
		286:22 290:2	215:8 232:15

237:12,15 <b>remainder</b> 196:2 <b>remaining</b> 273:12 <b>remember</b> 27:6 27:9,20 28:12 44:24 45:2,9 119:15,19 121:19,20,25 122:15 126:16 136:13 137:9 138:5,20 139:2 141:15 148:11 155:23 165:23 207:21 246:12 246:13 247:10 278:17 284:5 286:21 287:22 <b>remind</b> 159:15 <b>remotely</b> 303:8 <b>removal</b> 274:18 <b>remove</b> 277:2 <b>removed</b> 275:7 <b>renal</b> 7:15 73:5 73:5 80:17 86:20,23 87:2 87:10,18 92:8 93:11 107:7 110:6,21 111:11 113:3 113:22 115:23 116:16 124:12 126:6 135:21	273:9,10 <b>reopen</b> 18:11 <b>repair</b> 67:21 <b>repeat</b> 20:13 30:5 176:3 192:11 196:18 198:4 <b>repeated</b> 86:7 <b>rephrase</b> 17:16 17:23 112:23 116:11 272:17 296:19 <b>report</b> 6:7,14 22:4,13,18 24:7 24:20 25:12 30:9 31:12 32:16 33:8 57:22 58:2 74:14 87:15,17 87:20 91:7,10 108:17 119:2,8 121:16,20,22 121:24 123:13 123:21 126:11 126:14,21 129:9 131:5 135:16 139:6 139:21,25 143:4,12 149:4 154:6 156:9 158:2 160:6 165:4 168:22 173:11,23 175:9 176:18	179:16 184:8 184:16 185:16 185:22 186:12 187:21 194:10 199:11 200:16 201:18 203:10 219:12 220:5 222:13 228:10 229:16 233:7 235:4,22 237:3 237:10 239:22 244:8 248:3 250:9 252:4,22 262:9 264:24 265:8 270:25 272:4 292:3,12 292:13 294:14 299:14 <b>reported</b> 131:25 160:16 161:11 163:15 171:23 215:2 222:18 256:22 <b>reporter</b> 1:21 1:22 13:4 93:25 303:4,4,22,23 303:23 <b>reporter's</b> 4:14 10:12 <b>reporting</b> 38:16 179:25 193:12 <b>reports</b> 27:10 27:15,19 31:21 32:4,5 110:16	110:20 111:9 118:21 119:21 120:3,4,6 121:6 122:4,18 123:7 124:21 125:11 191:10,16 248:14 250:4 256:16 286:9 <b>represent</b> 14:23 19:4 73:6 281:7 <b>represented</b> 150:12 <b>representing</b> 150:11 <b>represents</b> 93:11 <b>reputation</b> 50:3 <b>request</b> 11:9 17:4 54:6 <b>require</b> 163:9 260:7 296:17 296:21 <b>required</b> 54:20 <b>requires</b> 275:23 276:18 297:9 <b>research</b> 25:14 33:7,12,16 125:24 126:8 152:24 <b>resected</b> 78:21 79:2 <b>resection</b> 72:25 <b>reserved</b> 5:12
--	--	--	---

<b>reside</b> 239:6	<b>retrospective</b>	136:25 141:16	<b>reynolds's</b>
<b>residence</b>	35:11,12,21	148:11,23	203:4 212:9,15
175:10,17,25	37:3,13,22 38:8	176:14 202:22	212:24 216:16
176:7	166:21 223:15	247:12 248:2	217:6 220:5
<b>resident</b> 176:22	224:3 299:11	286:2,20,23,24	233:7 238:3
<b>residential</b>	<b>return</b> 300:13	286:25 295:22	<b>rhode</b> 2:16
165:12,17	<b>revealing</b> 271:7	296:2	<b>rid</b> 35:23
221:17,21	<b>reveals</b> 286:16	<b>reviewer</b> 43:4,7	<b>ridge</b> 2:7
223:6	<b>review</b> 7:8 18:6	43:10	<b>right</b> 13:7 14:6
<b>resources</b> 42:3	42:17,22 43:14	<b>reviewing</b> 27:6	14:13 15:7 18:2
124:16 271:12	52:4 53:12 62:7	27:9,20 52:16	18:24 22:19
<b>respect</b> 6:12	90:5 92:5 111:5	91:20 102:17	24:24 28:25
24:18	119:8 129:8,15	121:16,22	29:20 32:19
<b>respected</b> 51:6	130:2 131:3,14	126:10 137:24	40:9 41:4 42:16
51:21	137:3 138:19	138:3,5,21	52:21 62:12,16
<b>respective</b> 5:6	141:13,17	141:15 154:12	70:9 74:4 76:22
<b>response</b> 17:7	228:3 234:14	168:18 234:15	77:20,23 78:3
40:15 101:4,8	247:17 257:11	247:15 285:25	78:21 82:13
101:11,16,16	257:25 277:11	295:16	86:18 90:12
101:20,21,23	277:18 278:3	<b>reynolds</b> 31:5	91:12 92:17
102:4,7,10,13	278:11 280:5,8	121:10 123:13	95:24 107:2,25
102:16,22	286:4 294:3,8	123:21 125:5	108:14,22
151:2 152:5	294:15	202:3,22 204:3	109:12 110:17
158:22	<b>reviewed</b> 26:23	207:24 211:17	112:8 116:18
<b>responses</b> 18:7	26:23 28:5,6,8	211:21 212:4	116:24 117:4
18:10	43:18,21 45:6	213:10 215:11	119:3 131:2,21
<b>result</b> 198:16	50:7,21 51:20	216:25 218:15	132:9 133:3
<b>results</b> 182:9	55:7 62:15	218:20 224:17	134:15 135:12
<b>retained</b> 279:23	102:21 103:2	226:2 229:24	135:17,19
280:2,4,8	104:5,6 118:21	230:3 231:23	138:9 139:4,9
281:10,23	118:25 119:13	232:16 233:25	139:21 140:2
<b>retainer</b> 281:12	121:9 123:17	234:19 236:4	141:9,22 142:2
281:17 283:7	124:6 129:13	236:11 237:17	142:17 143:4
283:18	129:18 136:23	238:24 287:3	143:11,14

144:5 145:3,8 145:12,23 146:11 147:22 148:15,21 149:10 153:13 154:25 156:18 157:3 158:10 160:16 161:12 161:19,22 162:8,16 163:17,19 165:4,9,13 166:3 167:2,4,7 168:3,21 169:4 169:9,12,18,22 170:3 171:24 172:5 173:12 173:19,23 174:8 177:21 178:2 179:17 180:3,12 184:16 185:7 185:12,18 186:4,8,14,17 186:21 187:3 187:10,13,23 188:4,9,15 189:13 191:11 191:22 192:3,8 193:14,19 194:6 195:11 196:15 197:3 198:12 200:7 200:18,21,24	201:7 204:24 207:14 208:2 209:15 211:19 212:6,11,17 213:14 214:9 216:7,12 219:18 220:2 221:14,17,25 222:15 226:22 227:5,14,23 228:24 231:3 233:2 234:21 236:15,24 239:7,11,19 242:17,18,21 242:25 246:20 248:4,13,20,24 250:6 252:2,23 255:10 256:4 256:18 260:3 264:23,25 265:2,19,23 266:23 268:2 272:9 277:14 281:2 285:20 291:10,14 294:21 299:20 299:23 <b>rise</b> 95:16 <b>risk</b> 40:21 65:2 71:22 72:14,24 73:16,24 74:3,8 74:12,15 76:9 76:23 78:15,19	78:24 79:4,8,14 79:16,16 84:8 84:14,18 86:13 86:24 87:18 88:18,23,25 89:9,12 90:19 91:4,16 94:4 95:7,9,14 96:3 96:25 97:8,15 102:23 106:10 110:6,17,21 112:6,13,18 113:3,9,12 114:15,23 118:12 121:2 124:4 127:12 142:23 145:21 157:21 158:14 162:18 163:12 163:14 173:6 181:14 199:23 202:7 208:7 237:21 239:2 242:9 263:21 270:22 273:8 295:12,13,15 295:18 <b>risks</b> 64:24 92:20 93:2 138:14 151:24 212:19 296:22 <b>role</b> 38:21 41:3 67:21 68:17	<b>room</b> 153:5 <b>roots</b> 63:6 <b>roughly</b> 115:6 <b>routine</b> 275:14 <b>row</b> 2:15 <b>rule</b> 39:3,8 42:4 42:10,14,19,24 43:11,16 103:13,14 171:10,16 173:7 174:18 178:8,17 180:17,21 <b>ruled</b> 39:16 40:3,23 <b>ruling</b> 41:3,8 41:16 <b>run</b> 182:12
<b>s</b>			
<b>s</b> 2:2 3:2 6:3 7:3 8:3 9:3 10:3 14:4,4 201:4,4 201:4 <b>s1</b> 170:2 <b>salt</b> 51:10 <b>sarcomas</b> 83:18 <b>saw</b> 16:14,15 278:14 <b>saying</b> 100:23 101:2 109:8 112:22 122:14 123:22 126:16 214:25 266:6 270:20			

<b>says</b> 23:9 26:21 133:17 134:6 134:13,13 165:21 191:5 248:19 <b>scar</b> 276:14,18 <b>schedule</b> 10:6 281:25 282:7 283:2 285:4,5 <b>science</b> 49:19 49:22 50:9,15 50:18,23 51:18 53:5,18 55:3 <b>sciences</b> 7:7 49:24 52:3 <b>scientific</b> 33:13 46:11 53:20 54:22 56:22,24 58:4,8,17,21 59:15,24 60:17 61:8,22 125:9 125:13 <b>sclerosis</b> 115:15 <b>score</b> 73:22 <b>se</b> 295:19 <b>sealing</b> 5:7 <b>search</b> 91:18 126:3 135:16 135:20,23 136:4,11,15 138:24 <b>searched</b> 126:5 <b>searches</b> 50:12	<b>second</b> 6:21 22:15 25:23 30:13 108:11 162:2 219:23 285:8 288:10 288:16 <b>secondhand</b> 98:14,23,25 <b>section</b> 3:7 29:3 54:11 57:25 137:19 138:13 144:25 248:10 262:12 265:11 265:11 <b>sections</b> 40:2 <b>see</b> 29:2 36:13 36:21 39:14 40:5 41:10 46:8 54:10 57:24 60:16 61:17 89:2,21,24 101:19 102:6,9 108:9 129:13 138:12,17 139:11 144:9 144:25 145:4 153:5,10,16 156:13 160:15 170:5 175:12 177:13 187:16 203:12,20 204:21 205:8 211:4 226:24 227:4 239:24	240:5 241:6 248:10 251:13 259:12,16 260:4,9,24 262:21 265:17 271:8 277:6 278:11 283:4 285:10 <b>seeing</b> 26:9 95:17 137:5 254:7 259:6 <b>seem</b> 228:5 231:25 <b>seemed</b> 122:12 126:15 131:13 132:4 228:5 <b>seems</b> 59:18 127:15 275:18 275:18 <b>seen</b> 44:3,9,17 45:11 46:10,13 48:25 52:12,19 56:8 80:22 148:8,14,22 157:16,18 225:20 246:14 247:6,8,12 256:9 257:10 275:11 282:16 <b>seer</b> 107:12,15 107:16,22 108:17 110:16 110:20 111:9	<b>semantics</b> 67:7 <b>sense</b> 21:12 93:13,15,18 95:25 215:17 <b>sensitive</b> 97:23 <b>sensitivity</b> 98:19 <b>sent</b> 259:18 282:21 <b>sentence</b> 29:6 39:14 54:14 57:25 138:13 139:11 145:5 240:6 262:12 265:10 <b>separate</b> 60:5 60:10,10,25 <b>separately</b> 137:12 <b>september</b> 279:5 280:7 284:3,8 286:2 <b>sequelae</b> 273:23 <b>series</b> 172:11 <b>serve</b> 43:3,9 <b>served</b> 289:17 <b>serves</b> 20:6,17 <b>service</b> 194:18 <b>services</b> 9:23 254:3 277:16 277:23 <b>session</b> 4:11
--	--	---	---

<b>set</b> 57:20 139:4 147:24 164:10 166:4 198:14 303:14 <b>setting</b> 114:13 182:13 <b>seventh</b> 114:10 262:3 <b>several</b> 122:18 132:8 138:8 166:25 200:5 <b>shape</b> 275:20 <b>shared</b> 140:17 <b>sharon</b> 3:9 12:25 107:3 130:6 225:11 246:21 253:23 282:4 <b>sheet</b> 111:2 281:18 283:7 300:8,11,14 <b>short</b> 141:20 151:4 287:17 <b>shortcomings</b> 136:18 <b>shorter</b> 223:8 268:18 <b>show</b> 34:21,22 35:13 36:4,4,6 36:8 38:11,12 47:2,3,25 48:18 182:16,16 183:4,8,19	<b>showed</b> 89:22 120:22 137:6 <b>showered</b> 206:18 <b>showering</b> 205:17 214:4 <b>showing</b> 75:3 <b>shown</b> 203:18 <b>shows</b> 75:18 <b>side</b> 44:13 47:16 160:17 226:22 <b>sides</b> 21:13 <b>sign</b> 299:23 300:10 <b>signature</b> 4:12 <b>signed</b> 5:16,17 283:12 301:20 <b>significance</b> 42:9 83:21 87:4 161:17 182:2 192:20 196:23 198:11 <b>significant</b> 38:12 89:6 161:21 162:7 162:10,12 170:10,25 171:2,4 172:17 182:8 193:6 195:9 196:7 197:2 198:16 199:3,12 241:17 255:21	<b>significantly</b> 172:7 <b>silver</b> 138:16 <b>similar</b> 46:14 48:25 64:20 86:18 101:7 139:12 151:4 153:5 156:23 158:12 179:10 187:20 209:16 222:22 234:10 234:12 236:11 <b>similarly</b> 42:22 172:3 174:5 202:12 230:20 <b>simple</b> 86:5 <b>simpler</b> 84:11 <b>single</b> 114:6 <b>sit</b> 74:18 <b>site</b> 14:18 <b>sites</b> 14:15 <b>situation</b> 259:14 299:8 <b>six</b> 16:3 23:14 106:7 194:17 280:25 281:4 285:24 286:5 286:12 <b>sixth</b> 114:10 262:3 <b>size</b> 181:24 182:7 <b>skin</b> 79:15 86:6 267:9,11,11,11	<b>skip</b> 168:23 219:23 <b>slices</b> 250:21 <b>slope</b> 103:3 <b>small</b> 80:6 115:3,19 118:9 118:13,18 170:23 172:9 174:23 175:4 181:10 182:7 225:22 <b>smaller</b> 95:11 182:2 183:5 246:14 <b>smoke</b> 93:8 94:22,23 98:6 98:15,23,24,25 <b>smoking</b> 88:19 94:4,13,17,20 94:24 95:4,5,12 97:13,16 98:10 209:13 263:16 267:2 <b>society</b> 50:6 <b>solely</b> 205:3 <b>solitary</b> 270:18 <b>solvents</b> 189:10 <b>somatic</b> 65:13 65:22 66:4 <b>someone's</b> 94:20 <b>something's</b> 171:2
---	--	---	---

<b>somewhat</b> 84:11 163:9 181:16 274:15 <b>sorry</b> 20:14 28:18 30:5 34:3 39:18,20,21 40:6 48:9 65:24 66:20 93:22 105:13 110:23 112:24 113:21 116:12 131:17 132:18 137:14 139:7,15,17 144:16 146:24 154:21 155:17 160:18 167:13 174:16 175:24 176:4 185:19 185:21 187:5,7 191:2 192:11 196:21 197:20 197:25 198:2 200:2 206:8 217:24 231:8 238:15 255:8 256:8 262:13 271:3 287:20 288:9 296:20 <b>sort</b> 56:13 66:3 124:22 150:25 255:16 267:24 275:3 <b>sound</b> 108:21	<b>sounds</b> 17:12 77:20 <b>source</b> 96:10,15 123:5 132:5 <b>sources</b> 96:14 <b>south</b> 2:8 <b>space</b> 35:25 300:7 <b>speak</b> 78:7 286:7 <b>specific</b> 6:6 7:21 22:3 130:11,24 131:12 137:6 277:23 <b>specifically</b> 125:3 131:10 135:3 138:2,5 138:20 199:17 226:3 266:6 297:2,4,12 <b>specifics</b> 122:2 126:17 278:18 <b>spell</b> 14:2 <b>spend</b> 239:17 <b>spending</b> 286:11 <b>spent</b> 194:11 285:22 <b>sprayregen</b> 3:9 12:25 13:2 51:24 167:15 <b>spread</b> 62:23 63:3	<b>ss</b> 301:5 <b>staff</b> 91:6 <b>stand</b> 122:19 <b>standard</b> 29:10 30:2 32:15,21 35:4 43:20,25 45:16,21 46:4 46:18 48:21,25 59:22 61:13,17 208:6,14 271:5 271:13 275:22 275:25 276:5,6 281:17 <b>standards</b> 21:20 29:19 30:8 60:16 <b>standpoint</b> 48:2 117:17 181:21 <b>start</b> 198:5 <b>started</b> 45:8 247:15 <b>starting</b> 203:13 239:25 251:14 <b>starts</b> 65:22 <b>stat</b> 7:14 107:6 <b>state</b> 13:19,23 57:25 300:6 301:4,24 303:5 <b>statement</b> 29:18 32:4 49:14 113:25 147:7,13,21 175:2 234:16 248:22	<b>statements</b> 31:2 33:4 125:6,15 238:22 <b>states</b> 1:2 3:5 12:24 13:2 14:23 18:10 291:6 <b>stationed</b> 194:17 <b>statistical</b> 42:9 161:16 174:25 181:3,21,25 192:19 198:11 <b>statistically</b> 161:21 162:7 162:11 170:10 170:25 171:2 172:4,18,21 174:7,11 181:10 182:8 182:23 183:14 183:19 193:6 195:8 196:7 197:2 198:16 199:3,12 <b>status</b> 73:12 <b>stay</b> 174:10 232:23 <b>staying</b> 226:9 <b>stays</b> 151:23 <b>stenographer</b> 13:6,14 <b>stenographic...</b> 303:13
--	--	--	--



<b>steven</b> 119:5 <b>stick</b> 40:8 136:23,24 <b>sticks</b> 122:25 <b>stimuli</b> 69:4 85:15 <b>stipulated</b> 5:4 5:10,14 <b>stipulations</b> 5:2 11:14 <b>stop</b> 77:17 200:22 242:18 <b>stopped</b> 276:3 <b>stratify</b> 73:20 <b>street</b> 2:7 3:10 205:22 <b>stringent</b> 137:4 <b>strong</b> 51:12 <b>structures</b> 80:12 <b>struggle</b> 99:5 257:21 <b>struggling</b> 119:14 <b>studies</b> 35:10 35:11,21 37:4 37:14,22 38:15 46:15 121:8 124:15 127:19 127:20,22,24 135:9 136:4,15 138:8,16 149:20 166:17 195:8 199:8,10	208:20 209:2 220:11 232:11 249:17 <b>study</b> 8:6,9,12 8:19 9:6,9,12 35:13,24 38:8 38:20 39:3,8 40:17 44:24 45:12 46:17,25 57:8,12 131:9 136:10,13,17 136:18,19,21 136:21 140:8 141:9,10 142:14 146:7 146:18 147:2 148:2 154:5 157:9 159:20 160:3 165:11 165:21,22 166:7,22 168:14 175:5 178:23 183:23 184:9 188:19 189:11,12,15 189:17,18,20 190:14 192:6 195:11 221:8 221:12,16 222:12,15,23 223:9,15 224:4 233:2,9 238:4 294:24	<b>study's</b> 42:19 42:24 43:11,16 <b>studying</b> 122:23 <b>subgroup</b> 191:8 <b>subjective</b> 241:18 <b>submit</b> 93:9 <b>submitted</b> 22:19,23 285:13 <b>subscribed</b> 301:20 <b>substance</b> 128:10,24 146:10 245:24 <b>substances</b> 128:17 289:20 293:14 <b>substantial</b> 201:19,24 202:2,9 205:2 205:12 206:5 <b>substantially</b> 240:13 241:14 241:15,19,23 <b>substantive</b> 19:14,25 <b>subtypes</b> 191:17 <b>sufficient</b> 29:11 29:13 30:3,13 31:13,22 32:11 120:25 134:14	<b>suggested</b> 204:10 <b>suggests</b> 127:11 <b>suite</b> 3:11 <b>sulpizio</b> 2:6 4:10 12:20,21 20:11,22 21:9 24:3 29:22 30:10 31:17,25 32:22 33:24 34:9,25 37:6,15 48:7,14 49:7 53:8,21 54:4 55:4,15 56:18 60:2,21 61:10 66:6,15 67:4 69:10,16,19 70:15,21 77:11 78:22 79:6 90:22 91:8 92:12 93:4,16 93:24 94:5 96:4 97:4,10,17 99:3 100:4,20 103:21 105:3 105:10,13,25 109:16 110:8 111:14,25 112:14,20 113:5 114:18 115:8 116:2,9 116:25 117:9 118:14 122:6 127:5 129:10
--	---	---	---



131:6,22	220:8,22 222:2	<b>summarizing</b>	162:5 168:8
132:16 133:4	222:8,25	135:6	176:4 185:20
133:11 134:4	223:10 224:9	<b>summary</b>	189:19 192:12
134:11,19	224:14 227:15	133:2 135:8	196:20 198:6
136:7 140:15	227:24 228:14	<b>supplement</b>	200:3 208:23
140:22 143:19	229:7,20 230:8	24:4 173:10	209:5,5 213:9
144:14 145:13	230:16,24	<b>supplemental</b>	218:2,8 219:7,7
145:24 146:20	232:7 233:10	6:18,22 25:19	219:10 229:15
147:18 150:22	234:4 235:5,23	25:23 168:14	235:9 236:10
152:6 154:9	236:7 237:13	168:19 171:22	242:4,16
155:14,17	238:5 239:8	173:15 177:20	255:12 264:18
157:4 158:3,18	241:8,25 242:6	<b>supplied</b> 240:10	268:5 277:19
159:12 160:11	242:11 243:16	<b>supplies</b> 246:4	280:11,16
161:23 162:23	244:22 245:7	246:11	<b>surgery</b> 71:20
163:23 165:5	246:6 248:17	<b>supply</b> 215:3	72:25 73:14,17
165:18 170:11	249:8 250:14	<b>support</b> 11:2	74:13 75:7 76:2
171:12 172:23	252:11,18	<b>sure</b> 13:25	109:22 274:11
173:25 174:19	254:22 255:6	16:10 18:3,13	277:2
175:13 176:10	255:25 258:5	19:21 20:15,24	<b>surgical</b> 75:11
178:12 179:18	261:8,14 263:9	23:5 30:7 34:4	75:14,19,23,24
181:4 185:25	264:13,20	39:13 48:10	77:7 79:25
190:16 192:9	267:5 268:3,12	50:11,16,16	276:14,18
194:13,20	277:25 279:15	65:3,6,25 66:19	<b>surprise</b> 249:5
195:3,12,23	280:13 281:14	85:17 87:16	249:10,23
196:10,16	283:14,21	89:14 92:23	<b>surveillance</b>
197:4,11,15,23	284:14,20	94:16 101:5	273:22 274:5
198:2,20 199:4	286:15 287:11	110:25 112:25	275:24 276:3,8
199:15,24	288:2 290:4	116:13 119:18	<b>survival</b> 76:15
200:8 203:6	296:24 297:6	132:19 133:16	110:2 190:4
207:2 208:9	298:2,15,18	133:19,23	<b>survive</b> 190:3
213:5,15	299:17,22	135:22 138:3	<b>susceptibility</b>
214:22 215:18	<b>sum</b> 203:13	146:23 148:13	163:3 268:19
216:19 217:12	<b>summarize</b>	148:13,25	<b>suspicious</b>
218:6,23 219:5	134:25	156:24 157:6	269:25 270:5

<b>sustained</b> 158:9 159:9,25 160:7 160:8 220:18 <b>swear</b> 13:5,9 <b>swells</b> 85:12 <b>sworn</b> 5:18 13:17 303:9 <b>synergistic</b> 293:4,9,24 <b>system</b> 68:22 240:11,24,25 <b>system's</b> 68:23	185:10 186:17 187:6,11,17,23 190:24 191:2,3 191:10 193:10 193:12,23 194:5,9,16 225:25 226:21 250:2 256:15 256:23 257:15 <b>tables</b> 168:6,15 168:19 171:22 173:15 177:21	<b>talked</b> 62:5 70:10 123:6 150:25 163:2 298:21 <b>talking</b> 52:22 65:16 76:19 111:3 115:2 123:20 135:4 <b>tce</b> 36:19 120:8 127:2,9,14 129:2,9,17 134:9,13 142:23 149:8 156:18 157:22 158:10,16 160:8 169:8 171:23 184:20 185:6,24 186:13,20,23 207:14 211:18 213:13 214:7 219:4 220:2,7 220:20 221:3 225:8 226:22 228:13,18 231:12 233:24 234:3,20,22,23 235:3,21 236:11 237:19 240:13 242:15 243:14 244:8 244:16,19,21 245:3,6 246:4 246:17,22	247:23 248:25 249:6,11,24 250:4,11 253:5 258:9 263:15 293:5,12 295:23 296:23 297:2,15 <b>tell</b> 22:11 63:24 74:18 133:16 286:13 <b>telling</b> 287:8 <b>ten</b> 23:12 81:5 <b>term</b> 46:7,13 49:4 52:23 53:6 53:19 54:17,19 55:18 59:23 75:21 99:9 100:23 103:7 241:18 273:23 <b>terminology</b> 46:14 129:21 <b>terms</b> 23:20 73:18 82:13 93:9 126:6 151:17 158:22 205:14 221:2,4 273:17 <b>test</b> 122:23 258:12,13 <b>tested</b> 198:24 <b>testified</b> 13:20 78:10 142:10 201:15 243:9 280:25 291:23
<b>t</b>	<b>take</b> 17:7,10 23:2 27:23 36:17 55:11 75:17 77:18 80:13 112:12 122:24 141:20 146:14 182:16 182:18,20 238:9 242:2 250:17,21 260:24 266:21 <b>taken</b> 51:7,19 77:25 80:18 141:24 145:12 145:17 147:10 147:16 201:2 242:23 291:12 301:10 303:13 <b>takes</b> 68:8 <b>talk</b> 34:12 65:8 124:6 209:2		
<b>t</b> 6:3 7:3 8:3 9:3 10:3 201:4 301:2 302:2 303:2,2 <b>t3n0</b> 275:8 <b>tab</b> 21:25 22:2 51:23 107:4 130:6 147:23 157:7 164:8 166:6 178:21 183:21 188:17 225:11 282:4 <b>table</b> 8:22 132:20,25 152:20,21 153:11 159:18 160:15,18,19 161:11 167:21 170:2 173:17 177:11,17 179:22 185:3,5			

298:10	<b>thesaurus</b>	89:15 90:25	204:14 206:6
<b>testifies</b> 19:12	41:21	92:16 95:10	207:4 209:3,21
19:23	<b>thick</b> 153:7	96:7 97:19	212:7,8 218:9
<b>testify</b> 19:9,13	<b>thing</b> 33:22	98:17 99:5,8,23	220:21,24
19:24 303:9	70:25 209:14	100:12,25	221:6 223:3,12
<b>testifying</b> 16:14	274:21	102:24 103:4	223:12,24
18:21	<b>things</b> 60:20	103:23 104:7	224:6,11,18,19
<b>testimony</b>	62:2 71:7,10,13	106:6,23	224:21,22,25
13:10 15:15,19	71:14 72:11	108:11 109:6	225:6 228:2
18:20 20:7,9,18	74:13 89:24	110:4,10	229:9,22 230:3
20:20 21:4,8,21	100:9 120:6	112:16 113:24	231:6,14,15,15
27:7,14 78:7	151:17 153:21	116:22 117:3	231:17 232:9,9
142:6 201:11	170:15 182:17	118:3 123:8	232:10,23
213:12 214:2	233:13 244:11	127:7,7,10,14	233:12 234:6,6
226:13 243:5	266:11 286:2	128:10,14,19	234:7,8 240:21
245:22 280:3	<b>think</b> 21:11,12	133:18 134:21	240:22 243:11
284:25 287:2	25:7 27:25	134:24 136:11	244:9 246:8
291:18 298:5	30:16,18 32:6	137:11,18	249:15 254:15
301:10,13	32:18 34:17	139:25 142:12	263:11,11,12
303:12	36:16 37:24,24	149:21 150:24	263:23,24
<b>testing</b> 42:8	44:10,13 45:4	151:6,6,7 152:3	266:2,2,13
161:16 196:23	45:23 48:16,16	152:13 155:19	267:13 268:15
271:5,14	48:17 49:3,9,10	158:20 159:3	269:15,18
<b>tests</b> 257:25	49:13 52:16,19	162:9 165:20	270:20 274:13
258:9	55:6,18,21,22	165:21 166:6	278:8,18 279:7
<b>texts</b> 33:13	56:5,20 57:5,18	170:14,15	280:24 284:6
<b>thank</b> 13:14	59:11,13 60:4,4	172:8 176:15	284:16,21
<b>theoretically</b>	60:6,10,23,24	176:15,17,17	<b>thinking</b> 109:2
84:24	60:24 61:12,13	176:21,21	109:4 200:18
<b>theory</b> 20:8,9	64:16,22 65:12	177:7,7 181:7,9	246:13
20:19,20 21:2,5	67:2,6,14 70:4	182:18 190:18	<b>thinks</b> 56:22
21:7,13,16	70:5,7,9 77:13	190:19,20,21	<b>thinners</b> 210:18
35:23 63:7	77:16 78:12	195:5 197:17	210:22
84:20 137:2	84:17,23 85:13	197:21 200:21	

<b>third</b> 63:23 203:17 219:24 226:19 240:5 262:11 292:16 <b>thirty</b> 300:15 <b>thorough</b> 90:4 <b>thought</b> 126:17 136:12 197:20 229:23 238:10 244:10 266:4,6 <b>thoughts</b> 56:15 <b>thousands</b> 183:10,10 <b>three</b> 23:17,25 227:20 <b>threshold</b> 202:13 <b>thresholds</b> 268:17 <b>time</b> 5:13 12:11 14:25 19:20 20:13 23:11 27:23 34:2,16 39:24 44:16 48:9 50:13 62:24 68:2 71:3 77:22 78:2 80:13 93:21 103:9 111:5 127:23,25 128:12,20 133:22 141:21 141:25 146:22 152:11 159:3	162:4 176:19 176:21,22 198:17 200:2 200:23 201:6 204:11 206:15 208:17 214:8 225:5 227:9 242:20,24 251:4 254:11 279:6 280:7 284:19 285:21 291:9,13 295:17 299:19 299:25 303:13 <b>times</b> 15:24 16:3 115:7 149:21 161:7 166:25 273:4 281:4 287:15 <b>timothy</b> 119:9 119:10 <b>titled</b> 144:25 <b>tobacco</b> 98:5,24 <b>today</b> 13:12 15:2,15 16:19 18:16,20 21:21 78:10 142:10 201:15 243:9 286:14 291:23 298:6,10,20 <b>today's</b> 12:10 <b>together</b> 14:25 22:14 75:25 91:3 96:12	141:3 232:17 234:17 240:21 294:9,10 <b>told</b> 273:20 <b>tolerate</b> 57:13 73:14 <b>tolerated</b> 47:22 <b>took</b> 45:10 <b>top</b> 39:25 108:7 139:18 191:8 200:19 267:14 292:16 <b>topics</b> 50:13 <b>tops</b> 281:7 <b>torts</b> 3:6,7 <b>total</b> 91:22 143:23 144:7 150:13,14 159:2,2 169:13 172:10 207:25 208:5,11,17,21 209:2,4 211:22 211:24 213:3 214:21 215:14 216:18 217:8 221:5,7,12 222:19 229:12 255:5 257:19 <b>totality</b> 257:11 <b>tough</b> 124:19 274:21 <b>towards</b> 270:20 271:2	<b>towers</b> 259:11 <b>tox</b> 246:22 <b>toxic</b> 46:24 47:21 258:2 289:20 <b>toxicological</b> 9:18,24 246:24 247:23 254:3 256:13 <b>toxicologist</b> 295:3 <b>toxicology</b> 257:8 <b>toxin</b> 85:7 104:17 <b>traceable</b> 66:3 <b>tracks</b> 107:17 <b>tract</b> 80:5 <b>trade</b> 259:22 260:2 261:11 <b>training</b> 19:14 19:25 206:20 206:21 240:10 296:18,22 <b>transcript</b> 4:3 4:16 18:7 206:3 300:17,18 301:10,12 303:12 <b>transcripts</b> 27:19 <b>translating</b> 227:22
--	---	--	--

<b>traumatic</b> 275:4 <b>travel</b> 285:2 <b>treat</b> 46:20 80:2 80:4,8 259:25 <b>treatable</b> 109:14 <b>treated</b> 109:22 274:22 275:5 296:7 <b>treating</b> 47:19 79:21 81:3,7 103:18 289:14 <b>treatment</b> 46:18,22 47:2,3 47:5,10,14 48:20,22 272:6 272:12,14,20 273:16,25 274:3,14 277:3 <b>treatments</b> 71:20 <b>tree</b> 82:10 <b>trial</b> 5:13 14:22 18:12 34:20,24 35:8,20 36:10 37:2 38:6,10 47:6,24 70:11 182:14 299:7 <b>trials</b> 35:3,22 36:12 37:11,20 102:10 182:13 298:20 299:4	<b>trichloroethyl...</b> 9:19 246:25 248:15 251:21 252:6,16 <b>tried</b> 46:19 87:21 <b>trillion</b> 252:2 <b>triple</b> 255:22 <b>trouble</b> 292:9 <b>true</b> 21:14 46:19 94:17 196:9,25 198:17,25 212:10 256:25 301:12,15 <b>truly</b> 65:15 128:24 151:8 <b>truth</b> 13:10,11 13:12 303:9,10 303:10 <b>truthfully</b> 20:24 76:18 133:13 252:20 256:24 274:23 <b>try</b> 17:10,15 33:4 251:5 <b>trying</b> 47:25 48:18 134:25 182:25 191:12 214:17 235:15 267:13 268:8 270:7 <b>tubular</b> 115:15	<b>tumor</b> 62:22,25 63:2 65:14 82:3 90:12,13 275:7 275:8 <b>tumors</b> 83:15 83:18 270:16 <b>turn</b> 28:22 39:11 53:25 87:14 108:2 110:13 132:22 137:17 144:2 144:23 152:20 159:18 171:19 173:16 177:10 179:22 185:2 187:3 190:24 193:9 203:9 226:18 239:21 248:7 250:2 251:10 256:5 265:7 272:3 292:2 <b>tv</b> 278:15 <b>tvoc</b> 169:21,24 <b>twice</b> 281:6 <b>two</b> 23:24 29:19 30:8 55:22 60:5 60:16,20,25 91:21 92:8 122:17 187:5 216:6 220:16 227:18 232:3 238:12 252:9 257:18 287:17	288:5 <b>type</b> 78:20,25 81:19,20 82:5 190:14 193:2 245:10 250:18 281:12 297:25 <b>types</b> 78:16 79:4 81:11 83:7 83:17,19 84:7 85:21,24 86:20 86:23 87:2 90:2 105:24 108:7 108:10,13 109:14 250:12 269:6,14 290:8 <b>typical</b> 251:14 251:18 252:7 261:19,23
			<b>u</b>
			<b>u.s.</b> 9:22 253:25 <b>ultimately</b> 66:3 <b>ultra</b> 262:5 <b>un</b> 229:22 <b>unable</b> 15:14 <b>unaware</b> 76:2 104:18,19 258:16 273:15 <b>uncertainty</b> 227:22 <b>unclear</b> 17:14 17:15 28:3 238:16 <b>uncommon</b> 172:13 266:20

275:13 <b>under</b> 18:16,17 28:2 117:17,22 118:4,17 262:8 271:5 280:21 301:10 <b>underestimate</b> 204:8 212:9 213:3 215:14 216:17,22 217:7 230:6,12 254:10 255:4 255:15,21 <b>underestimates</b> 229:24 230:3 <b>underestimates...</b> 216:14 <b>undergo</b> 276:8 <b>undergoing</b> 272:15,18 273:21 <b>undergone</b> 274:13 <b>underlying</b> 8:24 73:9 167:22 <b>underreprese...</b> 235:14 <b>understand</b> 15:2,5,11 16:10 16:18,23 17:3 17:13,22 18:5,9 18:15,19 20:25 84:24 100:22	100:25 197:17 208:23 213:7 219:8 264:18 280:11 281:21 281:22 <b>understanding</b> 30:21 31:13 119:25 126:25 127:3 131:16 131:18 132:12 134:16 146:5 151:23 158:13 160:25 218:10 220:4 240:18 266:24 275:6 <b>understood</b> 17:19 176:4 <b>underwent</b> 272:19 <b>unethical</b> 36:23 299:9 <b>unexplained</b> 103:19 <b>unfortunately</b> 109:6 228:2 231:25 <b>unidentified</b> 96:20 <b>unifocal</b> 90:17 <b>unilateral</b> 88:12 <b>unilocular</b> 88:12 90:11	<b>united</b> 1:2 3:5 12:24 13:2 14:23 18:10 291:6 <b>universe</b> 104:25 105:8 <b>unknown</b> 99:7 104:2 105:19 112:7 <b>unrelated</b> 71:19 <b>unusual</b> 113:24 <b>updated</b> 23:24 <b>urologic</b> 80:14 <b>urologist</b> 80:9 <b>usdoj.gov</b> 3:14 <b>use</b> 34:16 53:20 54:23 67:8 99:9 100:24 101:7 149:21 151:19 183:13 203:3 218:20 220:12 232:12 <b>used</b> 33:13,17 46:11,13,15 49:2 54:20 58:20 59:2,4,14 101:3 135:23 202:23,24 204:3 208:12 226:12 228:6 231:21 285:5 300:19	<b>uses</b> 43:24 45:15 46:7 <b>using</b> 41:20 45:20 123:6 140:24,25 232:2,18 238:13 299:10 <b>usual</b> 126:2 <b>usually</b> 65:15 74:21 75:25 102:10 173:3 183:12 270:10 270:13 <b>utility</b> 40:18 190:15 <b>utilize</b> 35:8 126:3 136:20 295:15 <b>utilized</b> 25:15 33:3 107:19 120:5,18 204:5 <b>utilizes</b> 44:10 <b>utilizing</b> 132:3 226:7 231:23 235:12 238:25  <b>v</b>  <b>value</b> 161:25 162:13 178:4 178:16 180:11 180:20 <b>values</b> 234:10 234:12 238:23 <b>variable</b> 147:3
---	--	---	---

<b>variables</b> 37:13 39:4 42:13	242:20,24 291:9,13	<b>want</b> 24:5 36:6 46:25 47:3	<b>way</b> 36:2,8,24 42:9,14 46:20
<b>variation</b> 211:15	299:19	61:17 74:17	56:14 67:25
<b>various</b> 250:4	<b>videotaped</b> 1:16	77:18 93:20	68:8 69:3 109:6
<b>vary</b> 78:20 79:5 81:15 269:6	<b>vinyl</b> 36:20 120:9 127:3,9	129:23 133:15	116:5 122:9
<b>vascular</b> 98:8	128:18 169:8	143:6 167:15	147:5 152:22
<b>vast</b> 113:12 261:25	211:18 244:16	183:8 257:6	152:23 176:5
<b>vc</b> 226:23 237:19 292:7	296:3 297:5,16	272:18 279:5	192:12 210:11
292:20 293:15	<b>vitae</b> 22:16,22 23:3,7,22	<b>wanted</b> 278:12 286:4 287:6	210:24,24
<b>ventilator</b> 73:17	<b>vlaanderen</b> 138:15	<b>warrant</b> 271:4	232:6 243:19
<b>verbal</b> 16:22	<b>voc</b> 169:13 211:22,24	<b>washington</b> 3:12	244:14 245:10
<b>verbatim</b> 303:12	<b>vocs</b> 125:17,20 125:25 202:13	<b>water</b> 1:7 7:20 12:15 130:10	252:21 254:24
<b>veritext</b> 1:24	227:8	130:23 132:2	255:2 256:24
<b>versa</b> 51:15	<b>volatile</b> 89:7 135:24 245:14	142:24 155:8	259:14 280:16
<b>version</b> 23:22 23:23	<b>volume</b> 85:12	155:13,25	299:9
<b>versus</b> 21:15,15 36:7 150:15	<b>von</b> 88:16 115:15	156:7 157:3	<b>ways</b> 33:2 146:18 147:2
162:21 191:6	<b>w</b>	166:20 179:14	<b>we've</b> 77:15 199:9
216:4 281:23	<b>w</b> 6:15 14:4 24:21 301:2	184:11 189:10	<b>week</b> 206:20
<b>veterans</b> 7:9 52:5 296:14	<b>wait</b> 162:2 284:7	192:7 199:14	<b>weighing</b> 47:15
<b>vice</b> 51:15	<b>waived</b> 5:9	199:17 203:19	<b>weight</b> 209:20 209:24 210:8
<b>videographer</b> 3:17 12:6,8	<b>walk</b> 234:25 235:19 237:7	205:5 206:19	210:15,23
13:3 77:22 78:2	<b>walking</b> 254:14	213:20 214:8	211:11,14
141:21,25		215:2 225:4	<b>weights</b> 211:16
200:23 201:6		226:17 240:10	<b>weiss</b> 1:17 4:8 6:6,9,14,17,18
		240:11,24	6:21,21 7:6,13
		245:4 246:4,11	7:18 8:6,9,12
		246:15 252:5,9	8:15,19,22 9:6
		253:4 254:21	9:9,12,15,18,22
		255:3 278:7	10:6,7,9 12:17
		279:13 289:10	13:16,23,25
		290:22 296:8	



22:3 24:16,20 25:18,19,22,23 52:2 78:6 107:5 130:8 142:5,13 148:2 157:9 164:11 166:7 167:20 178:23 183:23 188:19 201:10 225:13 246:23 253:25 282:6,7,9 291:17 301:8 301:18 303:8 <b>went</b> 28:10,10 120:13 286:20 286:22 <b>wide</b> 174:23 181:12 <b>widely</b> 20:10,21 21:5,7 248:15 <b>wider</b> 76:8 <b>width</b> 75:11 77:10 180:25 <b>widths</b> 78:15 <b>wife</b> 28:7 <b>william</b> 9:15 225:14 <b>willing</b> 278:11 <b>witness</b> 6:11 11:4 13:5,13,16 15:22 16:9,12 16:13 19:3,12 19:23 20:6,17 24:18 77:20	144:18 166:10 247:4 254:6 278:13 280:3 289:18 290:12 290:17,20 291:5 299:24 300:2 <b>witnesses</b> 16:5 27:7,10 <b>woburn</b> 8:15 164:11,24 165:12 223:6 <b>women</b> 111:10 113:8 <b>word</b> 41:20 <b>work</b> 48:19 50:17,22 67:25 121:10 266:17 284:2,12 285:17 290:2 <b>worked</b> 284:10 290:9,12,16,20 291:4 <b>worker</b> 194:12 239:19 <b>workers</b> 189:9 193:13,18,19 <b>working</b> 205:21 205:23 <b>works</b> 48:6,13 48:19 56:23,25 57:11 <b>world</b> 72:4 259:21 260:2	261:11 <b>worse</b> 75:5 <b>wound</b> 74:20 183:2,6,16 274:11 <b>write</b> 262:18 265:11 271:4 292:6,19 293:2 296:13 <b>writing</b> 201:20 <b>written</b> 284:19 <b>wrong</b> 215:3 217:18 252:23 <b>wrote</b> 240:8 262:23 <b>x</b> <b>x</b> 6:3 7:3 8:3 9:3 10:3 <b>y</b> <b>yeah</b> 21:3 39:10 39:22 40:7 45:6 61:12 62:20 77:3 87:20 90:24 101:20 102:5 103:10 106:19 108:20 110:12 112:16 116:14 117:11 123:4 126:8 134:3 144:19 148:20,22,22 148:23 149:5 150:10 154:13	160:21 168:16 168:16 176:2 180:10 197:20 214:24 219:13 227:17 248:5 253:15 262:16 265:3 268:5 269:7,7,15 272:25 284:8 288:3 <b>year</b> 22:19,23 23:13 97:14,15 106:7,8,22 108:25 110:2 151:25 152:2,2 152:25 156:20 159:21 160:2,4 160:9 161:22 161:25 162:7 165:22 209:10 209:13,17 221:17,20,25 222:7,10 223:7 224:8 278:20 281:7 <b>years</b> 43:8 94:21,23,25 98:16 154:18 154:23,24 155:4,4 156:12 156:17,25 219:4,25 220:6 239:14 263:5 263:20 264:2,5
--	---	--	--



264:6,11 265:15,25 266:13,15,15 266:21 267:10 267:17,19,21 269:2 273:2 <b>yep</b> 108:15 139:19 156:14 <b>york</b> 1:18 12:4 12:14 13:19 14:10,11 259:5 303:5 <b>young</b> 117:16 269:24 270:6 270:12 <b>younger</b> 100:10 100:11 261:18
<b>z</b>
<b>zach</b> 2:12 12:21 278:23,25 279:2,12 288:7 288:9,18,19 <b>zachary</b> 2:14 <b>zero</b> 115:25 116:7,18 117:7 118:16 158:9 160:7 162:14 163:16,21 164:4 220:19 <b>zeroed</b> 176:19 177:8 <b>zoom</b> 2:12

Federal Rules of Civil Procedure

Rule 30

(e) Review By the Witness; Changes.

(1) Review; Statement of Changes. On request by the deponent or a party before the deposition is completed, the deponent must be allowed 30 days after being notified by the officer that the transcript or recording is available in which:

(A) to review the transcript or recording; and

(B) if there are changes in form or substance, to sign a statement listing the changes and the reasons for making them.

(2) Changes Indicated in the Officer's Certificate. The officer must note in the certificate prescribed by Rule 30(f)(1) whether a review was requested and, if so, must attach any changes the deponent makes during the 30-day period.

DISCLAIMER: THE FOREGOING FEDERAL PROCEDURE RULES ARE PROVIDED FOR INFORMATIONAL PURPOSES ONLY.

THE ABOVE RULES ARE CURRENT AS OF APRIL 1, 2019. PLEASE REFER TO THE APPLICABLE FEDERAL RULES OF CIVIL PROCEDURE FOR UP-TO-DATE INFORMATION.

VERITEXT LEGAL SOLUTIONS

COMPANY CERTIFICATE AND DISCLOSURE STATEMENT

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

Veritext Legal Solutions is committed to maintaining the confidentiality of client and witness information, in accordance with the regulations promulgated under the Health Insurance Portability and Accountability Act (HIPAA), as amended with respect to protected health information and the Gramm-Leach-Bliley Act, as amended, with respect to Personally Identifiable Information (PII). Physical transcripts and exhibits are managed under strict facility and personnel access controls. Electronic files of documents are stored in encrypted form and are transmitted in an encrypted

fashion to authenticated parties who are permitted to access the material. Our data is hosted in a Tier 4 SSAE 16 certified facility.

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